Coroners Act 1996
[Section 26(1)]

Western *Australia*

RECORD OF INVESTIGATION INTO DEATH

Ref: 32/14

I, Sarah Helen Linton, Coroner, having investigated the death of ***TS (name suppressed)*** *with an inquest held at the* ***Perth Coroner's Court, Court 51, CLC Building, 501 Hay Street, Perth****, on* ***16 - 18 September 2014****, find that the identity of the deceased person was* ***TS (name suppressed)*** *and that death occurred on or about* ***15 October 2010*** *at* ***274 Wright Street, Cloverdale*** *in circumstances in which the cause of death is unascertained but consistent with epileptic seizure:*

Counsel Appearing:

Ms C Fitzgerald assisting the Coroner.
Ms C Thatcher (State Solicitors Office) appearing with Mr N Van Hattem (State Solicitors Office) on behalf of the Department for Child Protection and Family Support.

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SUPPRESSION ORDER

The names of the deceased and the deceased's sister, and any identifying information, are suppressed. The deceased is to be referred to as TS and the deceased's sister is to be referred to as TAS.

INTRODUCTION

1. On the evening of 14 October 2010 the deceased, TS, went about his usual evening activities before going to bed at about 9.00 pm. He did not complain of any illness and his grandparents did not observe anything out of the ordinary about him that night.

2. The following morning, when his grandfather went to wake the deceased just after 7.10 am, he was found unresponsive in his bed. Efforts by his grandparents to resuscitate him were unsuccessful and an ambulance officer certified his death at 7.29 am.[1] He was only ten years old.

3. The forensic pathologist who conducted a post mortem examination was unable to ascertain a definite cause of death, but concluded it was consistent with an epileptic seizure.[2]

4. At the time of his death, the deceased was subject to a protection order made in the Children's Court of Western Australia, which included him in the definition of a 'person held in care' under the *Coroner's Act 1996* (WA). Pursuant to s 22(1)(a) of the Act an inquest hearing was, therefore, mandatory.[3]

5. I held an inquest at the Perth Coroner's Court on 16 - 18 September 2014.

6. As the forensic pathologist who conducted the post mortem examination was unable to ascertain a definite cause of death,[4] the inquest hearing focused primarily on what medical information was available

[1] Exhibit 1, Tab 3 – Life Extinct Form.
[2] Exhibit 1, Tab 32.
[3] The deceased was subject to a protection order (time-limited) made in the Children's Court of Western Australia on 23 September 2010 pursuant to sections 28 and 54 of the *Children and Community Services Act 2004* (WA). As such, he was a child in the Chief Executive Officer's care, which is included in the definition of a 'person held in care' in s 3 of the *Coroner's Act 1996* (WA). Pursuant to 22(1) (a) of the *Coroners Act*, it is mandatory that an inquest be held into the death of a 'person held in care'.
[4] Exhibit 1, Tab 32.

that might assist in establishing a cause of death. The care of the deceased in the period leading up to his death, as well as the circumstances of his death, were also the subject of evidence.

MEDICAL HISTORY OF THE DECEASED

7. The deceased was born on 20 August 2000.[5]

8. The deceased was the first child of his parents, although his father had an older child from a previous relationship who did not live with them. The deceased was later joined by a younger sister in 2003.[6]

9. The antenatal history records the deceased's mother was a smoker and an intravenous drug user who reported her last drug use was two days prior to delivery, although this does not accord with his mother's recollection of events.[7]

10. The deceased was born at 39 weeks gestation and was noted to be growth retarded at birth. He was observed to be intermittently jittery, but no congenital anomalies were noted and his jitteriness subsided after two days.[8] Other than a need to gain weight, there were no major concerns regarding his wellbeing when he was discharged into his parents' care three days after birth.[9]

11. Shortly after his birth the deceased's father went to prison and remained there for much of the deceased's life. The deceased lived predominantly with his mother and sister, but kept in regular contact with his father. [10]

[5] Exhibit 1, Tab 1.
[6] Exhibit 1, Tab 38, 1.
[7] Exhibit 1, Tab 24; in her statement the deceased's mother maintains she did not use drugs during her pregnancy although she did smoke cigarettes – Exhibit 1, Tab 5 [15] – [16].
[8] Exhibit 1, Tab 24.
[9] Exhibit 1, Tab 24.
[10] Exhibit 1, Tab 4, 2 & Tab 5 [21].

12. Apart from some minor childhood illnesses in 2002,[11] a brief hospital admission in 2004 for pneumonia and iron deficiency anaemia[12] and a fractured bone from a fall,[13] the deceased was reportedly a generally well child until 2007.

13. According to his mother, in about 2007 the deceased started to have seizures.[14] The seizures would only occur between 3.00 am and 4.00 am and would cause the deceased to lose vision, twitch and hallucinate.[15]

14. On 5 July 2008, the deceased presented to the Princess Margaret Hospital (PMH) Emergency Department due to a reported seizure. At that time the hospital records noted that the deceased was reported by his grandfather not to have fitted previously, which is inconsistent with the deceased's mother's account.[16] The principal diagnosis was of a generalised tonic-clonic seizure. He was discharged later that day with a recommendation to take oral iron and follow up with his general practitioner.[17]

15. It seems that sometime in 2008 the deceased began taking the drug clonidine. The deceased's mother advised police that she took the deceased to an unknown doctor, possibly in Belmont, who diagnosed him with ADHD and prescribed him with a medication containing clonidine for the seizures.[18] Police investigations were unable to locate any record of a doctor having prescribed this medication to the deceased.[19]

[11] Exhibit 1, Tab 4, 2 & Tab 28.
[12] Exhibit 1, Tab 21; Exhibit 2, Tab 3, 1st Admission.
[13] Exhibit 1, Tab 21.
[14] Exhibit 1, Tab 5 [31].
[15] Exhibit 1, Tab 4, 2.
[16] Exhibit 2, Tab 1, PMH Emergency Medical Summary to Dr Kerr, faxed 5.7.2008; Tab 3, Emergency Department record.
[17] Exhibit 1, Tab 21.
[18] Exhibit 1, Tab 4, 3; Tab 5 [46] – [47].
[19] Transcript 7; Exhibit 1, Tab 4, 3.

16. The deceased's mother also has a history of seizures[20] and she was therefore able to obtain prescriptions for clonidine on her own behalf.[21] Rather than take the deceased to the doctor to get his own prescriptions of clonidine, she maintained his daily dose by giving the deceased medication obtained from her own prescription.[22]

17. The deceased's mother also began to give the same medication to her daughter, TAS, on the basis that she believed her daughter also suffered from ADHD and the medication helped her to sleep. She later suggested that a diagnosis had been made for TAS of ADHD, but again was unable to identify a doctor who could be contacted to confirm the diagnosis.[23]

18. The deceased's mother reported that after the deceased commenced taking the clonidine his seizures stopped.[24] However, he did still have seizures on occasion as on 24 April 2009 the deceased again presented to the PMH Emergency Department with a complaint of seizures. He was reported to have experienced two seizures that day. The first, at 4.00 am, lasted a few minutes and the second, at 9.35 am, lasted ten minutes and caused him to be incontinent of urine and to bite his tongue.[25] A history of other seizures over the previous year was also given at that time.

19. The medical notes record that the hospital staff were aware the deceased was taking clonidine at that time. The notes record that he was taking clonidine for sleep difficulties associated with a diagnosis of ADHD.[26] The deceased was admitted to a ward for observation. No seizure activity was observed overnight.

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[20] Exhibit 2, Tab 1, Integrated Progress Notes.
[21] Exhibit 3, Tab 20.
[22] Exhibit 1, Tab 4, 3.
[23] Transcript 7; Exhibit 1, Tab 4, 3; Exhibit 6, 128.
[24] Exhibit 1, Tab 5 [49].
[25] Exhibit 2, Tab 1, PMH Emergency Medical Summary to Dr Kerr, faxed 24.4.2009; Tab 3, SJA Patient Care Record, 24.04.2009.
[26] Exhibit 2, Tab 3, Inpatient Progress Notes 24.4.2009 - 25.4.2009.

20. The deceased was discharged the following day with a plan to have an outpatient EEG, seizure safety training and see Dr Holmes in six weeks.[27] It was also suggested that his clonidine use be phased out as this might be causing nightmares or pseudo-seizures and a referral was made to the State Child Development Centre for clarification of the diagnosis of ADHD.[28]

21. In relation to his reported seizures, the deceased had an EEG performed on 11 May 2009. In the opinion of the paediatric neurologist who conducted it, the EEG was abnormal and the findings were consistent with a susceptibility to epilepsy. A repeat EEG including a sleep state was recommended and an outpatient appointment was scheduled for this to occur.[29] The deceased did not attend his outpatient appointment at PMH scheduled for 21 July 2009. A second appointment was then offered and scheduled for 25 November 2009.[30]

22. The deceased did not attend the second appointment scheduled for 25 November 2009. An attempt was made to locate the deceased's family and a PMH staff member telephoned the deceased's grandfather. They were told that the deceased's family had moved on and left no forwarding contact details.[31] As a result, a repeat EEG was not performed.[32] Further, no formal diagnosis of epilepsy was made and no medication to control the seizures was prescribed.

23. As a result of the referral to the State Child Development Centre the deceased was assessed in September 2009 and not found to meet the criteria for ADHD.[33] In February 2010, further contact was

[27] Exhibit 1, Tab 21.
[28] Exhibit 2, Tab 3, Inpatient Progress Notes 24.4.2009 - 25.4.2009 and Consultation Request 25.4.2009, Dr Paula Holmes.
[29] Exhibit 2, Tab 2, Outside Reports, EEG Report, Dr Simon Williams, 11.05.2009.
[30] Transcript 27; Exhibit 1, Tab 21.
[31] Transcript 27.
[32] Exhibit 1, Tab 21.
[33] Exhibit 2, Tab 2.

made with the deceased's mother to explore this issue further (as well as TAS' possible ADHD diagnosis).

FIRST CONTACT WITH THE DEPARTMENT

24. The Department for Child Protection and Family Support (the Department) had limited contact with the deceased and his family until March 2010.[34] Until that time, no child protection concerns were expressed about either the deceased or his sister.[35]

25. This was despite the fact that concerns had been raised by PMH staff in 2008 in relation to the deceased's sister's clonidine use.[36]

26. On 19 March 2010, a PMH social worker raised concerns with the Department in relation to the deceased's sister, TAS.[37] She had been admitted to PMH three times in six months due to health complications arising from the inappropriate administering of clonidine to her by her parents.[38] There were also concerns about whether the parents' substance misuse would impact on their ability to care for her.[39]

27. Following the report a Safety and Wellbeing Assessment was then undertaken by Department staff to consider the safety of both the deceased and his sister.[40]

28. As part of the assessment a meeting was conducted by Department staff with the deceased's parents and PMH staff on 23 March 2010.[41] The doctors tried to impress upon both parents the need to ensure that

[34] Exhibit 1, Tab 38, 1.
[35] Exhibit 1, Tab 38, 1.
[36] Exhibit 6, Tab 128.
[37] Exhibit 1, Tab 38, 1.
[38] Exhibit 1, Tab 27 & Tab 38, 1.
[39] Exhibit 1, Tab 38, 1.
[40] Exhibit 1, Tab 38, 2 and Attachment 2.
[41] Exhibit 6, Tab 128.

TAS ceased taking the drug clonidine and TS be weaned off the medication under supervision by a medical team.[42]

29. On 29 March 2010, a further meeting was conducted with PMH medical staff, the deceased's parents and the deceased's maternal grandmother. At the conclusion of the meeting, the deceased's parents agreed to attend arranged appointments at PMH with both children and to cooperate with a medical plan to safely wean TS off clonidine under supervision. Appointments were made for 1 April 2010 and 5 April 2010.

30. On 6 April 2010, Department staff were informed that TAS and TS had not attended the PMH appointments as arranged. They had also not attended their Child Development Centre appointments.[43]

31. The deceased's parents did attend PMH with TAS on 7 April 2010, but did not bring TS with them. The PMH doctor who saw TAS that day reiterated to her parents that TAS should definitely not be put back on clonidine.

THE DECEASED AND TAS ARE TAKEN INTO CARE

32. On 9 April 2010, the deceased's sister presented to PMH in a heavily sedated and unresponsive state. The sequence of events leading to her re-admittance was unclear. Doctors were of the opinion that her symptoms were consistent with a clonidine overdose.[44] TAS was admitted to the Intensive Care Unit and Department staff was notified. A decision was made that day for TAS to be brought into the provisional care of the Department due to continuing

[42] Exhibit 6, Tab 128.
[43] Exhibit 4, Tab 52, Attachment 1.
[44] Exhibit 1, Tab 38, Attachment 2, 3; Exhibit 3, Tab 1; Exhibit 4, Tab 75.

concerns about her safety and health should she be discharged into the care of her parents.

33. The deceased's parents were informed of this decision and told that the deceased could remain in their care provided he attended all scheduled appointments at PMH.

34. TAS remained in hospital until 12 April 2010, when she was discharged into the care of the CEO of the Department and taken to the home of a foster carer.[45]

35. On 13 April 2010, PMH staff contacted Department staff and informed them that the deceased had presented for a PMH appointment and had been found to have high blood pressure.[46] His mother could not provide clear information about when he had last taken clonidine.[47] The deceased's mother was told by a PMH doctor that, given the deceased's high blood pressure, he needed to be admitted to hospital in order to be medically supervised whilst being weaned off clonidine. The deceased's mother refused to allow him to be admitted as she was concerned that he would be taken into care.[48]

36. Department staff met with the deceased's mother at the Department's Cannington Office that day to discuss the medical advice and her expressed concerns. The deceased's mother stated that she would not allow him to be admitted at PMH as she did not trust the hospital or the Department. She was told that if she did not allow the deceased to be admitted he would be brought into the care of the CEO of the Department and then be admitted to hospital. The deceased's mother's response was to panic and flee the building with the deceased. They

[45] Exhibit 1, Tab 38, Attachment 2, 3.
[46] Exhibit 1, Tab 38.
[47] Exhibit 3, Tab 47; Exhibit 6, Tab 130 [28].
[48] Exhibit 1, Tab 38.

got into a car and drove away, despite the efforts of Department staff to stop them.[49]

37. Efforts to locate the deceased that night and the following day were unsuccessful. On 15 April 2010, the deceased's mother took the deceased to Cannington Police Station. The Department was notified and Department staff attended the police station and took the deceased into the care of the CEO. He was then transported to PMH and admitted for medical treatment.

38. The doctor managing the deceased's medical care from April 2010 onwards was Associate Professor Donald Payne, a general paediatrician at PMH. Associate Professor Payne had already been involved in the ongoing treatment of TAS in relation to her clonidine usage and he took over the care of the deceased upon his admission to hospital on 15 April 2010.[50] Associate Professor Payne's primary concern at that time was to ensure that the deceased was kept in a safe environment and weaned off clonidine in a controlled fashion.[51]

39. The deceased remained in hospital for five days while his clonidine dose was gradually reduced. He was then discharged on 20 April 2010 and taken to foster care.[52]

40. He was managed by way of home visits by nursing staff and reviewed as an outpatient at PMH from that time. His dosage of clonidine was progressively further reduced, and was discontinued completely on 3 May 2010.[53]

[49] Exhibit 1, Tab 38.
[50] Transcript 23 – 24; Exhibit 1, Tab 27.
[51] Transcript 25 – 26, 28.
[52] Transcript 26 - 27; Exhibit 1, Tab 27 & Tab 38.
[53] Exhibit 1, Tab 27.

MOVE TO LIVE WITH GRANDPARENTS

41. A Provisional Protection Care Plan meeting was held on 29 April 2010. The meeting was attended by Department staff, the deceased's parents and his maternal grandparents. At the meeting it was agreed that the deceased and his sister would reside with their maternal grandparents. Any contact with their parents was to be supervised.[54]

42. This is not to suggest that the deceased's relationship with his parents (and indeed his sister's relationship with them) was anything other than loving. The deceased's relationship with his mother, in particular, was very close and there is no doubt they loved each other very much and wanted to live together.[55] Unfortunately, despite her love for the deceased and his sister, the deceased's mother had demonstrated she was not able to care for their needs adequately at that time. It was hoped that, with time, that would change and the family could be reunited.[56] A plan was put in place to progress towards that goal.

43. In the meantime, the evidence indicates that the deceased and his sister thrived in the care of their grandparents, although the deceased continued to miss his mother greatly.[57]

44. On 14 May 2010, the deceased was reviewed at PMH by a doctor and there were no concerns about his health at that time.[58]

45. On 14 June 2010, the deceased underwent a formal health review conducted by Associate Professor Payne. By then he had ceased clonidine use for more than one month. The review indicated that he was not on any medication, his blood pressure was

[54] Exhibit 1, Tab 38, Attachment 3.
[55] Exhibit 1, Tab 4, 2 & Tab 5 [41] & Tab 8 [47].
[56] Exhibit 1, Tab 8 [68] – [70].
[57] Transcript 27, 67; Exhibit 1, Tab 8 [65] & Tab 38, 4.
[58] Transcript 27; Exhibit 27.

within normal range, he had no problems getting to sleep and was sleeping well. Associate Professor Payne was pleased with his progress and the deceased's grandparents expressed no concerns about his health on this occasion.[59]

46. Associate Professor Payne was aware at this time of the deceased's previous reported history of seizures and that an 'abnormal' EEG had been performed in 2009 and a repeat EEG had not been performed.[60] He also understood that there had been no reports of the deceased experiencing any further seizure since that time.[61] It was thought that it was possible the seizures had been secondary to clonidine use, although that was known to be a rare occurrence.[62]

47. Epilepsy is a clinical diagnosis, generally based on the history that is given of seizures.[63] The use of EEG's and MRI scans and other investigations is usually employed to try to identify the particular variety of epilepsy.[64]

48. Given there were no further reports of ongoing seizures at the time he was treating the deceased, Associate Professor Payne concluded that there was no indication to perform any further investigations around the earlier reported seizures.[65]

49. Department staff undertook home visits on 5 May 2010, 1 June 2010, 15 June 2010 and 26 August 2010 and no concerns were raised about the deceased's health or care. The quarterly report in September 2010 did, however, note that although the deceased loved his grandparents, he continued to

[59] Transcript 27; Exhibit 1, Tab 27 & Tab 38, Attachment 4.
[60] Transcript 27.
[61] Transcript 28 – 29; The deceased's mother had informed the Department that the deceased had not had any seizures since October 2009 – see Transcript 62 and Exhibit 4, Tab 54.
[62] Transcript 28 – 30; Transcript 27.
[63] Transcript 30, 46 - 47.
[64] Transcript 46.
[65] Transcript 29.

express his wish to live with his mother again as soon as possible.[66]

50. On 23 September 2010, a Protection Order (time limited) was granted for the deceased for a period of two years.[67]

51. On 11 October 2010, the deceased was seen again at PMH by Associate Professor Payne. His blood pressure was normal and his weight had increased by over a kilogram since June. In the approximately six months since the deceased had been taken into care and ceased clonidine use, there had been no reports of him experiencing any seizures.[68]

52. At the conclusion of the appointment on 11 October 2010, as there were no longer any concerns about the deceased's health and Associate Professor Payne was "really satisfied that things were going well",[69] the deceased was discharged from further regular follow-up at PMH.[70]

53. On 13 October 2010, Department staff conducted another home visit. The deceased and his sister were seen with their grandparents, mother and great grandmother. The children both looked in good health and were reported to be sleeping well and not exhibiting any concerning behaviour. The deceased told the Department staff that things were going "really well" at home and at school.[71]

54. On the basis of the above, the Department held no concerns in relation to the standard of care being provided to the deceased and his sister by their grandparents. By all accounts they were happy and well. They were also seeing their mother regularly with supervision and it was hoped that overnight

[66] Exhibit 1, Tab 38, 3; Exhibit 3, Tab 39.
[67] Exhibit 1, Tab 38, Attachment 5.
[68] Exhibit 1, Tab 27, 4.
[69] Transcript 27.
[70] Exhibit 1, Tab 27, 2.
[71] Exhibit 1, Tab 38, 4.

visits with their mother might soon be permitted.[72] All of this reinforces the fact that the deceased's sudden death two days later was entirely unexpected and tragic.

EVENTS SURROUNDING THE DEATH

55. During the afternoon of 14 October 2010, the deceased watched some television and visited a friend who lived nearby.[73] Later that evening he played outside with his grandparents and sister and then ate dinner. After the deceased's sister went to bed the deceased spent some time alone with his grandparents. He was chatty and cracking jokes, enjoying his time alone with them.[74] He did not complain at any time of feeling ill.[75]

56. At about 9.00 pm, the deceased's grandfather put him into bed and left him reading a book.[76] The deceased's grandparents went to bed about an hour later.[77]

57. The deceased's grandparents woke up about 6.00 am the following morning and started their usual morning routine. Just after 7.10 am the deceased's grandfather went to wake the deceased. He went into the deceased's bedroom and saw the deceased lying face down, with his face turned towards the wall.[78]

58. The deceased's grandfather called out the deceased's name.[79] When the deceased did not respond, he put his hand on the deceased's back to shake him.[80] He felt the deceased was stiff and thought the deceased

[72] Exhibit 1, Tab 8 [68] – [70]; Tab 38, 4.
[73] Exhibit 1, Tab 7, [6] – [27].
[74] Exhibit 1, Tab 7 [48].
[75] Exhibit 1, Tab 7 [49].
[76] Exhibit 1, Tab 7 [50] – [52].
[77] Exhibit 1, Tab 7 [54]
[78] Exhibit 1, Tab 7 [56] – [62].
[79] Exhibit 1, Tab 7 [63].
[80] Exhibit 1, Tab 7 [64].

was having a seizure, so he rolled him over.[81] He then saw the deceased was blue. He called out to his wife to call an ambulance and then he and his wife followed the ambulance staff's instructions to attempt to perform cardiopulmonary resuscitation although it became obvious that he had died.[82]

59. Ambulance officers arrived at the house at 7.27 am. On arrival a paramedic conducted observations of the deceased. He noted he was stiff and cold and concluded resuscitation was contraindicated. He then tested for any electrical activity of the deceased's heart and found none.[83] One of the paramedics then certified his death.[84]

60. Police officers attended the house, inspected the deceased's bedroom and spoke with his family. Everything they observed suggested the deceased had been well looked after by his grandparents in a caring, loving and safe environment.[85]

CAUSE OF DEATH

61. On 19 October 2010, the Chief Forensic Pathologist, Dr C T Cooke, conducted a post mortem examination of the deceased. Given the sudden and unexplained nature of the death, the autopsy incorporated all of the observations recommended in the Australasian SIDS Autopsy Protocol.

Examinations and Investigations

62. The examination showed the body organs to be normally developed and there were no evident injuries.[86] Further investigations were then

[81] Exhibit 1, Tab 7 [65] – [67].
[82] Exhibit 1, Tab 7 [69] – [74].
[83] Exhibit 1, Tab 10 & Tab 35.
[84] Exhibit 1, Tab 3.
[85] Exhibit 1, Tab 16.
[86] Exhibit 1, Tab 32, Confidential Report to the Coroner.

undertaken as part of the post mortem examination to assist in identifying a cause of death.

63. Histopathology was performed, which involved the examination of samples of tissue down the microscope. The histopathology examination of all the major body tissues was essentially normal.[87]

64. Samples were also taken for virology and bacterial testing. No bacterial infection was found and the only virus that was detected was metapneumovirus, which is an insignificant virus that can cause 'flu-like' illness[88] but was unlikely to have contributed to the death.

65. A Mast Cell Tryptase level was also done to test for any allergic reaction that might indicate anaphylaxis. The result was essentially in the normal post mortem level.[89]

66. Toxicology analysis showed no alcohol or common drugs.[90]

67. Finally, an examination of the deceased's brain was performed by a specialist Neuropathologist, Dr V Fabian. Dr Fabian performed both a macroscopic (naked eye) and microscopic examination of the brain. The results of the macroscopic examination showed an abnormal shape and some gyral abnormalities in the brain.[91] However, the subsequent microscopic examination did not confirm the abnormalities in the fine structure of the brain.[92] Following these examinations, Dr Fabian concluded that there was no confirmed structural developmental abnormality to the brain.[93] She was also able to exclude trauma

[87] Transcript 72; Exhibit 1, Tab 32.1, Histopathology Report.
[88] Transcript 72; Exhibit 1, Tabs 32.2 & 32.3.
[89] Transcript 72 - 73; Exhibit 1, Tabs 32.2.
[90] Transcript 72 – 73; Exhibit 1, Tab 34.
[91] Transcript 16 – 17; Exhibit 1, Tab 33, Macroscopic Examination Report.
[92] Transcript 17 - 19; Exhibit 1, Tab 33, Microscopic Examination Report.
[93] Transcript 19.

to the brain, haemorrhage, infection and a brain tumour.[94]

68. Although no structural abnormality was identified, Dr Fabian clarified that this did not mean that epilepsy, and the possibility that the deceased had a seizure, could be excluded. In Dr Fabian's experience thirty percent of patients who die in circumstances known as sudden unexpected death in epilepsy are found to have an entirely normal brain. Accordingly, the neuropathology results do not exclude the possibility that the deceased had an epileptic seizure.[95]

Opinion of Dr Cooke

69. At the conclusion of all of the investigations Dr Cooke was unable to identify a structural cause for the death. As such, the cause of death was 'unascertained'.[96]

70. However, having excluded any structural cause and taking into account the deceased's known history of seizures[97] and the known circumstances surrounding his death, Dr Cooke formed the opinion the appropriate cause of death was 'unascertained (consistent with epileptic seizure)'.[98] Alternatively, some pathologists use the term "SUDEP" or "sudden unexpected death in epilepsy" in the same circumstances.[99]

71. Dr Cooke explained there is more than one postulated mechanism for death in a SUDEP case. Some are related to respiratory interference arising from the seizure, either through something happening in the body to interfere with the person's ability to breathe or asphyxia due to an obstruction.

[94] Transcript 18 – 22.
[95] Transcript 19 - 22.
[96] Transcript 73.
[97] Transcript 75, 78.
[98] Transcript 73; Exhibit 1, Tab 32, Confidential Report to the Coroner – Supplementary Report.
[99] Transcript 73 - 74.

Alternatively, there can be a cardiac mechanism where, for some reason, the electrical discharge of the seizure in the brain can cause electrical abnormality in the heart and cause an arrhythmia (electrical disturbance) of the heart.[100]

72. Dr Cooke also noted the possibility that the cause of death in this instance could be related to a possible cardiac genetic abnormality that led to a fatal cardiac arrhythmia. There have been developments in testing for these syndromes in the years since the deceased's death and experts now believe about 10 percent of SUDEP cases may also have one of these genetic abnormalities.[101]

73. There is additional evidence available since the post mortem that supports Dr Cooke's original opinion that this was a SUDEP death. This evidence was given by a paediatric neurologist, Dr P Walsh, at the inquest.

Opinion of Dr Walsh

74. Dr Walsh works as a neurologist at both PMH and King Edward Memorial Hospital, as well as in private practice. Dr Walsh specialises in treating children. He stated that he has probably seen more children with epilepsy than any other practitioner in Australia, given the length of time he has been practising (about 30 years) and the fact that his predecessors have now retired.[102] I accept that Dr Walsh is an expert in the field of treating epilepsy in children.

75. Dr Walsh was provided with various relevant reports relating to the deceased's medical history and the investigations undertaken after his death.[103] Based upon his examination of those documents, Dr Walsh

[100] Transcript 74 – 75.
[101] Transcript 76 - 77.
[102] Transcript 42.
[103] Transcript 43.

believes there is "very good evidence that the deceased had epilepsy".[104] As noted above, that diagnosis is a clinical diagnosis.[105]

76. Dr Walsh advised that there are over 30 different types of epilepsy that children can have.[106] Of these, Dr Walsh is of the opinion that the deceased had a type of epilepsy known as occipital epilepsy, and his symptoms were consistent with a form of occipital epilepsy known as the Gastaut form.[107] The deceased was in the right age category for this type of epilepsy and his visual symptoms and ability to talk and communicate during a seizure are characteristic of this form of epilepsy.[108] It is often genetic-inherited, so his mother's history of epilepsy is also supportive of the diagnosis.[109]

77. Dr Walsh did not place any reliance on the reported 'abnormal' EEG in forming his opinion. In his view, the description of the EEG as abnormal was a "soft finding" and many other EEG readers might well have called it normal.[110] Dr Walsh advised that for occipital epilepsy, the main findings would come from an EEG done during sleep recording (as was intended for the scheduled second EEG), so it was not surprising that the results of the first EEG, done during wakefulness, were pretty normal.[111]

78. Consistent with the post mortem neuropathology findings, Dr Walsh indicated that where occipital epilepsy is considered to be a primary condition (with a genetic basis) you would also expect the neuropathology to be completely normal.[112] Therefore, the absence of any structural abnormality

[104] Transcript 43.
[105] Transcript 46.
[106] Transcript 43.
[107] Transcript 43;
[108] Transcript 44.
[109] Transcript 44.
[110] Transcript 46.
[111] Transcript 46.
[112] Transcript 45.

in the deceased's brain does not contradict the diagnosis.[113]

79. In the opinion of Dr Walsh (which Associate Professor Payne indicated he would defer to in this regard),[114] the deceased's seizures were not likely to have been caused by his clonidine use. His opinion was based upon the fact that while a high dose of clonidine makes seizures more likely, it is only at the time the high dose is taken. On the other hand, low doses of clonidine can actually have an anticonvulsant effect.[115] Therefore, in this case the clonidine *may* have helped to regulate the deceased's seizures, although this cannot be said with any certainty.[116]

80. In Dr Walsh's opinion it is highly unlikely that the deceased's prior use of clonidine could have caused him to have a seizure on the night of his death.[117]

81. Dr Walsh agreed with Dr Cooke that there remains the possibility that the deceased had a congenital heart problem, which led to a cardiac arrhythmia.[118] However, given his conclusion that the deceased had epilepsy, he thinks it is reasonable to speculate that the deceased had an epileptic seizure during the night that resulted in his death.[119] On that basis, Dr Walsh's opinion is that the death could be safely categorised as presumed SUDEP. This is, in effect, the same opinion expressed by Dr Cooke.

Conclusion as to Cause of Death

82. I accept and adopt the opinions of Dr Cooke and Dr Walsh and find that the deceased's cause of death, whilst unascertained, is consistent with

[113] Transcript 45.
[114] Transcript 29 – 30.
[115] Transcript 44.
[116] Transcript 45.
[117] Transcript 45.
[118] Transcript 51.
[119] Transcript 51.

epileptic seizure and is appropriately categorised as a SUDEP.

MANNER OF DEATH

83. At the time of his death, the deceased was a 10 year old boy living with his grandparents while under the temporary care and protection of the CEO of the Department.

84. On the last night of his life the deceased was happy and appeared well. He went to bed that evening in accordance with his usual bedtime routine and there was nothing to alert anyone that anything out of the ordinary was likely to occur.

85. I accept that the most likely explanation for his sudden death is that he had an epileptic seizure while in bed that evening or in the early hours of the following morning, which led to his death by one of the mechanisms described by Dr Cooke and Dr Walsh.

86. In the remote event that the death was not a SUDEP, the only other likely explanation is that the death was due to a genetic cardiac abnormality.

87. There is no evidence to suggest that death arose by any means other than natural causes.

88. Accordingly, I find that death occurred by way of natural causes.

QUALITY OF SUPERVISION, TREATMENT AND CARE

89. Pursuant to s 25(3) of the *Coroners Act*, where a death investigated by a coroner is of a person held in care, the coroner must comment on the quality of the

supervision, treatment and care of the person while in that care.

90. As noted above, the deceased was a child in the care of the CEO of the Department at the time of his death.[120] His Departmental case workers assisted in forming a plan for his care and supervision on the CEO's behalf.[121] Accordingly, my focus is on the quality of the care provided by the Department and its staff to the deceased.

91. The Department provided extensive materials to this Court in relation to the Department's involvement with the deceased and his sister.[122]

92. Ms Cheryl Barnett, the current Executive Director of Metropolitan Services for the Department, completed a comprehensive review of the Department's files related to the deceased[123] as well as giving oral evidence at the inquest.[124]

93. Consistent with the Department's philosophy of consulting with family in determining care arrangements and encouraging positive and stable relationships with family members where possible, the deceased was placed with his maternal grandparents, together with his sister. This was assessed to be a safe and stable placement for him whilst his parents worked towards reunification.[125] There is no evidence to suggest to the contrary and I am satisfied that this was the best environment for the deceased to be placed in at that time.

94. The Department's records reveal that Departmental officers maintained regular contact with the deceased and they were able to confirm that he was progressing well in his grandparents' care.[126]

[120] Exhibit 3, Tab 4.
[121] Exhibit 3, Tab 3.
[122] Exhibits 3 – 6.
[123] Exhibit 1, Tab 38.
[124] Transcript 61 – 67.
[125] Exhibit 1, Tab 38, Attachment 3.
[126] Transcript 67; Exhibit 1, Tab 38.

95. Because a primary reason for taking the deceased into care was concern surrounding his physical health, he had immediate contact with doctors at PMH after being taken into care. His medical care was managed by Associate Professor Payne, together with a team of medical staff at PMH.[127] The Department was guided by the medical advice provided by the PMH doctors and they ensured that his carers took the deceased to all the appointments scheduled by the doctors.

96. A question does arise as to whether the deceased's medical care was appropriately thorough in terms of investigating the possibility that he had epilepsy.

97. This is not a criticism of the Department, as they were entitled to rely upon the expertise of the doctors at PMH in this regard. However, the evidence of Dr Walsh that the clinical history of the deceased includes sufficient information to make a diagnosis of occipital epilepsy,[128] does raise the question whether this diagnosis should have been made, or investigated, prior to his death, and treatment provided.

98. Associate Professor Payne was aware that the deceased had a reported history of seizures in the past. In 2009, Dr Holmes at PMH had attempted to explore a diagnosis of epilepsy, scheduling a second EEG including sleep state to be performed and recommended seizure safety training and a follow-up appointment with Dr Holmes. Regrettably, the deceased was not brought to the appointments and the hospital lost contact with the deceased's family, so a diagnosis could not be progressed at that time.

99. By the time the deceased came to be seen by Associate Professor Payne in April 2010, he had not had a reported seizure since October 2009. Based upon the information that the deceased had not had

[127] Exhibit 1, Tab 27.
[128] Transcript 44.

any seizures for six months, and noting the somewhat unreliable history of them in the past and the confusion surrounding any possible contribution from non-prescribed clonidine use, he concluded that there was no indication to undertake further investigations to explore the possibility that the deceased had epilepsy at the time he was treating him in 2010.[129]

100. Dr Walsh was asked whether, if the deceased had presented to him in the same circumstances, he would have adopted the same position as Associate Professor Payne in this regard. Dr Walsh indicated that he definitely would have sought a further EEG, because that would help him in his decision-making.[130] He also considered that a clinical history of more than five seizures meant the deceased would have met his criteria for treatment with medication for epilepsy.[131]

101. Even with medication, Dr Walsh indicated that the risk of occurrence of seizures can't ever be completely prevented. However, Dr Walsh described the deceased's form of epilepsy as a relatively benign condition,[132] which in his view would have resulted in a substantially reduced likelihood of seizures if he had been on adequate doses of anticonvulsant.[133]

102. Dr Walsh acknowledged that parental agreement is required to commence medication and not all parents are willing to start their child on anticonvulsant medication immediately.[134] If this had occurred in the case of the deceased, he indicated that he "wouldn't have been too nervous about holding off medication."[135]

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[129] Transcript 28 – 29, 37.
[130] Transcript 53.
[131] Transcript 49.
[132] Meaning that the majority of children will eventually grow out of it and cease having seizures - Transcript 43.
[133] Transcript 52.
[134] Transcript 48.
[135] Transcript 56.

103. Associate Professor Payne was asked at the inquest whether he would have done anything differently, knowing now that it is more than likely that the deceased did have a form of epilepsy. He accepted that a follow-up EEG might have been an option to consider, although given his ongoing observations of the deceased and the difficulty with identifying epilepsy from an EEG, he maintains that it was not really indicated.[136] In terms of anticonvulsant therapy, he considered that the possible adverse effects of medication were a strong factor weighing against starting medication in circumstances where the seizures appeared to have seized.[137]

104. The difference in the opinions of Dr Walsh and Associate Professor Payne may arise from their different areas of expertise.

105. Associate Professor Payne works as a general paediatrician, managing the overall medical care of his young patients for a variety of medical conditions. In the particular case of the deceased, he understood the primary concern to be the need to safely wean him off clonidine use, and once that was achieved the improvement in the deceased's overall health and absence of any report of recent seizures, gave him reassurance that no further investigation was required unless the deceased had another seizure.[138]

106. Dr Walsh specialises in the treatment of epilepsy in children. His expertise in this area leads to a greater focus upon, and understanding of, the clinical history of seizures, which enables him to make a diagnosis from the deceased's clinical history of epilepsy.

107. Acknowledging Dr Walsh's greater expertise in this area, I accept his opinion that, on the basis of the

[136] Transcript 30, 37.
[137] Transcript 36 – 38.
[138] Transcript 37.

deceased's clinical history at the time he was taken into care in April 2010, it would have been optimal medical treatment to order a further EEG (covering sleep as well as wakefulness) to hopefully identify the specific form of epilepsy that the deceased had, and then to explore the option of medication with the deceased's caregivers. Precautionary measures should also have been discussed with the deceased's grandparents, as explained by Dr Walsh during the inquest.[139]

108. However, even if this had been done, it would have provided no guarantee that the deceased would not have died in exactly the same circumstances. Dr Walsh explained that although a child is less likely to die from a relatively benign form of epilepsy, it does still occur. Although it is a rare event, Dr Walsh has personally experienced children and young adults with a so-called benign form of epilepsy dying from SUDEP.[140] It can occur even if precautionary measures such as co-sleeping are adopted, and has been documented several times when a patient has been in an intensive care unit.[141]

109. Therefore, while the preferred approach would have been to conduct further investigations and explore the possibility of giving medication to the deceased to control seizures, it cannot be said that his death would have been prevented if this had been done. While considerable research is being conducted into SUDEP, it is still the case that no complete solution has been found to prevent it.[142]

[139] Transcript 53.
[140] Transcript 50.
[141] Transcript 51 – 52, 60.
[142] Transcript 60.

CONCLUSION

110. The deceased was a ten year old boy who had been taken into the care of the Department under a time-limited protection order due to concerns about his health and welfare.

111. At the time of his death he was living with his grandparents and was thriving and apparently healthy.

112. On the evening of 14 October 2010 he went to bed as usual. He died sometime that night or the following morning. His death was most likely due to complications arising from an epileptic seizure.

113. Although different medical treatment might have reduced the risk of the deceased having a seizure that night, there is no medical treatment that can prevent seizures entirely, or predict every time they will occur.

114. In those circumstances, there is nothing to indicate that the Department could, or ought, to have taken steps to prevent the death of the deceased. His death was a sudden and tragic event that arose as a result of natural causes.

S H Linton
Coroner
18 November 2014