IN THE CORONERS COURT
OF VICTORIA
AT MELBOURNE

Court Reference: 5545/09

FINDING INTO DEATH WITH INQUEST

Form 37 Rule 60(1)
Section 67 of the ***Coroners Act 2008***

Inquest into the Death of TYLER DI BLASI

Delivered On:	1 December 2011
Delivered At:	Coroner's Court of Victoria Level 11, 222 Exhibition Street Melbourne Victoria 3000
Hearing Dates:	23 November 2011
Findings of:	CORONER K. M. W. PARKINSON
Representation:	Mr Robert Harper of Counsel for The Royal Children's Hospital
Police Coronial Support Unit:	Leading Senior Constable G McFarlane

I, KIM PARKINSON, Coroner having investigated the death of TYLER DI BLASI

AND having held an inquest in relation to this death on 23rd November, 2011
at Melbourne

find that the identity of the deceased was TYLER DI BLASI

born on 18th September, 2008

and the death occurred on 13th November, 2009

at The Royal Children's Hospital, Flemington Road, Parkville, Victoria 3052

from:

1a. PARAINFLUENZA, PNEUMONITIS AND ASTHMA; HAEMORRHAGIC MUCOSITIS OF THE BOWEL AND GASTROINTESTINAL BLEEDING INDUCED BY CHEMOTHERAPY TREATMENT
1b. CHEMOTHERAPY INDUCED NEUTROPENIA AND THROMBOCYTOPENIA
2. ACUTE LYMPHOBLASTIC LEUKAEMIA

in the following circumstances:

1. An inquest was held into the death of Tyler Di Blasi on 23 November 2011. The following witnesses gave evidence in the proceeding: Professor Trevor Duke; Dr Adrian Mattke; Dr Nathan Smalley and RN DIV 1 Jillian Matchan. Statements were made by each of the witnesses and whilst I do not recite the contents of the statements in this finding, they have formed a part of my consideration in this case.

2. I have also had regard to the concerns raised by the family in their correspondence, which forms a part of the brief of evidence in this case. Their concerns included the level of medical monitoring of Tyler during the course of his admission, the lack of isolation from initial admission which family considered exposed him to the risk of infection, the medication regime administered, and the level of information about the clinical course and prognosis with which they were provided.

3. Tyler was born on 18 September 2008 to parents, Ms Suzanne Kostiuk and Mr Darren Di Blasi. He was 14 months old when he died on 13 November 2009.

4. On 14 October 2009, Tyler was diagnosed with acute lymphoblastic leukaemia for which he was being treated with a regime of chemotherapy. It was described by the clinicians as an aggressive form of the disease. On 23 October 2009, he had a double lumen Hickman inserted and he commenced on a daily initial regime of chemotherapy. He was discharged to the hospital in the home program.

5. On 29 October 2009, Tyler was re-admitted to the Royal Children's Hospital for management of febrile neutropaenia (low white cell count) which he developed as a consequence of the chemotherapy. He continued to be unwell on the ward and developed an acute respiratory tract infection and asthma, of which there was a family history. Parainfluenza type 3 was isolated from nasal secretions. Tyler was admitted to the Intensive Care Unit on 12 November 2009.

6. The source of his contracting parainfluenza is unable to be determined. The evidence is that parainfluenza virus is common in the community, but of particular concern if acquired by someone who is severely immuno compromised. Family were concerned that Tyler was in a shared ward and not a single room in view of his aggressive chemotherapy regime. The hospital stated in a reply made to concerns raised by the family that there were no single beds available at the time of admission and that his clinical presentation did not immediately warrant admission to Intensive Care Unit.

7. Hospital testing of three other patients who were located in the ward with Tyler did not reveal that any of those patients had parainfluenza virus. The virus is present in many people in the community and it is possible that it was acquired either in or out of hospital from contact with nursing or medical staff, family or visitors. The fact of the virus having been acquired was identified and responded to promptly.

8. Tyler was admitted to the Intensive Care Unit on 12 November 2009. He was examined at 1730 hours by Professor Trevor Duke. He was also under the care of Dr Smalley and Dr Mattke during the course of his admission.

9. Professor Duke stated:

> *"I examined Tyler at approximately 1730 hours. He was alert but irritable. He had moderate to severe respiratory distress, with chest hyperinflation and marked expiratory wheeze, which was audible with a stethoscope on the chest, and also from his bedside. His parents told me he had previously had an episode of asthma, related to a cold, and there were a strong family history of asthma and eczema. Tyler was receiving oxygen at a flow rate of 2L/minute by nasal prongs and had an oxygen saturation level measured by pulse oximetry above 93%. His chest x-ray showed lung hyperinflation and patchy infiltration consistent with viral pneumonitis. On a full blood examination carried out that morning there was severe thrombocytopenia (platelet count <10 x 109/L) and mild anaemia (Hb 99g/L). There had been some bleeding from his bowels throughout the day, which I understood had been going on for some time on the ward at a low level."*

10. Professor Duke concluded that Tyler had parainfluenza, pneumonitis and bronchospasm (asthma); bone marrow suppression following intensive chemotherapy; gastrointestinal bleeding secondary to thromcytopenia and possibly chemotherapy induced mucosal erosion or stress ulceration.

11. Professor Duke monitored and reviewed Tyler's medication and treatment regime. His evidence is that he maintained most of the existing treatments and instituted additional measures of intensive support including: high air oxygen mix to maintain oxygen saturation above 92%, full bronchodilator therapy to treat the asthma, including salbutamol, aminophylline, corticosteroids and magnesium sulphate. He was also treated with Osteltamavir (Tamiflu) for parainfluenza and Acyclovir for prevention of herpes viral infection. A platelet transfusion was ordered and administered, bringing his platelet count from 10 to 30.

12. Broad-spectrum antibiotics were continued even though his infection was viral to provide cover for bacterial infection, which was a risk as a result of the mucositis of the bowel. Morphine was maintained together with paracetamol as needed. Dexamethazone was continued as bone marrow testing had revealed that his leukaemia was not in remission and it was important that the chemotherapy be continued. This was also therapeutic for asthma and consultation occurred with the oncology consultant as to the appropriateness of this course.

13. A further platelet transfusion ordered later in the evening took longer than ideal to be arranged and administered, however this was ordered as a measure of maximising therapy, not in a context of critical response, and there is no evidence to suggest that this delay contributed to the deterioration of Tyler's condition or to his death.

14. Professor Duke also introduced pantoprazole for the treatment of Tyler's gastrointestinal mucosal ulceration. This was in substitution for Ranitidine with which he had earlier been treated. He commented that this was a more effective drug for treatment of stomach or duodenal ulceration, although neither drug would be effective for chemotherapy induced mucosal ulceration. His evidence was that there was no effective drug therapy available for that condition and this was the more likely cause of Tyler's gastrointestinal bleeding. Professor Duke also discussed regimes to address Tyler's neutropaenia with the oncologist, however this had the propensity to stimulate the cancer. It was also considered that surgery or endoscopy in an attempt to address his gastrointestinal bleed was dangerous in light of his condition and that pharmacological therapy was more appropriate.

15. At 1900 hours, a staff meeting was held to discuss a consistent plan for Tyler's treatment at which the treatment plan devised by Professor Duke was outlined. Discussion was also held as to possible future interventions, including nasopharyngeal CPAP and intubation. It was determined that intubation and mechanical ventilation should occur if required, however that in view of the risks involved non invasive respiratory support was desirable. It was explained that intubation markedly increased the risk of death in children with acute leukaemia, neutropaenia and multi organ failure and would have carried a significant risk in a child with severe obstructive airways disease and pneumonia.

16. Professor Duke's evidence was that during the evening of 12 November 2009, whilst Tyler remained irritable he had settled somewhat with an oxygen saturation of >95% on high flow air-oxygen mix of 8 L/min and ~ 60% oxygen. He noted that he still had chest wheezing and chest in drawing, but this was not worsening and he was able to independently ventilate. A blood gas test performed at 2100 hours demonstrated he had effective ventilation and normal oxygen levels. The plan was to disturb Tyler as little as possible during the course of the evening to permit him to rest provided his clinical course remained stable. Professor Duke left the intensive care unit shortly after midnight.

17. At 2200 hours on 12 November 2009, there was a medical round in which Professor Duke and Dr Smalley reviewed Tyler. No alteration was made to his management at that time. Dr Smalley stated that he saw Tyler on 6 occasions during the night. Dr Smalley stated that Tyler had some blood per rectum (which was a mixture of old and fresh blood) early in the evening and a platelet count, which was very low. Tyler's haemoglobin level was lower than it had been previously but his oxygen requirement was stable and Tyler remained settled in his parents arms. At 0100 hours on 13 November, further medication alteration was ordered by Dr Smalley, with the addition of magnesium sulphate another bronchodilator in a further attempt to assist respiration.

18. At 0340 hours on 13 November 2009, Tyler was lying on the bed having his nappy changed when he is reported to have had a period of apnoea lasting for not more than 10 seconds. This recovered spontaneously. He remained conscious and did not become cyanosed or bradycardic. Nurse Matchan notified Dr Smalley who attended Tyler and ordered the addition of Atrovent nebuliser. The medication was administered at 0420 hours. During the evening it was noted that Tyler's aminophylline levels were high and the dosage was reduced to address any possibility of true excessive levels, although the clinicians believe it more likely that the administration of the drug through the central line was responsible for the high reading.

19. Nurse Matchan reports that Tyler remained clinically stable until around 0650 hours on 13 November 2009. Dr Smalley and Dr Mattke's evidence was that during the course of the evening Tyler was a stable patient and that his rapid deterioration and death was unexpected.

20. The evidence is that the deterioration in Tyler's condition was rapid rather than a progressive deterioration over several hours. Professor Duke's evidence was that he formed this view based on the observations charted, and as a result of clinical indications, including that Tyler's mum fed him with a bottle of soup juice at 0600 hours on 13 November 2009, which he consumed willingly. Professor Duke's evidence was that in a case of severe respiratory distress babies are generally unable to feed and this suggested that Tyler's respiratory status at that time was not dire. These factors caused him to conclude that the deterioration was of sudden onset.

21. Whilst I have not set out in its entirety the treatment regime initiated, it can be seen that a complex and aggressive regime of intervention was developed and administered by the clinicians. It is apparent that there was careful attention paid to the course of Tyler's treatment by the medical clinicians.

22. The evidence is that a number of judgments were required of the treating doctors, rapidly, in balancing treatments with potential problematic side effects, against risks of not providing the treatment. Professor Duke described in his evidence how it was that he made the initial treatment decisions and Dr Smalley described the various responses to Tyler's clinical course over the evening.

23. Owing to the need by the doctors to respond quickly and flexibly to the various clinical developments it appears that information about the decisions being made was not readily available to Tyler's mother and father. As a result it appeared to them that there was some contradictory advice being provided as to not only Tyler's medical status, but also as to the treatment regime.

24. Nurse Matchan observed Tyler throughout the night of 12 and 13 November 2009, and the nursing notes provide a detailed record of Tyler's course, with careful and regular observations being made by her and appropriate notifications of urgent or critical events. There is no evidence of any deficiency in the nursing care provided to Tyler during the course of his admission to the Intensive Care Unit.

25. At 0630 hours on 13 November 2009, Tyler had another episode of malaena. At 0650 hours he went into respiratory arrest. Nurse Matchan commenced immediate respiratory support by bag and mask ventilation and asked Ms Kostiuk to press the emergency bell. When there was no immediate response she asked Ms Kostiuk to leave the room and call for assistance. It was distressing for Ms Kostiuk to leave her child in these circumstances and it is understandable in light of the delayed response, that she felt uncertain as to the adequacy of the care and response that her child was receiving.

26. Dr Mattke describes that he received the notification and proceeded immediately to respond. Unfortunately, he was notified of the wrong room and attended the room located a short distance away from Tyler's. His evidence was that the error was immediately known and that within 1 to 2 minutes he was at Tyler's bedside. During his oral evidence he stated that the time frame was probably more likely less.

27. His evidence and that of Dr Smalley is that the delay had no impact upon the emergency response care provided to Tyler. The evidence is that Nurse Matchan was providing respiratory support by bag and mask ventilation when they entered the room and Dr Mattke took over that procedure. Within a short time of ventilation, the transcutaneous oxygen saturation reached 90%. Tyler's chest was described as having poor air entry on both sides and there was audible bilateral wheezing.

28. Dr Mattke and Dr Smalley also gave evidence about the reasoning behind their initial decision not to intubate Tyler. This was largely for the reasons already discussed by Professor Duke. When blood gas results indicated severe hypoventilation with a respiratory acidosis, the doctors determined that it was necessary to intubate to provide mechanical ventilation. The evidence is that the intubation was not difficult and that there was clear sight of Tyler's airway, however the first intubation did not result in there being any observable chest movement. Dr Mattke states that in such a case it is appropriate to extubate and reintubate to ensure that the tube is appropriately placed. This occurred within a period of 20 to 30 seconds and did not compromise Tyler. After intubation they were able to ventilate Tyler, but with very high airways pressure and there remained little chest movement. The tube position was checked by laryngoscopy and by end tidal CO2 measurement . Shortly afterwards Tyler's heart rate fell to 70 -100 beats per minute. Despite administration of Adrenalin and Atropine there was no increase in his heart rate and Tyler had an asystolic arrest. Cardio Pulmonary Resuscitation (CPR) was administered and the endotracheal tube was again replaced as it had been dislodged during CPR. CPR continued and pneumothorax was excluded.

29. Whilst there is no evidence to suggest that the first intubation was unsuccessful, the clinical indicators meant that the decision to re-intubate was an appropriate clinical decision and consistent with resuscitation practice guidelines.

30. At 0800 hours on 13 November 2009, after discussion with mother and father, a decision was made to cease resuscitation efforts. Tyler was a very ill little boy, however, the evidence is that none of the clinicians had expected the decline which occurred at 0650 hours. The rapid deterioration was unexpected. Despite every reasonable effort, Tyler was unable to be rescued and he died in his father's arms at 0805 hours on 13 November 2009.

31. The cause of death was initially stated on a death certificate issued on 13 November 2009 to be due to 1(a) pneumonitis; 1(b) acute lymphoblastic leukaemia. A notification to the coroner did not occur until 25 November 2009, after an internal review process undertaken by the hospital. That notification stated that the cause of death was: 1(a) respiratory and cardiac arrest; 1(b) pneumonitis due to parainfluenza virus; 2 Contributing factors Acute lymphoid leukaemia and neutropenia. No express mention was made of the gastrointestinal bleed.

32. In his evidence Professor Duke stated that it was his view there were a number of contributing factors and that the cause of death was most accurately stated as: Acute Lymphoblastic leukaemia; Chemotherapy induced neutropenia and thrombocytopenia; parainfluenza, pneumonitis and asthma; and haemorrhagic mucositis of the bowel/gastrointestinal bleeding induced by chemotherapy treatment. His evidence was:

> *"On the limited evidence available one likely explanation was that the haemorrhage mucositis of the bowel suddenly became severe around 6.30am on the morning of 13 November 2009 and in the setting of severe bronchospasm this resulted in further deterioration in his breathing function and subsequent arrest. The mechanisms by which this could occur would be severe abdominal distension from severe bleeding and sloughing of mucosal lining of the bowel, putting pressure on his breathing muscles, particularly the diaphragm."*

33. There was no post mortem undertaken and as the death was not initially reported there was no examination by a forensic pathologist, however notwithstanding this, I am satisfied the cause of Tyler's death is most accurately stated by Professor Duke.

34. The evidence was that Tyler's death was 'not expected' in a clinical sense having regard to the course of his diagnosis, admission and treatment. The hospital report to the coroner stated that the reason for reporting was 'concerns about clinical management and death not fully expected'. In these circumstances, as I noted during the proceedings, Tyler's death ought to have been the subject of a report to the coroner at the time it occurred.

35. Whilst the hospital reported the death when it was realised that there were issues associated with the circumstances, as a matter of public interest, it is important that clinicians responsible for making the decision as to reporting deaths to the coroner carefully consider the issue of reporting at the earliest stage.

36. The death of Tyler was a very sad and distressing event. Mother and father are profoundly affected by the loss of their little boy. The loss of this little boy has clearly also touched the medical and nursing staff of the Royal Children's Hospital.

37. Mother and father raised a number of concerns in relation to the treatment and care provided to Tyler during his admission to the Intensive Care Unit. Whilst it appears that the information provided to the family during Tyler's admission may not have been sufficient to enable them to understand the course of the treatment plan or the reasons for changes in approach to the plan, the evidence does not support a conclusion that the care or treatment was inadequate.

38. I am satisfied that the medical management and nursing care was reasonable and appropriate in the circumstances and that there were no issues of care or treatment, which caused or contributed to Tyler's death.

39. It is appropriate to note that some of the issues raised by the family have been significant in the hospital's subsequent consideration of layout, location and design of children's intensive care facilities. The hospital acknowledged that the communication was not satisfactory. Professor Duke stated in his evidence that the hospital had learned much from this tragic death about how to manage children in the new hospital, in particular by ensuring easy visibility from all parts of the ward and the availability of single rooms in the Intensive Care Unit and Oncology.

40. Earlier correspondence from the hospital revealed that they had also responded at the former hospital site to concerns about the size and location of the room in which Tyler was located, by alterations to the room, visibility from the nurses station and the emergency notification arrangements. They acknowledged the distress caused to the family arising from these matters.

41. Having considered all of the available evidence I find that Tyler Di Blasi died on 13 November 2009 and that the cause of his death was:

1(a) Parainfluenza, pneumonitis and asthma; haemorrhagic mucositis of the bowel and gastrointestinal bleeding induced by chemotherapy treatment.
1(b) Chemotherapy induced neutropenia and thrombocytopenia
2. Acute Lymphoblastic leukaemia;

42. I direct that a copy of these findings be provided to the family; the interested parties and to Professor Trevor Duke; Dr Adrian Mattke; Dr Nathan Smalley and RN DIV 1 Jillian Matchan.

Signature:

KIM M W PARKINSON
CORONER
1st December, 2011

Coroners Court of Victoria