QUEENSLAND COURTS

OFFICE OF THE STATE CORONER

Non-inquest findings of the investigation into the death of William John Bligh

CITATION:	**Investigation into the death of William John Bligh**
TITLE OF COURT:	Coroner's Court
JURISDICTION:	Cairns
FINDINGS OF:	Ms Jane Bentley, Northern Coroner
CATCHWORDS:	CORONERS: melioidosis, Palm Island, clinical management.

Counsel Assisting: Ms Stephanie Williamson

At the time of his death, William John Bligh was 9 years old and lived at 165 Farm Road, Palm Island.

Surrounding Circumstances

On 2 February 2013 William was in Townsville with his mother, Ms Astrid Bligh. On that day he developed a fever and was lethargic. On 3 February William continued to be unwell so Ms Bligh took him to the Cranbrook Medical Centre. The doctor there said that maybe William was getting the flu and prescribed Nurofen to lower his temperature.

On 4 February William and Ms Bligh returned to Palm Island. William continued to be unwell with a fever.

On 11 February Ms Bligh took William to the hospital on Palm Island. He was seen by a doctor who checked his ears, throat and chest and who said he could find nothing wrong.

On 13 February Ms Bligh took William back to the hospital. He was checked again but there was still no diagnosis.

On 14 February William continued to have a fever and was still lethargic. Ms Bligh again took him to the hospital. William was seen by a different doctor who took blood tests and a chest x-ray. A spot was seen on his chest and antibiotics were prescribed.

On 20 February William finished his course of antibiotics. Although he had improved he was still unwell. By 20 February he again had a temperature and was lethargic. Ms Bligh took him to the hospital. He was admitted and antibiotics were administered intravenously. The next day the antibiotics were changed as William was not responding.

On 23 February 2013 William's condition was deteriorating and he was transferred to the Townsville Hospital by medivac.

Early on the morning of 24 February William's condition deteriorated further and he was transferred to the intensive care unit. William's parents were told that he may have a staph infection. At 11.30am his parents were told that he would not survive the day.

William was pronounced deceased at 12.08pm on 24 February 2013.

Post Mortem Examination

Professor David Williams, Consultant Forensic Pathologist, conducted an autopsy on 27 February 2013 and concluded that William died from disseminated melioidosis. Professor Williams noted:

> *Post mortem examination was performed on this 9 year old boy and demonstrated that he had extensive infection due to disseminated Melioidosis. This infective condition is notoriously difficult to diagnose. The course of Melioidosis can be quite*

varied but the acute illness usually presents as a pulmonary infection, sudden onset of fever and a dry cough.

This child had acute Melioidosis and, in this case acute septicaemic illness was demonstrated by many discrete abscesses throughout the body. All organs may be involved but the lesions are most frequently found in the lungs, liver and spleen, as demonstrated in the case of this child. The organisms could be seen within these abscesses by the use of special stains.

Review by Dr Hall

The treatment and clinical management of William was independently reviewed by Dr Gary Hall, Forensic Medical Officer, Clinical Forensic Medicine Unit.

Dr Hall thoroughly reviewed the medical records from the Cranbrook Medical Centre (Townsville), the Palm Island Hospital (JPHS) and the Townsville Hospital and provided the following information.

Chronology

2 February 2013 — William was lethargic and developed a fever.

3 February 2013 — Ms Bligh took William to the Cranbrook Medical Centre. The doctor could find nothing which could be ascribed to the temperature and suggested it might a viral 'flu'. No medication was prescribed and Ms Bligh gave him ibuprofen.

7 February 2013 — Ms Bligh took William to JPHS. Doctors recorded he had a fever and cough for three days, no other symptoms, well-hydrated, bright and active. Diagnosis was fever with no obvious source and recommendation was for observation of symptoms with review in two to three days with possible blood and urine tests if no improvement.

11 February 2013 — Ms Bligh took William to JPHS. The doctor could again find no cause for his fever and told Ms Bligh to give him paracetamol. It was noted that William's fevers were settling and he had developed a nocturnal cough. Examination showed clear lung fields with some palpable neck glands. Diagnosis was of a viral illness which appeared to be resolving. Recommendation was for observation and review as necessary.

14 February 2013 — Ms Bligh took William to JPHS where he saw a second doctor who ordered a chest x-ray and blood tests. It was noted that he had a fever on and off for two weeks, intermittent lethargy, continued cough, sweats and reduced appetite. Chest examination was clear. The x-

ray suggested a chest infection and he was prescribed and took oral antibiotics (amoxicillin) until 19 February 2013. Between 15 and 19 February 2013 William had improved and was able to eat better.

20 February 2013 William was again lethargic and febrile so Ms Bligh took him to JPHS where he was admitted. He was diagnosed with pneumonia and treated with intravenous fluids and antibiotics.

21 February 2013 William was reviewed on the ward round. At 3pm that day he had spiked a temperature to 40.7 degrees Celsius but had responded to paracetamol and he remained bright and had eaten a small amount of food.

22 February 2013 At about 9.10am William was again reviewed on the ward round by four doctors. His temperature spikes were noted. He was re-assessed at about 3pm that day. His respiratory rate had increased. The doctors consulted with the paediatric registrar from Townsville Hospital who advised adding flucloxacillin 500mg sixth hourly to cover Staphylococcal pneumonia. At about 7.50pm William complained of abdominal pain and some loose bowel motion; he had two vomits. It was noted that he was tender in the upper abdomen.

23 February 2013 William was reviewed on the ward round. He had increased difficulty breathing with back pain on coughing. A repeat chest x-ray was ordered and after consultation with the paediatric team at Townsville, arrangements were made to transfer William there. He was transferred to the Townsville Hospital by medivac.

William was admitted to the Children's ward and admitted by the paediatric registrar. William was responsive but lethargic. He had left upper lobe bronchial breathing suggestive of infection and intercostal recession (sucking in of chest wall muscles with respiration indicating increased work of breathing). There was abdominal distension with palpable liver. A CT scan of chest and abdomen were performed.

The CT scan showed extensive pneumonia with left upper lobe air bronchograms and suggestion of cavitating lesion (abscess) as well as multiple nodular densities throughout the left lung in keeping with septic embolisation ('seeded' - spread via bloodstream to other organs) with associated infarcts/abscesses in the spleen and possibly liver, some free abdominal fluid and possibly involvement of the small and large intestine.

A discussion was had with an infectious diseases physician who recommended a combination of meropenem and vanycomycin intravenously after blood cultures and streptococcus antigen were taken.

Blood tests revealed deteriorating kidney and liver function. William was closely monitored throughout the night and was visited several times by the paediatric registrar.

William continued to deteriorate until the early hours of 24 February 2013 when he was transferred to the intensive care unit. William was intubated and ventilated. Blood tests at 6am revealed William had overwhelming sepsis.

The ICU consultant doctor and the paediatric intensivist worked aggressively together to improve William's condition including further inotropes, transfusion and intravenous Intragam. Despite this, William's condition continued to deteriorate. By 11.30am the doctors had exhausted all their means to rescue William's condition. After discussion with the family, ventilation was withdrawn and William died soon afterwards.

Blood cultures performed at JPHS were negative as were serological tests for atypical respiratory organisms and respiratory viruses.

Blood cultures performed at Townsville Hospital were negative for melioidosis.

Melioidosis

Melioidosis is caused by infection with the gram negative organism *Burkholderia pseudomallei* which is endemic in the soil of tropical regions. It is predominantly found in the Northern Territory and Northern Thailand where the annual incidence ranges from 5.8 – 80 cases per 100,000 population. It is far less common in northern Queensland with 1.3 – 3.7 cases per 100,000.

The organism is particularly active during the tropical wet season and more cases present after monsoonal type rains with occupational-recreational exposures recorded as a result of exposure to surface pools of water and mud.

Melioidosis presents predominantly in adults with overseas studies suggesting children under 15 years of age account for only 10-17% of cases.

The mortality rate in patients with septic shock is 80-95% despite treatment with appropriate antibiotics (ceftazidime, imipenem or meropenem).

A Queensland study reviewed all paediatric patients admitted to Townsville Hospital between 1996 and 2006. There were eight paediatric cases out of a total of 150 patients with confirmed melioidosis for that period (5.3%). There were only two recognised risk factors – one child was Aboriginal who had chronic renal disease due to systemic lupus erythematosus. There were two other Aboriginal children with no risk factors. The researchers noted that of all those with melioidosis (adult and children) Aboriginal people accounted for 30% of cases despite their population being 7 to 8% of the region. There is suggestion that the reason for the increased prevalence among Aboriginal people is their strong cultural relationship to the land, i.e. greater environmental exposure rather than any other particular factor.

Review of Treatment

When William presented to the clinic in Townsville he had just begun to develop a fever and upper respiratory symptoms. There was no evidence of bacterial pathology and the 'wait and see' approach taken by the doctor was sensible and correct.

On 7 February 2013 when William was taken to JPHS there was no reason for the doctor to suspect that he had pneumonia and the approach of having Ms Bligh monitor his symptoms and re-present if necessary was correct in the circumstances. The doctor documented that he would consider blood and urine tests if William re-presented. Most GP's in the circumstances would have made the same decisions.

When William re-presented on 11 February 2013 William's symptoms were still consistent with a virus and he seemed to be improving. In those circumstances the decision not to do blood tests at that time was reasonable.

On 13 February 2013 William saw a different doctor who 're-visited the illness from the beginning with a thorough clinical history and examination, including blood tests'. At that time William was well in himself, had no fever and normal oxygen saturations. Although not documented, it appears that a chest x-ray was ordered and the results (airspace shadowing in the left upper lobe of the lung, 'likely in keeping with infection') caused the doctor to prescribe antibiotics in the form of oral amoxicillin and azithromycin.

At this point the doctor was treating William's infection as community-acquired pneumonia.

William re-presented to JPHS on 20 February 2013 as he was not improving despite treatment for pneumonia. He was lethargic and had a dry cough; however, he remained bright with no respiratory distress.

His presentation, according to Qld Health guidelines, was that of moderate (non-severe) community-acquired pneumonia in a tropical region with no risk factors. The recommended treatment is benzylpenicillin intravenously plus oral roxithromycin which is what William received.

Dr Hall noted:

> *One could question whether William should have been commenced on a different regime at this point given that he had not responded or only partially responded to oral amoxicillin and azithromycin. A logical next step might have been ceftriaxione plus gentamicin as per the … guidelines … however the decision to use intravenous benzlpenicillin plus oral roxithromycin is within the Queensland Health guidelines and not an inappropriate decision.*

William continued on the new antibiotics for 48 hours and was reviewed regularly and remained stable. However, on the afternoon of 21 February 2013 and the early hours of 22 February he spiked two high temperatures. This is not an infrequent occurrence in cases of viral infection and infective processes, but it was after this that his condition began to deteriorate. The changes in his vital signs developed acutely and may reflect that the initial bacteraemia/septicaemic spread had commenced.

At this time initial contact was made with the paediatric registrar in Townsville who recommended the addition of flucloxacillin to the antibiotic regime to cover a staph infection.

Dr Hall noted, 'This is also the point where the doctors on Palm Island could have been advised to consider melioidosis as well.'

Later in the evening William began experiencing abdominal pains and left shoulder pains. In retrospect, it is likely that, at this point, septicaemic spread of the infection was occurring with seeding of infection within the spleen and abscess formation.

After a further 16 hours of treatment with no improvement it was decided to transfer William to Townsville.

Dr Hall noted:

> *Blood culture at that time was negative for atypical organisms and respiratory viruses and such would not have affected his management, except to say that melioidosis would not be suspected with the negative blood culture.*

On admission to Townsville Hospital William was quickly assessed by the paediatric team. Chest x-rays and CT scan established disseminated infection and liaison with the infectious diseases physicians resulted in William receiving meropenem and vancomycin to cover staphylococcal infection and melioidosis.

Dr Hall states:

> *William's treatment at Townsville hospital was excellent and appropriate antibiotics were commenced in a timely fashion*

> *…the outcome would have been no different had William been admitted directly to the ICU from Palm Island rather than go to the ward first.*

Sputum cultures did indicate melioidosis but these were not available until after William had died.

Dr Hall's Opinion of William's Clinical Management

Disseminated melioidosis is a rare cause of fatal pneumonia in otherwise healthy children. Why did William contract a severe form of the disease given that he was a healthy and active boy?

Dr Hall states:

> *It is accepted that healthy individuals can develop fulminant disease without underlying illness. William was diagnosed with ADHD for which he was taking methylphenidate (Concerta, Ritalin). He was also described as being a slight boy who was below the 3rd percentile of weight for his age. He also had a low grade anaemia. Methylphenidate is chemically a phenethylamine derivative similar to the amphetamines and to anorectic agents used in weight loss. It is recognised as an agent which can cause decreased appetite and weight loss in children, and caution is recommended in the prescribing of this drug that one should monitor the child's weight and nutritional status. There is no suggestion however that William's nutritional status was poor, thus it cannot be stated with any degree of certainty that methylphenidate contributed to William's susceptibility to the disease. It has been mentioned that there is a possible correlation between the virulence of B. pseudomallei and iron metabolism, so his anaemia perhaps could have put William at increased risk of serious infection, however work in this area is limited. Another factor in the development of severe disease is bacterial load. This has occurred during the wet season. It is well recognised that there is an increased risk of severe disease and faster incubation times in episodes of near drowning. There is a possibility that William may have been exposed to a high load of bacteria as a result from swimming in affected water or playing in mud etc, as many young boys of his age are wont to do after a deluge of rain.*

Dr Hall stated that he has no concerns with the management of William's illness in the early stages (7 February 2013) and his subsequent presentations on 11 and 13 February 2013 were also handled appropriately. The inpatient care of William on Palm Island was appropriate for a community-acquired pneumonia and he was given antibiotics in accordance with Qld Health guidelines.

William could have been treated with the addition of gentamicin and/or substitution of ceftriaxone for benzulpenicillin, however, even if this had occurred, there is no guarantee that the outcome would have changed. Unless melioidosis had been seriously considered at that stage (and there was no reason to do so) meropenem was not, according to the guidelines, a realistic first-line choice of antibiotic agent.

The revision of diagnosis and management after 48 hours, the consultation with the paediatric registrar and the resulting addition of flucloxacillin were appropriate, given the symptoms and circumstances.

Dr Hall states:

> *It is at this point that the diagnosis of melioidosis could have been considered, specifically because of location (geographically), the increased incidence in Aboriginal persons and the non-response to conventional antibiotic therapy, particularly as this is alluded to in the Queensland Health guidelines. The paediatric registrar from Townsville however has not considered melioidosis as a possibility at this point which implies that other causes (e.g. Staphylococcal) were considered more likely on clinical presentation at that time given that the incidence of Staphylococcal pneumonia is far greater than melioidosis. One needs to ask whether the commencement of meropenem at this point would have been life-saving in William's case, which is problematic. This is where his condition begins to deteriorate, and is most likely when he developed sepsis if not some time before.*
>
> *It appears clinically that there was a very narrow window of opportunity within which to potentially administer life-saving therapy and this was not recognised. It was not due to the lack of diligence or experience of the doctors involved, but more the presentation of the illness in William's case, and the absence of other risk factors which led to him not being given meropenem earlier … The doctors involved have followed Queensland Health guidelines to the letter in the treatment of William's pneumonia.*
>
> *Given the high mortality rate in melioidosis in the presence of sepsis (up to 95% in some studies), particularly with disseminated infection with abscess formation, I am of the opinion that had meropenem been commenced at this time the outcome would not have changed.*

Dr Hall noted the concerns raised by William's family in regard to his treatment on Palm Island, whether he should have been transferred to Townsville earlier and that medical staff on Palm Island did not seem concerned about William.

Dr Hall stated that in his opinion, there was no reason to transfer William to Townsville any earlier than was done so and, had he been transferred earlier there is no reason to believe that the outcome would have been any different.

Dr Hall noted that the perception that medical staff were unconcerned was not borne out in William's medical notes and by the fact that he was regularly attended to by staff for review.

Dr Hall concluded:

> *The family were obviously concerned with William and appropriately took him to JPHS for review. The attention he received at JPHS was appropriate and as good as (and some may argue, better than) what he would receive at any public hospital in Brisbane.*

Dr Hall noted that some concerns were raised in the press following William's death in regard to the employment of interns on Palm Island. Dr Hall's review of the medical notes revealed that there was only one intern employed on Palm Island and that person was not involved in the treatment of William. All of his presentations and management decisions were made by experienced medical staff. Inquiries have revealed that, other than the one intern at JPHS, the doctors have at least three years clinical experience as doctors. The intern duly documented the decisions made by her superiors in William's medical record on the formal ward rounds and her notes were well-written, legible and exemplary.

Dr Hall stated:

> *I would also like to comment that all the Queensland Health medical records including JPHS and Townsville were of an exceptionally high standard, such that to an external auditor the process of history taking, examination, and clinical decision making were easily identified, well structured and appropriate to the presentation described. I have no concerns that William received anything but the best medical care available to him.*

Review by Professor Currie

Background

Professor Bart Currie, Professor in Medicine and Head of the Infectious Diseases Department at Royal Darwin Hospital and of the Melioidosis research programs at the Menzies School of Health Research and Infectious Diseases Department of the Royal Darwin Hospital, provided an expert opinion in relation to the death of William.

Professor Currie has coordinated the Darwin Prospective Melioidosis Study at Menzies for the last 23 and a half years. Over that time he has documented all cases of melioidosis in the Top End of the Northern Territory. As of 1

October 2012, 42 of 785 total cases occurred in children less than 16 years old i.e. 5.4% of all cases with 48% of those being indigenous children. Of the cases in children 9% had an identifiable risk factor for melioidosis but these three children included all the fatal cases in children in the region over the 23 ½ year time frame. The rate of melioidosis in children is uncommon in the Northern Territory at 50 per 100,000 per year but is still considerably higher than the incidence of the disease in children in Townsville and Cairns.

Treatment at JPHS

Professor Currie found that the treatment received by William at Palm Island was appropriate, well-documented and in accordance with Qld Health guidelines for community-acquired pneumonia. The medical notes showed a functioning team of doctors with an appropriately supervised intern. The referral letter to Townsville Hospital was excellent, showed good clinical perspective and provided the appropriate information and level of concern.

In regards to time of referral to the Townsville Hospital, Professor Currie stated that even if William had been transferred to the Townsville Hospital on the day prior to the actual transfer and when deterioration was documented at JPHS, based on the clinical features, deterioration and the autopsy report, a fatal outcome would still have been very likely.

If he had been admitted to the Townsville Hospital on 20 February 2013 rather than JPHS and diagnosed with melioidosis and received specific melioidosis medication within 24 hours it is possible that he may have survived, however, even if he had been admitted it is unlikely that such a diagnosis would have been made given his negative blood cultures, the fact that autopsy blood cultures were also negative and that he had no identified risk factors or underlying chronic conditions which would alert doctors that he was predisposed to melioidosis.

Treatment at the Townsville Hospital

Professor Currie found that the treatment was appropriate with one proviso which was that the antibiotic, meropenem was prescribed at 3.40pm by the paediatric team but not administered until 8pm that evening. Professor Currie notes that this deficiency has been identified by the RCA report but that even if it had been given earlier it is his opinion that the outcome would not have been any different.

Conclusion

Professor Currie suspects that William had an unrecognised/undiagnosed predisposing condition affecting his immune system which would explain why he developed disseminated melioidosis which was eventually fatal. His treatment at Palm Island and Townsville hospitals was appropriate and even if he had been admitted to the Royal Darwin Hospital where the disease is more common it is likely that he would not have been diagnosed any earlier nor had a different outcome given his lack of recognised risk factors and the negative blood cultures.

Root Cause Analysis Report

The Queensland Health Department carried out a review of the clinical management and treatment of William at JPHS and the Townsville Hospital the outcome of which was a Root Cause Analysis (RCA) Report.

The report concluded that there were identified occasions in William's care where there may have been opportunity for further consultation and escalation may have provided an earlier opportunity of diagnosis confirmation and intervention. The review was unable to determine whether William's death would have been avoidable had earlier intervention and/or diagnosis occurred (however, according to Professor Currie it is very unlikely that earlier intervention would have resulted in a different outcome or that earlier diagnosis would have been possible).

The report identified the following issues:

JPHS

1. There is no formal introduction or specific training in tropical medicine for the JPHS medical staff;
2. The 'Children Early Warning Tool' (CEWT) when employed at JPHS did not identify William's emerging pathology;
3. There were no formal guidelines for referral and transfer to the Townsville Hospital;
4. No further blood analysis was conducted at JPHS;
5. There was a period in the 24 hours before transfer of recognised deterioration with new symptoms. That may have been an opportunity to escalate care.

Townsville Hospital Emergency Department

6. The 'Patient Access and Flow Health Service Directive' poses a genuine risk to the inter hospital transfer patient who is seriously unwell and where there is unspecified time between arrival at the receiving facility and ward team assessment;
7. The Rapid Assessment Team (RAT) process was not correctly followed with no notification to an Emergency Department consultant of William, his severity of presenting features and need for medical attention and subsequently, the results of interventions made by the RAT were not followed up;
8. William was not assessed further until review by the paediatric team 90 minutes after his arrival;
9. New antibiotics were prescribed at 3.40pm but not administered until admission to the paediatric ward. The first dose of meropenem was administered at 8pm.

Townsville Hospital Paediatric Ward

10. The admitting CEWT score was 9 and a score of 8+ recommends a Medical Emergency Team response;
11. Full vital sign documentation was not recorded on the CEWT form;

12. The CEWT did not identify William's emerging pathology and, as a consequence, the ongoing abnormal vital signs did not trigger escalation of other medical team care;
13. There was a disconnect between the severity of documented signs of abnormal physiology and the escalation of intervention;
14. Consultation with the paediatric intensive care service occurred when the child was in extremis.

The report recognised that due to subjective signs, such as William talking and taking fluids, clinical judgement was subject to confirmation bias which led to a lack of escalation of care.

It was recommended that:

1. Confirmation bias be researched further;
2. Training in regard to infectious diseases be revised;
3. Guidelines be developed for communication and consultation regarding paediatric admissions to the JPHS;
4. the RAT procedures be revised to provide guidelines on senior supervision, handover and maintenance of responsibilities when serious illness or instability is identified;
5. Procedures be put in place to ensure the early administration of antibiotics in acutely unwell patients;
6. The Therapeutic Guidelines be revised to more clearly reflect regional issues in relation to infectious diseases;
7. The state wide CEWT committee be advised of the issues identified when the CEWT was used in this case.

Although the RCA identified systems issues in relation to the treatment of William at the Townsville Hospital and intends to address those issues in the hope that such cases may be identified earlier in future, the conclusion to be drawn from the reports of Dr Hall and Professor Currie is that even had William been admitted to a tertiary facility earlier it is unlikely that he would have been diagnosed any earlier and also, that had he been given the specific antibiotic as soon as it was prescribed on 23 February 2013 the outcome for William would not have been any different.

Conclusion

I find that William John Bligh died at 12.08pm on 24 February 2013 at the Townsville Hospital. William died from disseminated melioidosis. His medical care and treatment at Palm Island and at the Townsville Hospital was appropriate.

Jane Bentley
Northern Coroner
Cairns
12 September 2013