QUEENSLAND COURTS

OFFICE OF THE STATE CORONER

FINDINGS OF INQUEST

CITATION:	**Inquest into the death of Thomas (Tom) Andrew Olive**
TITLE OF COURT:	Coroners Court
JURISDICTION:	Maroochydore
FILE NO(s):	2010/2935
DELIVERED ON:	5 August 2014
DELIVERED AT:	Brisbane
HEARING DATE(s):	11/11/2013, 3/3 – 7/3 2014, 14/3/14
FINDINGS OF:	John Lock, Deputy State Coroner
CATCHWORDS:	Coroners: inquest, health care related death, rhabdomyolysis, LPIN1 gene.

REPRESENTATION:

Counsel Assisting:	Ms M. Zerner I/B Office of the State Coroner
Olive family:	Mr Boyce, Solicitor, Butler McDermott
Sunshine Coast Hospital & Health Service:	Ms S Gallagher, Special Counsel, Corrs Chambers Westgarth
Drs Lee and Findlay:	Ms D Callaghan I/B Avant Law
Qld Ambulance Service:	Ms J Rosengren I/B QAS
Dr S Parsons:	Ms P Feeney I/B Moray Agnew Lawyers
Healthscope: Pathology:	Mr Schneiderwein I/B Minter Ellison

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Introduction

1. Thomas (Tom) Andrew Olive was aged four having been born on 6 November 2005.

2. In the two years leading to his death, Tom had sporadic symptoms of discoloured urine, sleeping a great deal and muscle/joint pains. Two general practitioner clinics and a paediatrician investigated these symptoms. In general, the assessments and investigations did not reveal any ongoing abnormalities; any abnormal results were noted to have resolved; and he appeared to be well.

3. On 30 June 2010, Tom complained he could not walk due to lower back pain. As a result his parents took him to Nambour General Hospital (the Hospital). He was not admitted to the ward. His parents reported that around this time he became very sensitive to light. Blood tests were conducted which appeared to be normal, other than that there were abnormal liver function results.

4. On 25 August 2010, Tom was complaining of pain in his knee, which then extended to other parts of the body. He was lethargic and deteriorated rapidly becoming very red and then non-responsive. Queensland Ambulance Service (QAS) was called. He was taken to the Hospital but deteriorated whilst in the Emergency Department (ED). Resuscitation was attempted but he could not be revived.

5. The cause of death found at autopsy was severe rhabdomyolysis[1] as a result of a LPIN1 gene mutation. Rhabdomyolysis generally is a breakdown of skeletal muscle, which can cause the release of myoglobin in the bloodstream and cause damage to the kidney cells. Symptoms can include abnormal dark urine colour, decreased urine production, general weakness, muscle stiffening, pain and fatigue. Although there can be multiple causes of rhabdomyolysis, relevantly in this case they can include metabolic and genetic factors.

6. The LPIN1 is a genetic abnormality causing skeletal muscle to break down and has been associated with sudden death in young children. It is an extremely rare disease, which up until Tom's death probably had never been described in Australia.

7. Tom's parents have expressed concerns in relation to the actions of medical staff, including GPs and paediatricians who had been consulted prior to the 30 June 2010.

8. They also expressed concerns as to the actions of QAS and hospital staff, querying whether or not their actions contributed to their son's death. The concerns relating to QAS and hospital staff principally relate

[1] A Glossary of Medical Terms can be found at the end of these findings. The terms in this finding are hyperlinked to the Glossary

to Tom's presentations to the Hospital on 30 June 2010 and 25 August 2010.

9. The concerns with respect to the presentation on 30 June 2010 principally relate to whether more should have been done about the symptoms Tom was exhibiting at that time, whether he should have been admitted to the ward and whether there was insufficient follow-up in regard to treatment and ongoing investigations.

10. The concerns with respect to the presentation on the 25 August 2010 relate to the actions of QAS and the ED medical and nursing staff in their responses to the medical emergency and alleged inadequate CPR when Tom suffered the cardiac arrest.

11. An internal review was conducted by the Hospital. As a result of a complaint lodged by Tom's parents with the Health Quality and Complaints Commission (HQCC), the HQCC gathered much evidence, including an expert opinion and statements from many witnesses. These were provided to the Office of State Coroner. Further statements and expert opinions were gathered. Upon review of this information, many aspects of what happened became clearer.

12. However, there was still some uncertainty concerning the circumstances leading up to Tom's death, and particularly whether there were earlier clinical signs, which should have alerted medical staff to undertake further reviews and investigations. Accordingly, I decided to hold an inquest into his death.

Issues for inquest

13. The issues identified at the pre-inquest conference to be explored at the inquest were:

- The findings required by section 45(2) of the *Coroners Act 2003:* namely; the identity of the deceased; when, where and how he died; and, what caused his death;
- Whether the assessment and treatment of Tom on 30 June 2010 by the Hospital was appropriate and adequate;
- Why a diagnosis of rhabdomyolysis was not made prior to 25 August 2010;
- Whether the QAS's management and handover of Tom to the Hospital on 25 August 2010 was appropriate and adequate;
- Whether the initial assessment and treatment of Tom on 25 August 2010 by the Hospital was appropriate and adequate;
- Whether the resuscitation of Tom by the Hospital on 25 August 2010 was appropriate and adequate;
- The adequacy of the policies and procedures of the QAS that relate directly to the care and treatment provided to Tom;
- The adequacy of the policies and procedures of the Hospital that relate directly to the care and treatment provided to Tom ; and

- The adequacy of the policies and procedures of the pathology company Healthscope that relate directly to the care and treatment provided to Tom.

14. After hearing the evidence, I received very helpful written submissions from a number of the parties given leave to appear. In particular, I have received comprehensive submissions from Ms Zerner, Counsel Assisting. These submissions set out in some detail the evidence heard in the inquest, and other than some disagreement as to how that evidence should be interpreted, there is largely no dispute as to the facts she has recorded. I have also received comprehensive submissions by Mr Boyce representing the Olive family and have paid close regard to these and the careful and balanced approach adopted.

15. Understandably, and given the contentious issues being addressed, not everyone will be happy with my conclusions about the evidence, but as best as I can, these findings seek to explain how the death occurred and consider whether any changes to policies or practices could reduce the likelihood of deaths occurring in similar circumstances in the future.

Autopsy results

16. The uncertainty as to what caused Tom's sudden deterioration and death became clearer after the comprehensive autopsy examination conducted by Professor Peter Ellis.

17. Tom appeared to be well nourished and there were no signs of trauma or any other non-natural condition.

18. Internally there was some congestion of the lungs and mucus in the airways. A macroscopic examination of all internal organs appeared normal to the naked eye.

19. X-rays of the body indicated they were mostly normal although showing a skeletal age slightly lower than chronological age.

20. Extensive further testing including toxicology, biochemistry, microbiology, metabolic and genetic testing was performed.

21. No cardiogenic bacteria or viruses were detected. Biochemistry showed no significant abnormalities. Toxicology did not find anything considered contributory.

22. A microscopic examination of the various organs showed mild non-specific abnormalities that were typical of sudden natural death. Special testing found lipid deposition in the liver, heart, skeletal muscle and kidney. Such deposition is often a feature of metabolic abnormalities.

23. Special chemical analysis of muscle revealed what was reported as changes resembling severe breakdown. Electron microscopy was performed on skeletal muscle and revealed abnormal mitochondrial structure.

24. A detailed analysis of DNA revealed two separate mutations in the structure of the LPIN1 gene. Specialist testing was undertaken in France. Two mutations, one received from each parent, act in combination to cause a fault in the structural integrity of many muscle cells. It was believed that such an abnormality can be responsible for the extensive breakdown of muscle cells, which was considered to be the underlying process behind Tom's collapse and death.

25. Testing on Tom's parents and younger sister showed that each one of them had one or the other of the mutations but not both.

26. Professor Ellis opined the cause of death was considered to be due to severe rhabdomyolysis as a result of LPIN1 gene mutation. LPIN1 gene mutation is a very rare genetic disease and this was the first reported case in Australia. The clinical features of LPIN1 include: severe life-threatening rhabdomyolysis and acute metabolic decompensation; normal strength, development and neurological examination between attacks; and normal fat distribution.

27. Professor Ellis stated that whilst rhabdomyolysis is a known phenomenon, the link between rhabdomyolysis and LPIN1 mutations is a relatively recent discovery and may have been discovered as recently as 2010. Triggers for rhabdomyolysis in LPIN1 include: fasting, fever, exercise, dehydration, overheating, intercurrent illness, or may occur spontaneously.

Evidence of the circumstances leading up to death

Medical Treatment in 12 months prior to death

28. In the 12 months leading up to Tom’s death, he had sporadic symptoms of discoloured urine and muscle/joint pains. Two General Practitioner Clinics and a paediatrician investigated these conditions at a number of consultations.

29. On 25 September 2009, Tom attended the Coastal Family Health Clinic where Dr Fiona McGrath reviewed him. Amongst other concerns, Tom’s parents were concerned about his gait when he was tired or walking up hill. A physical examination did not reveal any abnormalities. An x-ray of the lumbosacral spine and hips was ordered.

30. On 19 November 2009, Tom again attended the Coastal Family Health Clinic and was seen by Dr Alison Butler. Tom was reported by his mother to have pain on walking, localised to the right side of his back; was restless at night; had a possible temperature at night; and had dark coloured smelly urine. At the time of consultation he did not have an

elevated temperature, or evidence of pain on walking. A 'dipstick' urinalysis revealed blood +++ and protein+++. Dr Butler thought Tom may have a urinary tract infection. An urgent renal ultrasound was ordered. It was normal. A urine microscopy result revealed protein++ and blood+++ but no red or white cells. The pathology report stated "*Probable haemolysis of red blood cells".*

31. On 23 November 2009, Tom's usual GP, Dr Parsons of the Coastal Family Health Clinic reviewed him. The previous history was noted. Mrs Olive says she told Dr Parsons she was concerned something was wrong with Tom. She reported that the discolouration of urine quickly resolved and he had returned to his normal self. Tom was examined and no abnormalities were reported. Dr Parsons says he was confused about Tom's symptoms in the context of the pathology report and focused on a kidney issue. A biochemistry of his urine was ordered to check for increased calcium secretion, which can sometimes present as haematuria and back pain. It was normal and there were no blood cells in the urine. A repeat check confirmed the original results.

32. On 14 January 2010, a routine visit for a four year immunisation and health check was undertaken by Dr Parsons. Dr Parsons recorded in his notes that there had been no reoccurrence of dark coloured urine. Although there was some suggestion to the contrary, it is accepted he asked the necessary questions to make that note. The only concern expressed by Mrs Olive was that he was sleeping a great deal.

33. On 23 February 2010, Dr Parsons reviewed Tom as his parents had concerns about him having frequency in voiding, some increased drinking, bedwetting and some constipation. It was reported and noted by Dr Parsons that the urine smelt like an old man's urine. The examination did not reveal any abnormalities. A 'dipstick' urinalysis did not reveal any abnormalities. A urine sample was sent to the pathology lab. It was unremarkable. Dr Parsons put the symptoms down to the commencement of kindergarten being a stressful time, which can lead to constipation, and in turn, voiding issues. A complaint of smelly urine was a common one expressed by parents. Dr Parsons had not considered metabolic disorders. He did not consider it necessary to organize a CPK test as there were no features of rhabdomyolysis present when he examined Tom.

34. On 22 March 2010, Tom was taken to the Landsborough Medical Centre for review of right hip pain and 'funny' smelling urine. Dr Lee assessed Tom. On assessment she considered the hip pain had resolved but due to the reported history from his father, Dr Lee ordered a number of investigations, including blood tests, a MCS microurine and an x-ray of his right hip. Dr Lee recalls the urine being a normal colour and she does not recall smelling it. Dr Lee says Tom's father reported Tom had already had an ultrasound of his kidneys performed by his radiographer mother, and that it was normal. Dr Lee asked that Tom be brought back for further review in a few days time.

35. On 24 March 2010, Dr Lee received the pathology results. They showed elevated liver enzymes but were otherwise normal. Dr Lee discussed the case with her supervisor, Dr Vuocolo. She then called Dr Parsons. Dr Parsons says he was contacted by Dr Lee about this presentation and suggested he or a paediatrician review Tom, on the basis that although he thought it was viral, given there were repeating symptoms, a further review should take place.

36. Dr Lee discussed Tom with Dr Findlay, a paediatrician. Dr Findlay suggested further tests including an ultrasound of the liver. Tom's parents were notified and Tom was referred to Dr Findlay. Dr Lee thought Tom could be suffering a metabolic disorder but did not consider rhabdomyolysis as she would have expected discoloured urine and muscle aches and pains, with which he was not presenting. Dr Lee had not previously experienced rhabdomyolysis in a child.

37. On 7 April 2010, Dr Findlay reviewed Tom. Tom's mother reported him having brief hip pain possibly twice in the previous five months and persistent 'smelly urine', which she described as being the smell from an old person's home. She also told Dr Findlay that there had been brown/red urine and a sore back and legs.

38. Dr Findlay carried out a physical assessment and ordered a number of pathology tests and x-rays. No abnormalities were detected. Dr Findlay says his impression was that Tom might have had a virus, which had caused an inflammation of his joints. In relation to the 'smelly' urine, Dr Findlay says he was unsure of the cause but thought it remotely possible that Tom had an inborn error of metabolism.

39. Dr Findlay says as his assessment did not reveal any abnormalities and the investigations he had ordered were essentially normal, he wrote to Dr Lee suggesting that they adopt a 'wait and see' approach for any clinical change in Tom's health.

40. Dr Findlay said he had never treated a child with rhabdomyolysis and he would not have known the classic symptoms. After Tom's death, Dr Findlay had email contact with Dr McGill, an expert in paediatric metabolic disorders. Dr McGill said *it is clear that the high AST and ALT are muscle in origin and the normal LFTs that you ordered indicate that Thomas had intermittent episodes of rhabdomyolysis.*

41. On 30 June 2010, Tom presented to the Landsborough Medical Centre with lethargy, sore eyes and fevers. Dr Khan reviewed him. Dr Khan suspected a viral illness and suggested rest, clear fluids and analgesia. He advised, if the symptoms got worse to ring 000, or to take Tom to the hospital. Later in the afternoon Mrs Olive again attended and this time Tom had back pain, a temperature of 38.1 and he was unable to pass urine. Dr Khan referred Tom to the Hospital.

Presentation to Nambour Hospital on 30 June 2010

42. On 30 June 2010 at sometime after 5 pm, Tom was taken to the Hospital ED with a high temperature, sore throat, headache, and back pain. He had also complained of photophobia, which had settled. Tom had 'very dark red/brown' urine. His mother advised his urine had been dark before but investigations were always negative.

43. Tom was first reviewed by Dr Mark Coghlan by way of a rapid assessment. This is performed on patients who due to their assigned triaged category or other opinion may be in need of an early assessment and treatment. The aim is to detect immediate, acute problems which require rapid investigation and treatment. It is not a comprehensive and full clinical assessment and takes 5 to 10 minutes. Dr Coghlan does not have any specific recollection of performing the rapid assessment other than what is contained in the medical records. His notes did not make reference to being told by Mrs Olive that Tom was limping or having dark urine.

44. The rapid assessment did not indicate to him that Tom required admission to the paediatric ward for inpatient care. Tom did not look unwell; he had no worrying rash, neck stiffness or photophobia. He thought Tom was probably suffering a viral illness. A decision for admission would be made at the time of the more thorough assessment which followed a rapid assessment.

45. The more thorough assessment was performed by an intern, Dr Graham. A urine sample was taken and this appeared very dark/red. Dr Graham had been advised by Mrs Olive this had occurred before but results were negative for no reason. A 'dipstick" urinalysis revealed a large amount of blood and protein. Dr Graham found no tenderness on examination of the hips and legs. Dr Graham's impression was that Tom had pyrexia with an unknown cause but that he was not extremely unwell. She ordered a MCS microurine. This test ultimately did not reveal any abnormalities, but was not back by the time of discharge.

46. Dr Graham discussed Tom with her supervising consultant, Dr Todhunter. Dr Todhunter did not write anything in the medical notes and has no independent recollection of the event but I accept the discussion took place. Dr Graham reported in her progress notes:
    D/w Dr Todhunter:
    - given pt clinically well; don't have cause for fever however no urgent Sx/conditions apparent and is safe for discharge;
    - advised mum to return if Sx change/worsen, rash develops, increasingly unwell or other concerns;
    - f/u with GP later this week or earlier as required – letter – suggest repeat urine mcs when pt well to f/u ?haematuria
    - mum happy with above.

47. It is evident Mrs Olive does not agree she was 'happy' with the above at all. Dr Graham says she drafted a letter to Tom's GP, Dr Parsons. A

copy of the letter has been located in the medical records, but Dr Parsons says he did not receive it. Mrs Olive also says she did not receive the letter. Dr Graham has no recollection of handing the letter to Mrs Olive, although on her usual practice and as indicated in the records she says she would have given her a letter and discussed follow up.

48. Importantly in the conclusion of the letter, Dr Graham stated, *it may be worthwhile repeating his urine MCS following his acute illness, for resolution of haematuria.*

49. It is now evident the microscopy results became available at around 8:34pm, after Tom had been discharged. The results of the MCS were negative for red blood cells. The significance of this result is that urine tested positive for protein and blood on dipstick analysis. Further, the results in combination were not reviewed and therefore the possibility that the protein found may have been myoglobin, was not considered or appreciated by the Hospital.

50. The results of the MCS were sent to Dr Parsons. He said that as they were normal he would not have taken any further action. Dr Parsons, of course was unaware of the earlier dipstick analysis results and of the attendance at the ED. He had not seen Tom since February 2010 and by virtue of the conversation with Dr Lee was aware Tom was seeing another GP. However, if Dr Parsons had received the discharge letter, he says, and I accept, he would have put in place further investigations, perhaps a referral to a renal physician, as Dr Parsons would still have considered haematuria as a probable diagnosis.

51. Mrs Olive says Tom had no other symptoms such as aches and pains or discoloured or smelly urine after this presentation. She used her work facilities to conduct an ultrasound which showed no renal abnormalities. She did not take Tom back to any of the doctors as there were no abnormal symptoms.

52. Tom was not reviewed again by any medical practitioner until the day of his death.

53. The issue of what happened to the discharge letter is perplexing. I am convinced the content of the letter was inputted by Dr Graham. The letter reflects her notes and I have no reason at all to reject her evidence that she inputted it at the time for the purpose of providing a letter. However, it is likely, and for reasons that remain undetermined, the letter was not given to Mrs Olive. Mrs Olive wanted answers to the problems Tom was having, evident by her attendances at GPs and the Hospital itself. To suggest, in that context that she would ignore the suggestions of follow up referred to in the letter, simply does not add up.

Presentation on 25 August 2010

54. On the morning of 25 August 2010, Tom complained of a sore knee and that he could not walk. He was visibly in pain and clearly unwell as confirmed by a short video of Tom taken by his mother at the time. An appointment was made with Dr Parsons for later in the day. At Tom's request he was placed in a bath. He became increasingly lethargic. The bath was hot so his father added cold water, which splashed onto him. Tom did not react to the cold water. His father noted a red rash up Tom's shoulder, neck region and around his forehead and that his heart was racing. His parents checked his heart rate and it was 25 beats in 10 seconds. They decided to take Tom straight to Dr Parsons rather than wait for an appointment. However, as he continued to deteriorate, his parents called the QAS.

55. The QAS received the call from Tom's father at 9.43am. An ambulance was dispatched at 9.44am and arrived at 10.05am. Advanced Care Paramedic (ACP), Kenneth Crowley, and a third year student paramedic, Gary Lewis assessed Tom. ACP Crowley says Tom was pale, had a Glascow Coma Scale (GCS) of 10; a low temperature of 33.9; a fast pulse of 143; a blood pressure of 105/60, and a respiratory rate of 25.

56. ACP Crowley suspected sepsis and tried to warm Tom up, noting he had had a hot bath. A call for Intensive Care Paramedic back-up was made. QAS left at 10.18am as ACP Crowley said he was not going to waste any time. The paramedics met Intensive Care Paramedic (ICP), Linda Dow en route to the hospital.

57. ICP Dow met the crew at about 10.31am. She says Tom looked generally unwell (pale, diaphoretic, lethargic). He complained of abdominal pain but ICP Dow said he was able to respond to questions regarding 'Tom the Tank Engine'. His answers, whilst brief, demonstrated to her he was not confused. In fact, it may have been Tom's father who spoke about 'Thomas the Tank'. In any event, ICP Dow contacted the Hospital to notify them of Tom's pending arrival. She says she relayed his vital signs, pertinent history and stated that the patient appeared quite unwell. In a later statement she says she advised they had a *very sick four year old male*.

58. There is conflicting evidence as to whom ICP Dow spoke to at the Hospital and what was passed on. RN Nash says in a statement that he recalls a call coming in from QAS advising that they could expect the arrival of a sick child, possibly septic. Dr Ueno-Dewhirst stated he told RN Nash to warn RN Gelding, the triage nurse that they may require a resuscitation cubicle and for RN Gelding to inform him and Dr Ueno-Dewhirst of any specific concerns.

59. Dr Ueno-Dewhirst says he recalls the QAS telling him Tom had been unwell since the night before; complained of severe leg and back pains; had been placed in a bath to 'pep' him up after breakfast; had a seizure like episode whilst in the bath; was tachycardic, hypothermic, with an

altered GCS; his temperature was slowly improving and his GCS improved spontaneously in transit; their diagnosis was uncertain; and they gave an estimated time of arrival of 15 minutes. In a later statement Dr Ueno-Dewhirst says he does not recall if he took the phone call, nor the specific details relayed by the QAS. RN Nash says he does not recall if he told RN Gelding of the expected arrival of the deceased.

60. It is in fact likely the QAS call was taken by RN Menkins who also does not recall telling RN Gelding but told Dr Ueno-Dewhirst.

61. En route to the hospital, ICP Dow decided to undertake a 12 lead electrocardiograph (ECG). For this purpose she requested the driver slow down and downgraded the job to a Code 2. I am satisfied this was a reasonable decision; occurred a few minutes out from the hospital; and made little practical difference in the time of arrival or the outcome.

62. The ECG showed sinus tachycardia with mildly peaked 't' waves in the lateral leads. ICP Dow says the one thing that can be picked up on an ECG is hyperkalemia (high potassium). In her evidence she said she did not see significant changes indicative of hyperkalaemia. This view was confirmed by a number of other witnesses (Dr Jensen, Dr Kelly) who were asked to look at the ECG and opined the ECG did not show obvious hyperkalemia or anything of acute concern.

63. There was some contention raised about the ECG and whether the results were raised with hospital staff, given it will become clear that Tom was very likely hyperkalemic on arrival.

64. ACP Crowley and ICP Dow cannot recall if the ECG was mentioned to the hospital staff. RN Gelding, the triage nurse does not recall being advised of an ECG or discussing an ECG. Hence it is likely the ECG results were not considered, but as they did not indicate hyperkalemia, this made no difference to the outcome.

65. There were also clearly differences of opinion by QAS and hospital staff as to Tom's condition on arrival. The evidence of ACP Crowley was that there had been some improvement in Tom's condition by the time of arrival at the hospital. This view is reflected in the QAS electronic Ambulance Report Form (eARF) completed by ACP Crowley. ACP Crowley says he was not surprised that Tom was not being allocated a resuscitation bed. ACP Crowley says he had no issue with the child being moved to the acute area and says it was a few minutes later that Tom was moved to the resuscitation bay. ACP Crowley recalls Tom being fully alert and laughing when they wheeled him in.

66. Student paramedic Lewis says the child had improved since they left the house and that he was talking about his motorbike and Tom the Tank. He says when they unloaded Tom he seemed a lot better.

67. ICP Dow had a quite different view and conveyed to the hospital on the

way that an unwell child was being brought to the hospital. She completed a separate eARF. She refers in the eARF to Tom being lethargic, limp, pale diaphoretic and cold. She reports a 12 lead ECG showed sinus tachycardia with peaked 't' waves in the lateral leads. She notes the history of pungent smelling urine.

68. The triage nurse, RN Gelding assessed Tom on arrival to the hospital. RN Gelding says he did not have any prior notice of the arrival to the ED. That is probably the case. He assessed Tom as being awake and alert and that his vital signs were essentially normal. He considered he was stable and categorised him at a level 3 (to be seen within 30 minutes). RN Gelding liaised with the shift coordinator and it was decided Tom would be transferred to acute bay 7.

69. ICP Dow says she recalls the triage nurse being advised the child was quite sick and she was surprised that he was not moving directly to the resuscitation area. ICP Dow says they were told a resuscitation bed was not available at that time and that an acute bed was available. She says from her memory the nurse suggested a doctor would also be in attendance shortly. ICP Dow did not consider there was any change in Tom's condition from when she started caring for him. She says he barely responded when she attempted to put an intravenous cannula in, which she says was unusual.

70. RN Nash says he took one look at Tom and thought he was too unwell to be put in bay 7 so requested he be moved straight into a resuscitation bed. Tom was off loaded from the Ambulance trolley there.

71. RN Sinclair says she reviewed Tom in bay 7. She recalled he had a vacant stare. She left Tom in the care of the paramedics whilst she went to retrieve a paediatric oxygen probe. When she retuned Tom had an even more vacant stare, a steady pulse, and an unobtainable blood pressure. She seems to have made a decision to move him into the paediatric resuscitation bay (rather than RN Nash). RN Sinclair then got the attention of RN Menkins, who agreed to make contact with Dr Ueno-Dewhirst.

72. In considering the evidence, it is more likely than not that RN Gelding's assessment of Tom was influenced by the information provided to him on arrival that Tom had improved. RN Gelding had not been given the information relayed in the call by ICP Dow. With the benefit of hindsight, the rapid deterioration soon after arrival suggests this was a mistake. Dr Ueno-Dewhirst says the category 3 was unusual but the triage nurse was experienced and had seen the child. Dr Ueno-Dewhirst said he was advised of the child's improved state in transit and told Dr Jensen (who had been on standby) of Tom's arrival and results of the triage assessment. Dr Ueno-Dewhirst was in the tearoom when he was notified of Tom's deterioration and Dr Jensen offered to immediately go to the resuscitation bay to review the child.

73. On transfer to the resuscitation bay, telemetry was placed on Tom. This showed a QRS complex, which signified that something had changed. Whilst trying to obtain a 12 lead ECG, Dr Jensen arrived to assess Tom. Tom became very pale, his eyes rolled back, and he became unresponsive. His father moved to do a couple of chest compressions. RN Sinclair pushed the emergency button and then took over compressions. Dr Jensen managed the airway. Dr Jensen called for the correct size mask, as he could not locate one (he was using an adolescent mask). Dr Jensen called for this on a couple of occasions. Dr Jensen is of the view he was able to administer appropriate airflow prior to being given the correct mask.

74. Many people attended the emergency. Tom's parents obviously found it chaotic and clearly distressing.

75. Dr Jensen says he used the drug suxamethonium chloride (muscle relaxant) to help in intubating Tom. Dr Jensen says he was not aware Tom was suffering hyperkalemia when he administered suxamethonium (suxamethonium is contraindicated with patients with severe hyperkalemia).

76. Student Nurse Cripse did a 'dipstick' urinalysis whilst the resuscitation was in progress. She recalls it being abnormal and reporting it to RN Sinclair. The results were not documented.

77. Intravenous access was difficult. Dr Ueno-Dewhirst used the intraosseus drill to gain access to Tom's right tibia.

78. Dr Hurley, the Director of Paediatrics at the Hospital, and his registrar, Dr Cohn attended the resuscitation. The medical staff present consulted with each other to try and establish a cause for the arrest.

79. Blood results eventually came back, which revealed a severe metabolic acidosis, which included a very low bicarbonate and severe hyperkalemia. Calcium chloride and insulin/dextrose were added to the IV line, without any improvement.

80. Dr Jensen surmised because of the high potassium level, Tom had an unknown severe genetic muscle metabolic disorder. Dr Ueno-Dewhirst says because of the high potassium the working diagnosis became an unknown severe genetic metabolic disorder.

81. The resuscitation lasted for approximately 50 minutes.

82. Dr Ueno-Dewhirst says Tom was a young child who was very ill but did not display symptoms pointing to the possibility that he was likely to arrest within 30 minutes of arrival at the ED.

83. Although to determine the precise timings of the sequence of events is difficult, it is probable Tom arrived at the ED at around 10.45 or possibly

a few minutes later, and arrested around 30 minutes later. Although he was triaged as a category 3, in fact the decision to take him to the resuscitation bay and effectively upgrade Tom to a category 2 took place sometime after 11.05 and before the arrest at 11.15.

84. A category 2 requires a medical assessment within 10 minutes. Even if Tom had been triaged Category 2 on arrival, given some time needs to be taken for the triage assessment itself, the delay in Tom being medically reviewed was in the order of 5 to 10 minutes. The evidence suggests it was already too late for Tom by this time, as the potassium levels were likely to already be at unsurvivable levels.

Internal reviews

Hospital review

85. The ED undertook an audit into Tom's death. A Root Cause Analysis was not conducted, as the death was not considered a reportable clinical incident. An ED Action Plan was created. Three aims were identified in the review with various actions established to address the aims. The aims with the corresponding actions include:

1. To consolidate discharge process in the Department of Emergency (DEM)
   Action:
   - Specific information relating to this is contained in the District DEM orientation book for medical staff;
   - The medical orientation document to be modified to provide a summary for short stay staff outside of usual orientation schedules. Summary to include the essential requirements of documentation & GP communication;
   - Additional prompts for staff to be included in the patient documentation;
   - Further reminders to be published in poster format and situated around the department;
   - Patients will be issued with a prompt alerting them to request a discharge summary before they leave the department;
   - Written and verbal communication with the patients GP to be recorded in the patient chart;
   - Audit of compliance to be added to the clinical coders' role.

2. To increase the Department's capacity and preparedness for emergency paediatric cases.
   Action:
   - DEM nurses to attend an Emergency Paediatric Nursing workshop. This will augment the skills and increase the number of nurses with this particular knowledge;
   - An area in the DEM will be designated and equipped particularly for dealing with Paediatric emergency medicine;

3. To improve alerts, communication and follow up regarding test results.
   Action:
   - Communication and Liaison with Pathology to identify a process for an alert;
   - Procedure for an alert system to be established.

86. The Hospital has advised that all actions under Aim 1 have been completed. In relation to Aim 2, the Hospital expected that 95% of staff

would have completed specific training on paediatric resuscitation by 2014. As well, an area has been designated and equipped for paediatric emergency medicine.

87. In relation to Aim 3, the Hospital advised that a number of changes to pathology reporting and communication had been made. This includes the introduction of a policy whereby all pathology results are reviewed, including discharge patients, and the medical officer must document that the investigation results have been reviewed and that appropriate action has been taken for significantly abnormal results.

Review by QAS

88. The QAS undertook a review of the case using its internal audit program CART, and considered there was no deviation from clinical practice and standards.

89. Dr Ben Clarke, the acting Medical director of the QAS was of the opinion, the expected processes were followed in the pre-notification to the Hospital and that standard triaging and handover process occurred. He stated he was confident all paramedics in attendance provided a high level of clinical assessment and care and recognised a potentially significant underlying illness or condition. This is evidenced by the ACP crew requesting ICP backup, proceeding to the Hospital under code 1 (lights and sirens), and from ICP Dow's statement in which she describes a discussion with the triage nurse about placing Tom in a resuscitation bay. Dr Clarke stated the nature of pre hospital medicine does not always allow for a clinical diagnosis to occur. The clinical features of Tom's presentation were managed appropriately and he was of the opinion that such a devastating clinical course could not have been predicted by paramedics.

90. It is evident in this case that the eARF completed by ACP Crowley was provided to the hospital (it being in the records), but would not have been even completed (and hence not considered) by the time of Tom's arrest shortly after arrival. The evidence also indicates that the eARF completed by ICP Dow was not received by the hospital. For similar reasons, this fact had no contribution to the outcome, but QAS has acknowledged in the submissions made by its Counsel, Ms Rosengren, that the best practice approach would be for a hospital to be provided with the eARFs completed by both ACPs and ICPs.

91. It is also evident from the evidence that much of the information needs to be manually inputted and it was questioned as to whether some of the data could be automatically recorded, allowing paramedics to direct their attention to patients without the burden of repeated note taking.

92. QAS has advised that there are currently development activities underway to design and create a new electronic patient record system to consolidate multiple records relating to the one patient in a single patient record, which will be electronically transmitted and submitted to

the receiving facilities. It was anticipated that this electronic record system would be implemented within the next 12 to 18 months.

93. Further work has commenced to roll out an eARF/CAD (Computer Aided Dispatch) interface. The Interface allows CAD to automatically populate the document with certain information such as incident number, incident address, patient name and case times. It was also advised that a new version of the eARF was being developed and will have improved functionality to allow for automatic vital signs data upload. It is expected that this will capture more information in a more efficient manner.

Expert opinions

Dr Anne-Maree Kelly, Emergency Physician

94. Dr Kelly has provided a detailed and comprehensive report addressing the issues and the Olive family's concerns.

95. Dr Kelly believes the care provided to Tom on 30 June 2010 was appropriate. None of the doctors had access to Dr Findlay's correspondence with Dr Lee or the results of the previous tests. She said whilst there was no attempt to contact Dr Findlay she does not see this as a serious deficiency and says it reflects the common challenges of obtaining information in the ED. Despite opinions expressed by Dr Hurley (paediatrician at the Hospital) and Dr Priestly (Director of the Hospital Emergency Department), she does not consider the decision to discharge Tom was unreasonable. She considers Dr Hurley and Dr Priestly are approaching the case with hindsight. Tom's symptoms resolved, he had a diligent GP and was being followed up in the community.

96. Dr Kelly was unable to provide comment on what the actions of Dr Findlay or any of the GPs would have been should they have received the discharge letter of 30 June 2010 from the Hospital. It is possible it may have prompted further investigations. Dr Kelly said in evidence that had Dr Parsons received the discharge letter, it coupled with the microscopy results would have been complete information in the circumstances. However this is in the context that the dipstick-positive red-cell – negative information was not linked on the result.

97. Dr Kelly stated that her hospital had the same system as the Hospital concerning discharge letters in that they are provided to the patient or carers directly and are not sent electronically or otherwise to a patient's GP. It is then up to the patient to decide whether they want to have a follow-up.

98. Dr Kelly says there is no way of knowing whether Tom would have had a different outcome had he presented to the hospital earlier.

99. In relation to the triage assessment on 25 August 2010, Dr Kelly says,

with the benefit of hindsight, ATS category 3 may seem too low but says RN Gelding's logic was reasonable. Dr Kelly says she would have probably preferred category 2 based on the episodes of unresponsiveness that occurred and the uncertainty about what was going on. She confirms there were no grounds for Tom to be triaged as a category 1 (immediate threat to life).

100. Dr Kelly stated that it is evident Tom's condition deteriorated and RN Sinclair identified this and escalated his care and arranged for a senior doctor to urgently assess. Dr Kelly says from the material, she is of the opinion that staff recognised that a cardiac arrest occurred immediately; that resuscitation was commenced quickly; that it followed standard protocols; and was conducted according to relevant professional standards.

101. Given the high potassium level subsequently revealed in the tests, Dr Kelly was of the opinion Tom would have had a pre-arrest potassium level of more than 8. Arrhythmias can occur with a level of 5 to 6 but are more common in cases of 8 or 9. Dr Kelly was of the view that given this pre-arrest potassium level, and on the basis he arrested 10 minutes after arrival at Bay 7, it was very unlikely anything could have been done that would have resulted in a different outcome.

102. With respect to the QAS ECG, Dr Kelly said it did not indicate any need for urgent treatment and it does not demonstrate obvious hyperkalaemia.

103. Dr Kelly suggests ICP Dow may have completed her eARF with hindsight. She mentions the ECG and that it was interpreted as having sinus tachycardia with peaked T waves. However, Dr Kelly says there is no evidence the results of the ECG were communicated to the Hospital staff**.**

104. Dr Kelly says there was a failure to recognize that the urine, which tested positive for blood on a 'dipstick', did not have the presence of blood cells. She says this is probable because the clinicians were focused on haematuria and the possible causes of haematuria. She says at least five samples were sent to the pathologist in the months prior to Tom's death and the pathologist failed to notice the pattern and advise the treating clinicians, possibly prompting further testing.

105. Dr Kelly says the seminal paper identifying the LPIN1 gene mutation in children was only published in July 2010. Dr Kelly is of the view the journal would not usually be read by clinicians without a specific interest in genetic disease. She says it would be her expectation that the vast majority of paediatricians and probably all emergency physicians and GPs, were unaware of its existence at the time of the Tom's death.

106. Dr Kelly stated that she had never seen a child present with rhabdomyolysis, nor has she seen a case of spontaneous rhabdomyolysis in an adult. Her experience with rhabdomyolysis has

been in the context of trauma patients.

Professor John Christodoulou, Paediatrician

107. Dr Christodoulou opines the result of positive blood on a urinary dipstick, but no red blood cells on microscopy, with a history of dark coloured urine should have raised a suspicion that further review and investigations might be warranted. This would have included measuring the CPK level. In evidence he said that such action would not necessarily have been indicated on the first presentation, but would need to be considered with a recurrent presentation. He says low back pain is not classical of rhabdomyolysis, and he would not suspect the average GP to be aware of it.

108. Dr Christodoulou says if Dr Findlay had been aware of the preceding history or dark urine that subsequently cleared, in light of the presentation, he should have been alerted to the possibility of acute rhabdomyolysis.

109. Dr Christodoulou did not consider symptoms of sleeping or being more tired or the urinary and constipation symptoms as ones which would make him consider the possibility of rhabdomyolysis. The usual symptoms are muscle pain and some irritability with the pain more associated with muscle and muscle tenderness rather than hip or back pain.

110. He also said that with the benefit of hindsight the elevated liver function test, in particular the elevated AST and ALT and the ratio between the two, could potentially raise a suspicion of rhabdomyolysis but he did not think a general practitioner would be cognisant of that. The results were potentially also consistent with a viral illness.

111. Dr Christodoulou considered Dr Lee's provisional diagnosis of a viral illness and referral to a paediatrician was appropriate.

112. In relation to the presentation on 30 June 2010, Dr Christodoulou says if Hospital staff had the microscopy results prior to discharge, and were aware of the history of a previous episode of dark coloured urine with no blood cells, they should have considered the possibility that something else, like acute rhabdomyolysis, was at play. On the basis the MCS result was not available, the decision to discharge would have been reasonable, provided there was a mechanism for follow-up put in place that actually occurred.

113. Once a diagnosis of rhabdomyolysis is made, there would be a lengthy period to find out what was causing this.

114. Dr Christodoulou states: *the finding of acute hyperkalaemia during the resuscitation phase might have given a clue to the possibility that the clinical presentation might have been a consequence of acute rhabdomyolysis, but even if that was considered at the time of the*

resuscitation (and a CPK test was performed), I do not believe that it would have altered the acute management that needed to be implemented in a bid to save Tom's life. He stated that the elevated potassium was likely to reflect a high potassium level and not an artificial level because of haemolysis. If the level of 9 was real it would have caused a severe cardiac arrhythmia and he had never heard of anyone surviving with a potassium level of 9.

115. Dr Christodoulou says if rhabdomyolysis had been previously entertained and confirmed by measuring CPK levels at the time of acute pain, the further investigations would have occurred. Even if LPIN1 had not been diagnosed, the specific measures could have been put in place for managing the rhabdomyolysis, therefore possibly avoiding a catastrophic event.

Dr Brian Kable – General Practitioner

116. Dr Kable reviewed the medical records and considered that Tom was not presenting as a sickly child.

117. Dr Kable stated it was rare to see a comment *probable haemolysis of red blood cells* on MCS results. He stated that Dr Parsons did carry out the necessary tests in an attempt to work out the cause of the haemolysis although the most probable explanation was haemolysis of the erythrocytes.

118. Dr Kable did not consider there was a sufficient pattern of presentations and symptoms to conduct CPK or myoglobin tests as none of the symptoms were referable to Tom's large muscle groups. He considered it was reasonable to proceed on the basis the discoloured urine in November 2009 was as a result of haematuria.

119. He stated that if he had received the MCS results from the Hospital of 30 June 2010, it would not have caused him to take any action. The result would not raise any issues and Dr Parsons had no way of knowing the result of the urine dipstick or the colour of Tom's urine at the hospital.

120. He considered the tests ordered by Dr Lee were reasonable and the elevated liver function tests were consistent with a viral infection. The referral to Dr Findlay was also appropriate.

121. Dr Kable has also never seen rhabdomyolysis in a patient and would never have considered it in this case because there were no symptoms in the large muscle groups. He was of the opinion Dr Parsons and Dr Lee acted reasonably and appropriately in their management.

Conclusions on the issues

122. In reaching my conclusions it should be kept in mind that a coroner must not include in the findings or any comments or recommendations, statements that a person is or maybe guilty of an offence or is or

maybe civilly liable for something.[2] The focus is on discovering what happened, not on ascribing guilt, attributing blame or apportioning liability. The purpose is to inform the family and the public of how the death occurred with a view to reducing the likelihood of similar deaths.

123. If, from information obtained at an inquest or during the investigation, a coroner reasonably believes that the information may cause a disciplinary body for a person's profession or trade to inquire into or take steps in relation to the person's conduct, then the coroner may give that information to that body.

124. In matters involving health care, the impact of hindsight bias and affected bias[3] must also be considered, when determining the significance and interpretation of the evidence.

125. In my experience, where there are negative medical outcomes, there is often evidence of poor communication that contributes, and usually not just one event but a number of such events. As a result, critical information is lost, not communicated, or falls between the cracks and is therefore not considered.

126. There is evidence of such poor communication in this case, resulting in some missed opportunities to diagnose that Tom was suffering from rhabdomyolyis. That being said, any capacity to make a diagnosis of rhabdomyolysis was compounded by Tom having a virtually unknown underlying condition and most of the time displaying atypical symptoms.

127. There can be no criticism of the medical practitioners involved in Tom's care for being unaware of the LPIN1 gene mutation.

128. Similarly, none of the medical practitioners and even independent experts had come across spontaneous rhabdomyolysis in a child, or an adult for that matter. Therefore this condition was never going to be one that was high on their levels of suspicions as a differential diagnosis. Emergency experts were more likely to have seen the condition, but usually in the context of trauma, exposure and snake bite.

129. Although it is not beyond all doubt, it is likely Tom had acute episodes of rhabdomyolysis in November 2009, March 2010 and 30 June 2010, from which he quickly recovered. Once the episodes were over, testing to diagnose rhabdomyolysis would have been problematic, but had relevant testing been conducted at the time of the event, a diagnosis might have been possible. As Ms Zerner stated in her submissions, the evidence suggests that had a link been made it is possible Tom would have been sent for further investigation, including metabolic testing. Almost certainly the LPIN1 gene mutation would not have been

[2] s 45(5) *Coroners Act 2003*

[3] Where after an event has occurred, particularly where the outcome is serious, there is an inclination to see the event as predictable, despite there being few objective facts to support its prediction.

discovered by 25 August 2010, but as Dr Christodoulou said, specific measures could have been put in place for managing rhabdomyolysis, therefore possibly avoiding a catastrophic event.

130. In hindsight, the referral to Dr Findlay, a paediatrician lately more experienced in child behavioural issues, would have been better addressed to a paediatrician experienced in metabolic disorders. However, neither Dr Lee nor Dr Findlay was to know Tom's condition was metabolic in origin.

131. With the benefit of hindsight, in the period up to 30 June 2010, it could be argued there were other clinical indicators, such as the earlier contrary tests results, which could have resulted in further investigations, but I do not consider these were missed opportunities as such. Intermittent spontaneous rhabdomyolysis is a rare condition and Tom's presenting symptoms were atypical of the condition. Up until 30 June 2010, in my view, there can be no criticism of the efforts being made by the GPs and Dr Findlay.

132. The most significant missed opportunities occurred on and after 30 June 2010. On 30 June Tom was very likely experiencing an acute episode of rhabdomyolysis. The dipstick result and the later MCS results became available that evening, but the significance of the contrary results, in conjunction with the previous history, was not considered by anyone, let alone linked. A CPK test performed that day may have diagnosed rhabdomyolysis and further testing could have commenced.

133. Dr Hurley, the Director of Paediatrics at the Hospital reviewed the medical chart and in particular saw that the urine sample sent to the laboratory was contrary to the results of the dipstick tests in that no red blood cells showed up in Tom's urine. Given this was an acute presentation and there were no red blood cells in the laboratory tests, his opinion was that Tom should have been admitted as a paediatric admission to have further investigations. If Tom had been admitted, the path of testing and further investigations would have commenced. In this respect Dr Stephen Priestley, the Director of the ED agrees. Dr Hurley did note that it takes weeks for genetic testing to occur, and even if this commenced, the final results may not have been known by 25 August 2010 and the outcome would not have been different. Dr Hurley however stated that nonetheless, there remains a possibility that Tom's death may have been avoided.

134. Dr Kelly and Professor Christodoulou are not so critical of the decision to discharge, provided there was a mechanism for follow-up put in place that actually occurred. The proposed follow-up was set out in the discharge letter suggesting to Dr Parsons that he may consider repeating the urine MCS following Tom's acute illness. The letter was not given to Mrs Olive and the letter was not sent to Dr Parsons. No-one rang or checked with Dr Parsons as to whether he had done anything about follow up. The critical follow-up expected by Dr Kelly and

Professor Christodoulou was not put in place.

135. To compound the non-communication of the follow-up plan, Dr Parsons received the results of the MCS, which on the face of it were essentially normal. Without the knowledge and context of the acute admission to the ED and the contrary dipstick result, he, not unreasonably, took no further action. If he had been made aware, Dr Parsons said he was likely to have made further investigations, albeit commencing with a renal focus. Again it is uncertain that a diagnosis of rhabdomyolysis would have been made before 25 August 2010, let alone that there was a metabolic basis for the condition, but this remains a possibility.

136. There was a further missed opportunity when Hospital staff failed to review the MCS result at all. A review of both results could have made a diagnostic link that something was occurring, and recalled Tom for review or contacted his GP.

137. On 25 August 2010, Tom was in the throes of an acute episode of rhabdomyolysis. Dr Hurley stated it was likely to have been occurring for up to 24 hours. It was in fact too late for Tom by the time he got to the Hospital. An earlier presentation, with the knowledge of his condition, and therefore attending to it as a medical emergency and providing the immediate care appropriate to a diagnosis of rhabdomyolysis, may have made a difference and he could have survived. Such emergency treatment plans are now in place for other family members with the LPIN1 gene mutation.

138. QAS proceeded to take Tom to the Hospital in accordance with good practice. Information was passed en route advising of Tom being unwell. The information was passed on to the senior medical staff and there were plans put in place to review Tom after arrival. That information was not passed on to the triage nurse. It should have.

139. There was some difference of opinion between the QAS paramedics as to whether Tom had improved en route. It appears that the opinion there was some improvement was the one handed over at triage and this influenced the decision to a category 3 requiring medical review in 30 minutes.

140. There is some question as to the appropriateness of the category 3 assessment in that it should have been a category 2, although hindsight might play a role in such fine judgements now. However, other staff quickly recognised the severity of Tom's condition, escalated and sought an immediate medical review and brought him to the resuscitation bay. The delay was in the order of minutes, and was regrettable, but this did not contribute to the outcome. There was no handover of the ECG results taken in the ambulance. There had been some suggestion there were elements of the ECG suggestive of hyperkalaemia, but no-one now says this is the case and the lack of handover of the ECG results is therefore not contributory.

141. When Tom arrested, advanced resuscitation commenced. I accept the Olive family's impression was that it was unorganised, chaotic and therefore the Hospital failed. The evidence does not support that conclusion, but emergency resuscitations are stressful events and Mr and Mrs Olive would have had no previous experience of them. The administration of suxamethonium was reasonable given there was no suggestion at that stage Tom had high potassium. The expert opinion, in conjunction with a consideration of the evidence, supports a conclusion that the resuscitation was appropriate.

Findings required by s. 45

Identity of the deceased – Thomas Andrew Olive

How he died – Tom died as a result of a rare metabolic gene mutation causing rhabdomyolysis. There had been previous acute episodes of the condition in the months leading up to his death. There had been a number of missed opportunities to diagnose the condition and put in place a treatment and management plan, particularly if such an event occurred again. It is uncertain, given the rarity and complexity of the condition, whether if such missed opportunities were availed of, the outcome would have been different, but it is certainly possible.

Place of death – Nambour General Hospital, Hospital Road Nambour Queensland 4560

Date of death– 25 August 2010

Cause of death – 1(a) Severe Rhabdomyolysis
1(b) LPIN1 Gene Mutation

Comments and recommendations

There was evidence given about policy and procedure issues. The matters concerning QAS and the distribution of the eARF is being addressed by QAS and does not require any further comment. Enhancements to the eARF system are being developed to provide for the automatic inputting of incident and clinical data.

I make no adverse findings or comments in relation to the Healthscope policies with respect to the reporting of the pathology result in this case.

The Hospital has made changes to its procedures in a number of respects, which may address the issues of distribution of discharge letters and the review of patient pathology results. Whilst policies are all very well, such matters should in reality be matters of sensible and good clinical practice.

What is important is that such principles are adhered to in practice. This is not the first case I have been involved in where similar issues have arisen and recent media has highlighted concerns about widespread failures to review thousands of X-ray results at another hospital in South East Queensland. It should not require a policy for obvious good sensible practice to occur.

I express my condolences to the Olive family. They have been actively advocating on behalf of their son so that if changes can be made, his death in these circumstances was not in vain. They clearly miss him. Through their efforts they have brought to the attention of the medical world in Australia this rare condition and how it can be treated and managed.

I have found there were a number of missed opportunities where potentially a link could have been made that led to a diagnosis before Tom died, but this is not certain. Tom's family did everything they could.

I close the inquest.

John Lock
Deputy State Coroner
Brisbane
5 August 2014

Glossary of medical terms

Dipstick urinanalysis – a basic tool to determine changes in a patient's urine, and tests for the presence of proteins, glucose, ketones, haemoglobin, bilirubin, urobilinogen, acetone, nitrite and leucocytes

Haematuria – the presence of blood in the urine; can be a symptom of urinary tract infection

Haemolysis – destruction of red blood cells

Hyperkalemia (high potassium) – If very high can be a medical emergency due to risk of arrhythmia. It can be caused by rhabdomyolysis. ECG findings (development of peaked 't' waves for instance) can be an indicator of hyperkalemia. ECG findings are not definitive or reliable to diagnose hyperkalemia and need to be confirmed by blood tests

LPIN1 gene mutation is a very rare genetic disease. It was only reported in the medical literature in 2008. The clinical features of LPIN1 include: severe life-threatening rhabdomyolysis and acute metabolic decompensation; normal strength, development and neurological examination between attacks; and normal fat distribution

MCS Microscopy Urinanalysis – A medical diagnostic test that examines urine under a microscope and is used to assess the condition of the kidney and urine

Myoglobin – protein found in muscle tissue and related to haemoglobin (a protein found in red blood cells). Myoglobin is only found in the bloodstream after muscle injury. Relevant to this case, the finding on dipstick of protein and blood and no finding of red blood cells on MCS may be suggestive of haemolysis; but alternatively, could be diagnostic such that the protein was in fact myoglobin produced during an acute episode of rhabdomyolysis

Rhabdomyolysis is the breakdown of skeletal muscle, with the disruption of the muscle cells. The breakdown of the muscle causes the release of myoglobin into the bloodstream, which can cause damage to the kidney cells. The symptoms include:

- abnormal urine colour (dark, red, or cola colour);
- decreased urine production;
- general weakness;
- muscle stiffness or aching (myalgia);
- muscle tenderness;
- weakness of the affected muscles;
- fatigue;
- joint pain;
- seizures; and
- weight gain.

The causes of rhabdomyolysis include:

- trauma;
- increased muscle oxygen consumption;
- decreased muscle energy production;
- infection;
- **metabolic and genetic factors;**
- drugs and toxins;
- exposure.

To definitively diagnose rhabdomyolysis one needs to test for myoglobin in urine or to measure the Creatine Kinase (CPK) level in blood, which will give a clear indication if there is any significant rhabdomyolysis. However, these tests are only likely to reveal abnormal results during an acute episode of rhabdomyolysis.