Auckland District Health Board

A Report by the
Health and Disability Commissioner

(Case 18HDC00063)

Health and Disability Commissioner
Te Toihau Hauora, Hauātanga

Contents

Executive summary

1. This report concerns the care provided to a six-year-old girl in 2017. The girl presented to the Emergency Department (ED) with a cough and fever and was discharged with a likely diagnosis of a lung infection (pneumonia).

2. Two days later, the girl was admitted to hospital for treatment of pneumonia. She was transferred to another hospital briefly, but was readmitted to the first hospital for treatment of excess fluid build-up in her right lung. Largely she was cared for in PICU, with input from other services, until her tragic death.

Findings

3. In relation to the girl's first presentation to the ED, the Commissioner found no breach of the Code.

4. However, the Commissioner considered that during the girl's second admission, and prior to her transfer to another hospital, there was a failure to recognise her deterioration, and a lack of consistent and timely escalation of her care (in accordance with the early warning system in place and the girl's lack of improvement).

5. The Commissioner also considered that the cause of her illness was not investigated adequately in a timely manner, and the resulting diagnostic delays meant that empiric antibiotics were not started in a timely manner.

6. The Commissioner concluded that ADHB failed to provide services to the girl with reasonable care and skill, and therefore breached Right 4(1) of the Code.

7. The Commissioner made adverse comment about the guidelines in place at the time, which were not sufficiently clear to guide staff regarding further investigations and treatment of a child with severe pneumonia. The Commissioner also criticised aspects of the care provided by the PICU service.

Recommendations

8. The Commissioner recommended that ADHB provide HDC with an update on the implementation and effectiveness of the revised Pneumonia Guideline and PEWS chart, and audit compliance with these; communicate the changes made to the Pneumonia Guideline to other DHBs; use the findings of this complaint as a basis for training paediatric services staff in management of pneumonia; consider the possibility of further systems improvement to prevent human error in relation to review prior to transfer, such as a nursing checkpoint for escalating the changing condition of a patient prior to transfer, and updating the nursing transfer letter template; further consider the recommendations made by HDC's paediatric intensivist advisor; remind its staff about the importance of full and accurate documentation of clinical care; and provide a written letter of apology to the family for the aspects of care identified as deficient.

Complaint and investigation

9. The Commissioner received a complaint from Mr and Mrs A about the services provided to their late daughter, Miss A, by Auckland District Health Board (ADHB). The following issue was identified for investigation:

- *Whether Auckland District Health Board provided Miss A with an appropriate standard of care in 2017.*

10. The parties directly involved in the investigation were:

Mrs A	Complainant/mother
Mr A	Complainant/father
ADHB	Provider/DHB

11. Also mentioned in this report:

RN B	Nurse specialist
Dr C	Emergency medicine registrar
Dr D	Paediatric registrar
Dr E	Infectious Diseases Service (IDS) consultant
Dr F	General paediatrics consultant
Dr G	PICU doctor
Dr H	PICU consultant
Dr I	Paediatric emergency medicine specialist
Dr J	Paediatric surgeon

12. Further information was received from a second district health board.

13. Independent expert advice was obtained from emergency medicine specialist Dr John Bonning (Appendix A), paediatrician Dr John Doran (Appendix B), paediatric nurse Mr Thomas Gorte (Appendix C), paediatric surgeon Professor Mark Stringer (Appendix D), and paediatric intensivist Associate Professor James Tibballs (Appendix E).

14. Appendix F is a summary of ADHB's serious incident review.

15. Sadly, Miss A died. I extend my sincere condolences to her family for the loss of their precious daughter and sister.

Information gathered during investigation

Introduction

16. This report concerns the care provided to Miss A (six years old at the time of events) at ADHB in 2017. Miss A presented to the Emergency Department (ED) with a cough and fever on 23 Month1,[1] and was discharged. The doctors thought that Miss A might have a lung infection (pneumonia).[2]

17. On 25 Month1, Miss A was admitted to ADHB for treatment of pneumonia. She was transferred to DHB2 briefly, but was readmitted to ADHB on 28 Month1 for treatment of excess fluid build-up in her right lung.[3]

18. Tragically, Miss A died at ADHB on 11 Month2. At the time of her death she was suffering from a strain of the flu[4] and atypical pneumonia.[5] This caused respiratory failure that needed life support,[6] ultimately resulting in swelling and bleeding in her brain.[7]

19. This report examines the standard of care provided to Miss A by multiple services at ADHB.

Presentation to Emergency Department on 23 Month1

20. On 23 Month1, Miss A presented to ADHB ED at around 8.15am, having had a fever and cough for three days. Mrs A recalled that the doctor could not hear the right lung clearly but did not consult a senior doctor or perform further investigation.

21. The clinical notes show that Miss A was examined by emergency medicine registrar Dr C. It was noted that Miss A's sibling had been treated for pneumonia. Dr C's examination findings[8] were normal other than a mild fever and reduced breath sounds.[9] A provisional

[1] Relevant months are referred to as Months 1–2 to protect privacy.
[2] Often referred to as community acquired pneumonia.
[3] Right-sided pleural effusion — pleural effusion is when excess fluid builds up between two layers of tissue surrounding the lung, taking up space in the lung and impairing a person's ability to breathe.
[4] Influenza B.
[5] Pneumonia that may present with a different clinical picture than typical pneumonia and is due to less common organisms that may be more difficult to detect (in this case *Mycoplasma pneumoniae*) and may require different treatment.
[6] Miss A was placed on veno-venous (VV) extracorporeal membrane oxygenation (ECMO). ECMO is a life support machine that replaces the function of the heart and lungs. VV ECMO involves placing cannulae (thin tubes) into a large vein only, primarily to support lung function (getting oxygen into the bloodstream).
[7] Brain stem herniation caused by intracerebral haemorrhages. Brain herniation occurs when increased pressure in the brain from swelling, haemorrhage, or blocked cerebrospinal fluid drainage forces brain tissue through a narrow opening at the base of the skull. Intracerebral haemorrhage is bleeding within the brain tissue.
[8] The examination found that Miss A's temperature was 37.9°C, her heart rate was 115bpm (beats per minute), her respiratory rate was 28 breaths per minute, and her oxygen saturation was 98%. It was noted that Miss A was alert, had no distress, was slightly flushed, mobilised normally, and her heart sounds were dual and no heart murmur could be heard. The examination also found normal work of breathing (ie, the energy used for respiration), quiet breath sounds in the right lower zones, and no crackling sounds (crepitations) or wheeze.
[9] Breath sounds refer to the noises produced by the structures of the lungs during breathing.

diagnosis of lower respiratory tract infection (pneumonia) was made. Miss A was discharged from hospital around 9.30am with antibiotics and pain medication.

22. The discharge summary[10] documented the plan for antibiotics and pain relief medication, and that Miss A's parents had been given safety-netting advice about encouraging fluid intake and when to seek medical attention if Miss A did not improve or her condition worsened.

23. ADHB confirmed that no tests or X-rays were carried out on 23 Month1. ADHB explained that this approach was in line with ADHB Clinical Guidelines, and at this time Miss A did not meet any of the criteria for further investigation such as a chest X-ray.

Presentation and admission to ADHB 25–27 Month1

24. On 25 Month1, Miss A re-presented to ADHB because her condition had worsened. After being assessed in ED, Miss A was admitted to the paediatric ward in the early hours of 26 Month1, with diagnoses of pneumonia,[11] mild dehydration, and red blood cells in her urine.[12] Her chest X-ray from this admission showed pneumonia,[13] as well as a small fluid build-up (pleural effusion) around her right lung.

25. On 27 Month1, the medical team looking after Miss A made a decision to transfer her to DHB2 (the hospital closest to Miss A's home). Nursing notes indicate that shortly before the transfer Miss A began to deteriorate. However, her deterioration was not escalated to the medical team, and the transfer occurred. Shortly after admission to DHB2, Miss A was transferred back to ADHB, for management of the increasing fluid build-up around her lung. The key issues in relation to this admission were the assessments of Miss A's condition and whether there was appropriate escalation of her care prior to her transfer to DHB2 on 27 Month1.

26. The Paediatric Early Warning System (PEWS) identifies patients at risk of clinical deterioration. It is calculated from objective vital sign measures such as respiratory rate, respiratory distress, oxygen saturations, oxygen requirements, heart rate, blood pressure, and capillary refill time. The more abnormal the vital signs, the higher the score. The score determines the recommended level of escalation of care required, as follows:

Total score[14]	Action
0–3	Notification to the nurse in charge at the ward safety briefing, assess the child and record PEWS four-hourly, and for routine doctor review.
4–5	Notification to the nurse in charge within one hour, assess the child and record PEWS one- to two-hourly, and for doctor review within four hours.

[10] Completed by Dr C for the consultant.
[11] Right middle lobe pneumonia.
[12] Microscopic haematuria.
[13] Right middle lobe and right lower lobe pneumonia.
[14] Scores are totalled based on observations of respiratory rate, respiratory distress, oxygen saturations, oxygen requirements, heart rate, blood pressure, and capillary refill time.

6–7	Immediate notification to the nurse in charge, notification to the PaR[15] nurse, assess the child and record PEWS every 30–60 minutes, for PaR nurse review within 15 minutes, for doctor review within two hours, for senior doctor review within four hours, to document the plan including interventions and review timeframe, to consider continuous monitoring, and consider Code Pink.[16]
8+	Immediate notification to the nurse in charge, notification to the PaR nurse ASAP, assess the child and record PEWS every 15 minutes initially and then every 15–60 minutes after review, for doctor review within 15 minutes, for senior doctor review within 30 minutes, to document the plan including interventions and review timeframe, consider continuous monitoring, and consider Code Pink.

27. The table below outlines Miss A's progress during this admission, and also shows occasions on which observations were not documented:

Date	Time	PEWS score	Respiratory distress	Not recorded
26 Month1	3am	1	Nil	
	4.30am	1	Not recorded	Respiratory distress
	8am	3	Nil	
	10am	Not totalled	Nil but 0.5L oxygen started	BP, PEWS score
	11am	Not totalled	Nil	BP, PEWS score
	12pm	Not totalled	Nil	BP, PEWS score
	1pm	3	Nil	
	2pm	7 appears to have been corrected to 9	Mild	BP
	2.40pm	Nurse Specialist (NS) was asked to see Miss A for a score of 9, and the NS's assessment also determined a score of 9.		
	3pm	7 or 8	Mild	
	4pm	8	Mild	
	5pm	7	Mild	
	6pm	7	Mild	
	6.40pm	7	Mild	
	7.50pm	7	Mild	
	8.50pm	Not totalled	Mild	BP, PEWS score
	9pm	NS asked to see Miss A for score of 7; NS's assessment also determined a score of 7.		
	9.50pm	Not totalled	Mild	BP, PEWS score
	10.50pm	6	Mild	
	11.50pm	6	Mild	
27 Month1	1am	7	Mild	
	2am	8	Mild	
	2.30am	9	Moderate	
	2.50am	9	Moderate	
	4am	Not totalled	Mild	PEWS score
	5am	8	Mild	
	6am	6	Mild	
	6.45am	8	Mild	
	7.50am	8	Mild	

[15] Patient at Risk nurse — a specialised nursing role.
[16] Emergency call to summon a rapid response team.

	8.53am	NS RN B asked to see Miss A for score of 9; NS's assessment determined a score of 5.		
	9am[17]	7	Mild	
	10am	9	Mild	
	11am	9	Mild but increased oxygen requirement to 2L	
	11.25am	NS RN B asked to see Miss A for score of 10; NS's assessment determined a score of 7.		
	12pm	Not totalled	Mild and oxygen requirement back to 0.5L	BP, PEWS score
	Unknown time	8	Mild	Time

28. To summarise the above, on 26 Month1, Miss A's PEWS scores had been relatively low (score of 3) until 2pm, when they increased to at least 7.[18] Because of this, Miss A was reviewed by a nurse specialist and a doctor. Dr D, a paediatric registrar, reviewed Miss A at 4.30pm, as she continued to have fevers. He also noted her fast heart rate and considered that she might be developing an inflammatory reaction that was affecting her whole body,[19] but not as a direct result of her infection.[20]

29. Miss A continued to be reviewed over the course of the day and overnight, and her PEWS score fluctuated between 6 and 9. Several of the staff members involved in Miss A's care recall that despite her high PEWS scores, she did not reflect this physically, and looked less unwell than indicated by her scores.

Medical review prior to transfer

30. Dr D reviewed Miss A at 8.30am on 27 Month1. She was noted to be significantly better and drinking water.[21] Dr D's impression was that Miss A was stable, but would likely need several more days in hospital. ADHB stated that during handover, Dr D's impression was discussed with the on-call consultant, who considered that the decision to transfer to DHB2 was reasonable.

31. Mrs A recalled that subsequently a nurse noted that Miss A's condition appeared to have worsened, and Mrs A is concerned that despite this, Miss A was not assessed again by a doctor prior to transfer. Mrs A told HDC that she feels that Miss A should never have been transferred.

32. The clinical notes show that between 8.30am and 1.27pm, the following entries were made:

[17] The time is unclear as it states "09" and is not written in full 24-hour time, and it appears that a "9" was drawn over an "8".

[18] The score on the chart is unclear, as it appears that the number was written over the top of another number.

[19] Systemic inflammatory response syndrome (SIRS) — this occurs when the body mounts an over-vigorous response to a stimulus such as infection or trauma, with the body's response having potential to cause further harm, including failure of multiple organs.

[20] Not sepsis.

[21] It was noted that she had a heart rate of 120–130bpm, oxygen requirements of 0.5L, comfortable work of breathing, and a respiratory rate of 30 breaths per minute. She did not have a fever at the time of review.

- At 9am, NS RN B reviewed Miss A for surveillance and for a PEWS score of 9, although the PEWS chart documented a score of 7, and RN B's assessment determined her PEWS to be 5. RN B told HDC that Miss A was certainly a child well enough to be at DHB2, and was not of concern that day or requiring increased monitoring.
- At 10am, Miss A's PEWS had increased to 9.
- At 11am, Miss A's PEWS remained at 9, and her oxygen requirement increased to 2L. Prior to this, it had consistently been 0.5L since around 10am on 26 Month1.
- At 11.25am, RN B reviewed Miss A for an increased PEWS score of 10, but RN B's assessment determined Miss A's PEWS score to be 7.
- At 12pm, Miss A's oxygen requirement had reduced to 0.5L.
- At 1.50pm, a nurse documented that during the shift, Miss A's respiratory rate had increased to about 50–60, with mild work of breathing and shortness of breath on exertion at times. The nurse stated that she did not have any clinical concerns about Miss A, as she was one of the more well children she was looking after that day despite the scoring on PEWS, and she looked well enough to transfer to DHB2. The nurse said that the charge nurse completed the clinical case review with her, and she recalls that the charge nurse was also happy with the transfer.
- The last recording on the PEWS chart was a score of 8 (time unknown).

33. At approximately 1.27pm, Miss A was transferred to DHB2.

34. ADHB stated that the decision to transfer Miss A, made at 8.30am by Dr D, was very reasonable based on his assessment at the time. However, ADHB also stated that Miss A had two subsequent specialist nurse reviews, and then clinical notes written by her ward nurse that document deterioration in Miss A's observations, but the medical team were not informed of this change.

35. ADHB accepts that there was a failure to escalate a degree of clinical deterioration on 27 Month1. It stated that the above changes in Miss A's observations (ie, between Dr D's review and Miss A's transfer) were not escalated to medical staff, and Miss A was not reviewed again by a doctor prior to her transfer to DHB2. ADHB told HDC that had escalation occurred, Miss A may not have been transferred to DHB2.

36. ADHB stated that mandatory reporting is not in place for increased PEWS, and the escalation response requires clinical judgement. ADHB noted that a child who has developed a fever, associated fast heart rate,[22] and abnormal rapid breathing, in the context of no respiratory distress with a planned specialist team review, may not require escalation to an urgent medical review of care at the time of trigger. The reporting and escalation of PEWS is based on recommendations as outlined on the PEWS chart, and should not replace the nurse's judgement to ensure that all aspects of the situation are considered. ADHB noted that this in turn also encourages the use of clinical judgement to

[22] Tachycardia.

report children with low PEWS scores who are felt to be clinically unwell, rather than waiting for a trigger.

Transfer documentation

37. The discharge summary documented that Miss A was discharged at 1.27pm, and outlined her history, examination, investigations undertaken, and progress. It also suggested follow-up for blood cells in her urine (haematuria) when she was well.[23]

38. The nursing transfer letter noted Miss A's diagnosis of "lobar pneumonia[24] Day 4". It outlined her history, and that her "problem list" included oxygen requirement (noted as 0.5L), mild work of breathing and decreased breath sounds in the right lower lobe, and a productive cough. The nurse specialist reviews that occurred were not noted on this letter.

Summary of admission to DHB2 27–28 Month1

39. Miss A was admitted to DHB2 at 3.49pm on 27 Month1 and was a patient there until around 10am on 28 Month1. She was reviewed by the paediatric service during her admission. At 8.30am on 28 Month1, a paediatric consultant documented that Miss A's father and nursing staff were reporting increased work of breathing, fevers, and increased oxygen requirement. The paediatric consultant documented that on examination, Miss A looked septic and miserable.[25]

40. The paediatric consultant documented an impression of pus build-up around the outside of the lungs[26] as a result of worsening pneumonia. A chest X-ray and chest ultrasound showed a large fluid build-up around the right lung.[27] It was planned to transfer Miss A back to ADHB, and for her to be nil by mouth in view of probable surgery for a chest drain (to drain the fluid from around her lung).

41. The nursing notes document that ADHB was contacted about the transfer for further management, and that Miss A's PEWS score was now 12, with her oxygen requirement increased to 2.5L, a temperature of 40.3°C, heart rate of 155bpm, and respiratory rate of 52. Miss A was transferred back to ADHB at around 10.10am on 28 Month1.

Summary of readmission to ADHB: 28 Month1–11 Month2

42. The key events of this admission are summarised below.

[23] Via protein creatinine ratio, calcium creatinine ratio, and urine microscopy.
[24] A type of pneumonia that affects the lobe or a large section of a lung.
[25] Her respiratory rate was greater than 50, her oxygen saturations were 95% on 2L of oxygen, and she had stony dullness and no audible breath sounds in her entire right anterior chest wall consistent with fluid or pus within the chest cavity on that side.
[26] Right-sided empyema (pus within the space between the lung and the lining of the chest cavity).
[27] A large right-sided pleural effusion.

Paediatric surgical and general paediatric care

43. Following transfer back to ADHB on 28 Month1, Miss A was assessed and reviewed by the paediatric and paediatric surgical services. The paediatric registrar noted: "[I]mportant to send culture for microscopy and pneumonia/[P]olymerase [C]hain [R]eaction panel.[28]"

44. The paediatric registrar documented that Miss A's management had been discussed with the Infectious Diseases Service (IDS), and that following surgery (chest drain insertion), and depending on the result of the test for pneumococcal disease,[29] further antibiotics might need to be added. It was therefore important that the drained fluid was sent for investigations.[30]

45. A chest drain in Miss A's right lung[31] was inserted at 5.30pm under sedation. The operation record noted that fluid[32] was collected and sent for testing (culture) to identify the type of bacteria responsible for Miss A's pneumonia. A *Streptococcus pneumoniae* antigen test[33] was also carried out.

46. Miss A was then transferred to the Post Anaesthetic Care Unit (PACU) for care following the insertion of her chest drain, and later admitted to the general paediatric ward at 7.00pm.

47. On the general paediatric ward, Miss A's PEWS score increased, reaching 13, and her oxygen requirement was 6L. The documented impression was of a SIRS reaction.[34] A chest X-ray was taken, and it was documented that if there was further deterioration, Paediatric Intensive Care Unit (PICU) review and high flow nasal prong (HFNP) therapy (a form of non-invasive respiratory support to help her breathe[35]) should be considered.

48. Overnight, Miss A was noted to be stable and showed slight improvement. At 7am, the PICU registrar reviewed her and, following discussion with the PICU consultant, agreed that she would benefit from PICU admission.

Paediatric Intensive Care Unit

49. On 29 Month1, at around 7.30am, Miss A was transferred to PICU and started on HFNP.

[28] Respiratory PCR panel.
[29] Pneumococcus (*Streptococcus pneumoniae*) is the most common bacterial cause of pneumonia acquired in the community.
[30] It noted: "[A]fter theatre — if pneumococcal antigen positive then continue IV cefuroxime alone, but if negative/or result not back also add on clindamycin at 50mg/kg/dose … [T]herefore important that drain fluid is sent for MC+S as well as pneumococcal antigen in theatre please ..."
[31] An intercostal (space between two ribs) chest drain.
[32] Pleural fluid.
[33] A rapid test to detect the presence of *Streptococcus pneumoniae* antigen (usually in the urine), a positive test suggesting pneumonia caused by *Streptococcus pneumoniae* (a kind of bacteria).
[34] See footnote 19.
[35] HFNP involves delivery of humidified oxygen at high flow via nasal tubes. HFNP may act as a bridge between low flow oxygen therapies and invasive ventilatory support that requires insertion of a tube down the throat.

50. The PICU registrar had a meeting with Miss A's family and documented that he explained that the "bug" responsible for Miss A's pneumonia was still unknown and was being covered by broad-spectrum antibiotics, which would be narrowed if the "bug" was identified.

51. On 29 Month1, at 3pm, the results of the *Streptococcus pneumoniae* antigen test on the urine taken the previous day returned a negative result.

52. On 30 Month1, Miss A's condition was noted to be stable with a slight improvement clinically. ADHB said that after Miss A's initial improvement, she deteriorated overnight on 30–31 Month1.

53. On 31 Month1, Miss A's chest drain fell out while she was being transferred from her bed to the commode chair. It is documented that reinsertion of the drain was considered but decided against.[36] A paediatric surgical registrar reviewed Miss A and documented a plan to follow up the microbiology results (to identify the "bug" causing her pneumonia).

54. On 1 Month2, an ultrasound showed only a small collection of fluid around her lung, but extensive lung consolidation.[37] Miss A remained on HFNP as she did not tolerate lower oxygen flow, and she continued to have ongoing temperature spikes.

55. The clinical notes document the need to follow up the microbiology results and tests for any viral infection or gut infection. The nursing notes also record that a stool culture was negative for all viruses.

56. On 2 Month2, the surgical registrar noted that no obvious infective agent had been identified on the test results currently available. Following this, IDS documented (at an unknown time) the suggestion of a test for pneumococcal disease[38] on the fluid drained from around Miss A's lung. At 3.14pm, the *Streptococcus pneumoniae* antigen test of the pleural fluid collected on 29 Month1 was reported as negative.

57. On 3 Month2, a paediatric IDS registrar review noted that the IDS team was happy to continue with single antibiotic treatment. A typed note by a PICU consultant recorded that no cause of Miss A's disease had been identified, with streptococcal tests being negative, and that a specimen had yet to be taken from her main airway (trachea).

58. A chest X-ray showed worsening right lung consolidation and deterioration in chest appearance since the previous study, and a repeat ultrasound that afternoon showed an increased amount of fluid around the lung. Miss A was reviewed by the surgical registrar,

[36] A chest X-ray was taken to review the lung fields. The notes by the PICU registrar and the surgical registrar indicate that a possible plan for reinserting the chest drain was considered and Miss A was kept nil by mouth for this possibility. However, following discussions with the surgical team, general paediatric team, and radiology team, and following an ultrasound, it was decided not to insert a chest drain, as there was only a small amount of fluid remaining from the previous collection.
[37] The air that usually fills the small airways of the lung is replaced with something else (eg, pus).
[38] Pneumococcal antigen test.

and it was decided to drain the fluid the following day via minimally invasive chest surgery.[39]

59. Miss A underwent the surgery on 4 Month2 and was commenced on mechanical ventilation.[40] Two chest drains, a nasogastric tube,[41] and a PICC line[42] were inserted (this is a long tube inserted into one of the body's larger veins (usually in the arm) to enable long-term access to the vein for IV fluids, medications, blood tests, etc). Fluid was suctioned out during the procedure, and a sample was sent for microbiology testing.

60. The paediatric surgeon who performed the procedure documented on his operation note that consideration should be given to trying other antibiotics while the cause of Miss A's pneumonia was still unknown, and to consider other diagnoses and causes for Miss A's condition.

61. After surgery, Miss A returned to PICU on a ventilator and with a breathing tube in place. Unfortunately, postoperatively Miss A deteriorated further.

62. On the morning of 5 Month2, Miss A was reviewed by the surgical registrar, who documented that she had shown increased signs of sepsis overnight and required increased respiratory support.

63. A chest X-ray at 8.54am showed the presence of air in the tissues under the skin (subcutaneous emphysema), which had developed since the previous imaging and was very extensive.

64. The PICU team's progress report noted the plan to follow up Miss A's test results (cultures).

65. Later that day, a paediatric IDS registrar documented that currently Miss A was on only one antibiotic, and that this should be adequate for the typical causes of pneumonia.[43] However, the registrar also noted that the antibiotic was not covering an atypical cause (Mycoplasma infection).[44] The registrar recorded in Miss A's medical notes that this would be an unusual cause of such disease, but that because of the lack of improvement in Miss A's condition, consideration should be given to adding a different antibiotic (from the macrolide class[45]) that would cover the possibility of Mycoplasma infection. At 12.45pm, a new antibiotic (erythromycin) was started.

[39] Using video-assisted thoracoscopic surgery (VATS) — a surgical technique used to diagnose and treat problems involving the chest area, and involving insertion of a tube containing a camera and other instruments as needed through one or more small incisions in the chest.
[40] Intermittent positive pressure ventilation (IPPV) — use of a breathing machine (ventilator) to provide oxygen to the lungs.
[41] A tube passed through the nose, down the throat, and into the stomach.
[42] A peripherally inserted central catheter.
[43] It was noted that clindamycin should be adequate antibiotic cover for MRSA (methicillin-resistant *Staphylococcus aureus*), SA (*Staphylococcus aureus*), Group A Streptococcus, or Pneumococcus.
[44] See footnote 5.
[45] A class of antibiotics used to treat Mycoplasma pneumonia.

66. A chest CT scan taken at 2.45pm showed extensive lung changes.[46] Miss A's significant overall deterioration was documented. At 8pm, a paediatric surgeon documented that Miss A appeared to have changes suggesting that the middle lobe of her right lung was being destroyed (necrosis).

67. Overnight nursing notes[47] document that Miss A was critically unwell and potentially heading for life support measures (ECMO). ECMO is extra-corporeal membrane oxygenation — essentially an artificial lung (heart/lung machine). Blood is pumped from the body into a machine that provides oxygen to the blood, which is then pumped back into the body, bypassing the lungs.

68. On 6 Month2, a chest X-ray showed further deterioration since the imaging taken the previous day. There was complete white-out on the right lung (no air was visible in the lung), and the left lung had deteriorated.[48]

69. The CT aspirate taken the previous day was tested and showed no specific infecting organism when examined under the microscope (a negative Gram stain) or on antigen testing for *Streptococcus pneumoniae*.

70. On 7 Month2, a PICU consultant documented that the cause of Miss A's pneumonia remained unknown. He recorded that Miss A remained critically ill, and that the prospect of undergoing surgery to remove the lung or part of the lung "remain[ed] daunting at the moment". He noted:

> "[I]t is possible that ECMO may offer salvage therapy if further deterioration occurs. [Miss A's] parents have been included in all discussions and are well aware of the seriousness of her current situation."

71. At 2.15pm, surgery for the insertion of ECMO was performed by a paediatric cardiac surgeon.

72. From around 4pm onwards, Miss A had a period of high blood pressure.[49] The drug milrinone was started at around 8pm to dilate (open up) blood vessels and lower the blood pressure.

73. At 5.15pm, a sample of fluid (aspirate) was taken from the back of Miss A's throat and sent for further testing. The result identified influenza B, but it is unclear when the result was reported.

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[46] The scan showed widespread air trapping under the skin (surgical emphysema/subcutaneous emphysema); extensive lung consolidation, with pus or other fluid or solid matter directly within the lung tissue — the right lung worse than the left; gas-filled cysts (pneumatoceles) in the right lung, possibly related to ventilator-induced injury or the underlying infection; fluid around the right lung that had formed into pockets (loculated effusion) with some fluid also around the left lung (simple effusion); small areas of dead lung tissue (parenchymal necrosis) in the right lung; and the development of a possible abnormal connection between a larger airway in the right middle lobe directly to the outside of the lung (bronchopleural fistula).
[47] From 7pm on 5 Month2 to 7am on 6 Month2.
[48] The left lung had deteriorated with now patchy consolidation throughout.
[49] BP 110–140/65–95, with an hourly MAP average of 97mmHg.

74. On 8 Month2, the morning surgical review noted that Miss A's blood chemistry test results and observations of blood pressure and pulse had improved on ECMO. The plan was to watch and wait.

75. Miss A was on heparin (blood-thinning therapy) for VV-ECMO. From approximately 3am until 9am on 8 Month2, the tests monitoring her response to heparin (ACT and APTT) were outside the desired range.[50] This was documented as managed "according to protocol".

76. At 10.10am, an IDS registrar reviewed Miss A and documented that she had appropriate antibiotic cover for atypical causes of pneumonia and was on the maximal dose of both antibiotics.[51] The registrar suggested awaiting further pending test results before any change in management.[52] IDS consultant Dr E documented next to these entries (at an undocumented time) that Miss A's case was "highly suspicious of mycoplasma based on history — [sibling's] prior illness — and rash". The registrar documented that they explained to Miss A's mother that Miss A was on the best antibiotics for her current situation, and that the tests pending were to help them to figure out what had caused the illness.

77. At 5pm on 8 Month2, Miss A's pupils were checked and noted to be fixed and dilated. A CT scan showed a devastating brain bleed that was non-survivable. A PICU consultant documented that this was explained to Miss A's family in a meeting, and that in the interim she was to receive comfort cares.

78. On 9 Month2, Miss A was continued on life support measures while awaiting the arrival of her family from overseas before stopping treatment.

79. At around 2.30pm on 9 Month2, the results of the atypical pneumonia PCR test on the pleural fluid collected during Miss A's surgery on 28 Month1 and on 4 Month2 were reported. These tests had been ordered on 7 Month2. The results showed that Miss A had atypical pneumonia caused by *Mycoplasma pneumoniae*.

80. On 10 Month2, Miss A's extended family arrived and discussions around ceasing supports took place that night. In the evening, the PICU consultant documented that there had been discussions about a coronial inquiry, and the Coroner was contacted. Miss A's parents did not wish for a post mortem to be performed, and they decided that they did not want a coronial inquiry. At 5pm, testing confirmed brain death.

81. On 11 Month2, Miss A was placed in her mother's arms, with her father near and her family present. Miss A was extubated and ECMO stopped. Miss A died at 9.30am.

[50] The activated clotting time (ACT) levels remained beyond the desired range (160–180 seconds) at a heparin dose of 10u/kg/hr. During this time the platelet count was adequate but the activated partial thromboplastin time (APPT) was beyond the therapeutic range (50–80 seconds) at 96 seconds at 3am.
[51] Erythromycin and clindamycin.
[52] Pending results included pleural fluid immunoglobulins, and the Atypical Pneumonia PCR Panel on the tracheal aspirate that had been taken on 6 Month2. The panel tests for a variety of atypical causes of pneumonia, including *Mycoplasma pneumoniae* (see ADHB Test Guide).

28 Month1 to 11 Month2 — investigations into the cause of Miss A's illness

82. The key issue in relation to Miss A's re-admission to ADHB relates to the timeliness of the investigations into the cause of her illness.

83. Mrs A considers that there was a delayed diagnosis of the cause of Miss A's pneumonia, and a lack of further investigation to identify and treat the pneumonia appropriately despite Miss A's worsening condition. Mrs A also recalled that her suggestion (after consulting with medical professionals overseas) to consider giving medication for Mycoplasma was ignored by staff. Mrs A considers that the doctors should have performed tests to identify which type of pneumonia Miss A had prior to giving her broad-spectrum antibiotics.

84. ADHB advised HDC:

> "We acknowledge that there was a delay in the diagnosis of the non-bacterial causes of [Miss A's] pneumonia, including both mycoplasma and influenza. Most guidelines would suggest that tests for these causes should occur when pneumonia is severe and/or progressive despite standard antibiotic treatment. Our hospital clinical guidelines were not clear about this and have subsequently been amended. We sincerely apologise for this omission in her care."

85. ADHB agreed that when Miss A returned to ADHB she had pneumonia that was not responding to treatment and was becoming more severe. ADHB accepted that testing for viral and atypical causes, including a nasopharyngeal aspirate, should have occurred around 1–2 Month2 when Miss A's pneumonia was worsening. It noted that certain diagnostic tests[53] could not have been done until Miss A was intubated, which occurred on 4 Month2. It agreed that alternative causes of Miss A's pneumonia should have been considered earlier.

86. ADHB confirmed that the Mycoplasma PCR on the pleural fluid collected on 28 Month1 (which identified *Mycoplasma pneumoniae*) was asked for retrospectively by the ID consultant who reviewed Miss A on 7 Month2, and therefore this test was not performed and results not available until after 7 Month2. It considered that all other tests were followed up in a timely way.

87. ADHB explained that in relation to viral causes, these were considered and assumed likely, and that in Miss A's case there may have been a bacterial infection in addition to a viral infection. Viral causes or secondary bacterial infection on top of a viral infection are all common. Viral and other bacterial causes are far more likely than Mycoplasma to cause severe necrotising pneumonia,[54] which Miss A developed. Testing for viruses does not influence management, as there are no treatments for this apart from oseltamivir[55] early in the course of influenza. Any possible minor benefit of oseltamivir is described when

[53] A tracheal aspirate and broncho-alveolar lavage.
[54] Necrotising pneumonia is a rare and severe complication of bacterial community-acquired pneumonia. It causes significant damage to lung tissue.
[55] Oseltamivir is an antiviral medication used to treat influenza.

started early (within the first 2–3 days). At the time when Miss A became severely unwell, when testing was appropriate, she was more than 10 days into her illness.

Guidelines for pneumonia

88. The guideline in place at the time of events is outlined below under "Changes made". At the time of events, it did not include specific guidance for the investigations to be undertaken and when.

89. ADHB advised:

> "[W]e agree that the ADHB guideline for severe pneumonia was not as clear as it could be in terms of testing for viral and atypical causes in children with severe pneumonia. The guideline has been updated and considerable effort has been put in to education about this."

90. Further detail about the updated guideline can be found under "Changes made", and in Appendix F.

Documentation

91. During Miss A's admission, on multiple occasions reviews were either not documented, or the time or person who performed the review was not recorded or was unclear, and on occasion the notes were incorrect.

92. ADHB acknowledged that "documentation of surgical and medical procedures and consultations could have been better in Miss A's case".

Further information

Miss A's family

93. Mrs A stated:

> "[Miss A] was a very healthy girl and had great talent in swimming. She was also very active and played different sports … She was in and out of the Hospital for few times. During her time there, doctors used wrong antibiotics on her. All treatments [Miss A] had received did not help her to get better and even made her body worse and weaker. As parents we were very worried to see our lovely daughter's condition getting worse and worse. We felt helpless and only trusted that doctors can help [her]. Unfortunately my daughter never got better at ADHB and she passed away on 11 [Month2]. The whole family and friends were shocked and felt heartbroken for losing her. As parents we are not satisfied about the treatment my daughter had received for pneumonia at [ADHB]. We believe that if her illness had been correctly tested and treated at the earliest stages my daughter would not leave us. We brought her to [ADHB] quite early and we could not accept the sad news. It was a disaster for the whole family and we could not believe this happened in New Zealand."

94. Mrs A also stated that they would like to know what actions the hospital will take to prevent it from happening to another child in the future.

ADHB

95. ADHB extended its sincere sympathies to Miss A's family, and stated that the staff involved in Miss A's care were deeply saddened when Miss A died. ADHB acknowledged how traumatic this would have been for Mr and Mrs A.

Responses to provisional opinion

Mr and Mrs A

96. Mr and Mrs A were given the opportunity to comment on the "information gathered" section of the provisional opinion. Mr and Mrs A wished to convey that they felt there was a lack of communication about Miss A's condition and treatment, and concerns about her symptoms and level of nutrition were ignored. They also recalled requesting a change in antibiotics and asking doctors to investigate to find out the cause of her condition but their concerns were not listened to, and they felt helpless to see their daughter suffer from her illness.

97. Mr and Mrs A felt that the main reason for Miss A's death was that she did not get the right diagnosis early enough, and her condition was treatable and curable but medical treatment she received did not help her at all. They are concerned that Mycoplasma pneumonia was not considered, important test results came too late, and there was a delay in receiving effective treatment, which should not have happened and could have been avoided. Mrs A said:

> "We lost our dearest daughter. We are very dissatisfied and disappointed for the treatment outcomes and poor patient care. I am writing in tears, my husband and I blame ourselves for losing such a lovely daughter and [Miss A] is greatly missed by her family and friends."

ADHB

98. ADHB was given an opportunity to comment on the provisional opinion, and its response has been incorporated below where relevant. ADHB advised that it continues to disagree on a number of aspects of the care provided, but it accepts my recommendations and believes they are balanced and orientated toward improving its systems.

Opinion: ADHB — breach

Introduction

99. Having reviewed this deeply saddening case, it is clear that Miss A's presentation was complex and atypical. At the outset, I offer my sincere condolences to Mr and Mrs A for the loss of their precious daughter in such tragic, unexpected circumstances.

100. As a healthcare provider, ADHB is responsible for the operation of the clinical services it provides, and for providing these services in accordance with the Code of Health and

Disability Services Consumers' Rights (the Code). Miss A had the right to have services provided to her with reasonable care and skill.

101. Numerous clinical opinions have been sought on the care that was provided to Miss A in Month1 and Month2. I discuss these, with respect to the key issues of each of Miss A's presentations and admissions, below.

ED presentation on 23 Month1 — no breach

102. I refer to the paragraphs above under "Presentation to Emergency Department on 23 Month1".

103. My expert advisor, Dr John Bonning, an emergency medicine specialist, advised that the assessment and management of Miss A at this presentation, including discharge with oral antibiotics, was appropriate and met the expected standard of care.

104. When assessing whether the ED clinicians provided Miss A with care of an appropriate standard I must assess that care against the standards of the ED specialty. While I note that my expert paediatric intensivist advisor, Associate Professor James Tibballs, was broadly critical of the history-taking, investigations, and treatment at this consultation, I consider it more appropriate to rely on Dr Bonning's advice (being the direct peer of the doctors involved), and am satisfied that the care provided to Miss A on this date met accepted standards.

Care provided during 25–27 Month1 — breach

105. Miss A was re-admitted to ADHB on 25 Month1. The key issues in relation to this admission relate to the nursing assessments of Miss A's condition, and whether there was appropriate escalation by nursing staff of her condition prior to her transfer to DHB2 on 27 Month1. To assist with my assessment of these issues, I have relied on the advice of my paediatric nursing advisor, Mr Thomas Gorte, and my paediatric advisor, Dr John Doran.[56]

Nursing assessments of condition during Miss A's admission

106. I refer to paragraph 26.

107. Mr Gorte advised that from admission, there were errors in the observation chart and several gaps with no PEWS scores recorded, when blood pressures were not measured. He noted examples where the recommended actions on the observation chart based on PEWS were not consistently adhered to, such as a senior doctor review within a specific timeframe, or increased recording of observations. He stated that while mandatory reporting is not a requirement and clinical judgement is to be respected, the introduction

[56] Dr Doran advised that the clinical assessments and investigations during this admission were consistent with the primary admitting diagnosis. He advised that Miss A's treatment with IV fluids and IV antibiotics was appropriate, and noted that at this point it was not usual to investigate for possible aetiological infecting organisms routinely. He advised that investigations such as nasopharyngeal swabs or aspirates for viral or atypical pneumonia were not indicated at the time, and that Miss A's clinical picture was still evolving. He considered that the clinical team caring for Miss A acted as would be expected in response to her signs and symptoms at the time.

of the PEWS was in response to previously missed subtle changes in a patient's condition and early intervention opportunities. He advised that while an escalation secondary to the elevated PEWS may not have led to a change in circumstances, it is paramount that collaborative clinical review (between doctors and nurses) occurs.

108. Mr Gorte advised that the lack of consistent evaluation and timely escalation, based on the PEWS score and lack of improvement of Miss A's condition, was a moderate departure from accepted practice. He also considered that the specialist nurse team (who are regarded as experts to whom nurses look for support and guidance) should have provided some additional advice to nursing staff on the occasions on which Miss A's observations were escalated to the specialist nurse team, including encouraging increasing the frequency of observations.

Nurses' lack of recognition of deterioration on 27 Month1 prior to transfer to DHB2

109. At 8.30am on 27 Month1, Miss A was assessed by the paediatric registrar, who, in consultation with the on-call consultant, considered Miss A to be stable for transfer. As my paediatric advisor, Dr John Doran, notes, at the morning round Miss A was noted to "feel significantly better today". Her PEWS score at 8.55am was 5, having improved from 9 at 2.30am and 7 the previous evening, which supported the view of some clinical improvement with interventions. Dr Doran does not believe that the transfer to DHB2 caused delay in more definitive treatment, as the extent of pleural effusion was diagnosed rapidly at DHB2.

110. However, between the decision to transfer, made at 8.30am, and the actual transfer at approximately 1.30pm, Miss A deteriorated, and either this was not appropriately recognised or appropriately escalated by nursing staff. This meant that medical staff, who were responsible for the decision to transfer, were unaware of the changes in Miss A's condition.

111. I refer to the paragraphs above under "Presentation and admission to ADHB 25–27 Month1 — Medical review prior to transfer". Mr Gorte advised that between 7am and 1.30pm on 27 Month1, there were clinical signs of deterioration. He considered that Miss A's lack of improvement and her deterioration despite four to five days of antibiotics, including 24 hours of IV cefuroxime, was concerning, and that the lack of progress following this suggested an atypical chest infection.

112. Mr Gorte was concerned that there was no request for Miss A to be reviewed by a senior doctor following the 11.25am specialist nurse review on 27 Month1, despite the increased use of oxygen from 0.5L to 2L, a respiratory rate of 50 scoring 4 on the PEWS chart, and borderline hypotension (low blood pressure). He noted that the PEWS scores from 2am until transfer were mostly 8 or higher. He considered that it would have been prudent for the specialist nurse to request a senior medical review based on the increased evidence of work of breathing and possible circulatory compromise. He noted that the importance of ensuring the stability of a patient prior to transfer is significant.

113. After consideration of Mr Gorte's advice, ADHB accepted that there was a failure to escalate a degree of clinical deterioration on 27 Month1. It acknowledged that the changes

in Miss A's observations (i.e., between Dr D's review and Miss A's transfer) were not escalated to medical staff, and Miss A was not reviewed again by a doctor prior to her transfer to DHB2. ADHB told HDC that had escalation occurred, Miss A may not have been transferred to DHB2.

Transfer documentation

114. Mr Gorte advised that the information provided in the nursing transfer letter for DHB2 was missing the multiple specialist nurse team reviews, and that the health of the child had deteriorated since her first presentation. He advised that this did not accurately reflect the condition of the patient, and considered this to represent a moderate departure from accepted standards.

Conclusion — care provided during 25–27 Month1 admission

115. Guided by Mr Gorte's advice above, I am concerned about aspects of the nursing care provided during Miss A's admission on 25–27 Month1.

116. In relation to the issue of transferring Miss A to DHB2, I am not critical of the decision made at the 8.30am medical review, to transfer Miss A. On the information available to me, it was reasonable to make that decision based on the information known to the medical staff at the time. I also acknowledge the submission made by ADHB that it was likely that the transfer did not contribute to Miss A's deterioration, and I note that Dr Doran advised that this was unlikely to have delayed more definitive treatment.

117. However, I am critical of the nursing assessments undertaken during Miss A's admission, and that nursing staff failed to recognise Miss A's deterioration and escalate it to medical staff prior to her transfer at 1.30pm. This was a missed opportunity to arrange a further medical review, and a reassessment of the decision to transfer Miss A. I note that ADHB acknowledged that while it may not have affected the ultimate outcome, the decision to transfer would have been re-evaluated and possibly deferred given the change in Miss A's observations.

118. It also concerns me that the nursing transfer letter did not contain all the appropriate information and accurately reflect Miss A's condition. I stress the importance of documentation and communication between providers to ensure continuity of care.

119. For these reasons, I consider that by staff members' lack of consistent evaluation and timely escalation, based on the PEWS score and lack of improvement of Miss A's condition during her 25–27 Month1 admission, and the lack of recognition of Miss A's deterioration and escalation prior to her transfer on 27 Month1, ADHB failed to provide Miss A with services with reasonable care and skill, and therefore breached Right 4(1) of the Code.[57]

[57] Right 4(1) states: "Every consumer has the right to have services provided with reasonable care and skill."

Care provided 28 Month1–11 Month2 — breach

Delay in identifying cause of Miss A's illness and in starting empiric antibiotics during 28 Month1 admission

120. The key issues in relation to this admission relate to the delay in diagnosing the cause of Miss A's illness, and the delay in starting empiric[58] antibiotics.

121. Before discussing this aspect of Miss A's care it is necessary to address concerns raised by ADHB that the opinion of my expert advisor, Associate Professor James Tibballs,[59] was biased by hindsight, and that he was not appropriately qualified to give an expert opinion on ECMO (discussed further below under "Care provided by PICU service"). ADHB also contended that Associate Professor Tibballs' opinion extended beyond his specialty area. Associate Professor Tibballs has satisfied me that he is appropriately qualified to give an opinion on ECMO processes and protocols. In addition, I am always mindful of bias, and to that end assess the evidence with reference to the information available to clinicians at the time of the care. I have therefore approached Associate Professor Tibballs' evidence with the same lens. I have also sought specific expert evidence on the areas of care outside Associate Professor Tibballs' direct speciality (nursing advice, emergency physician advice, and general paediatric advice). As evident in this opinion, I have relied on those experts as peer reviewers for those specific episodes of care.

122. Miss A returned to ADHB on 28 Month1 in worsening condition. Throughout her clinical notes for this admission, there are several references to not knowing the cause of her illness, and the need to identify this. Despite this:

- Empiric antibiotics to cover atypical cause/s were not started until 5 Month2, being Day 9 of her admission.
- Anti-viral treatment/medication was not given at all.[60]
- The Infectious Diseases Service (IDS) was first consulted on 28 Month1, before Miss A was admitted to PICU, but this was not documented. The next documented review by IDS was on 2 Month2 (her first review while in PICU).
- Influenza B was identified at some point on or around 7 Month2.
- The Mycoplasma PCR test on the pleural fluid collected on 28 Month1 was asked for retrospectively on 7 Month2. Mycoplasma pneumonia was identified on 9 Month2.

123. Miss A was admitted to PICU on 29 Month1, and remained there until her death. My paediatric intensivist advisor, Associate Professor Tibballs, considered that the PICU staff,

[58] Empiric therapy refers to treatment initiated prior to a definitive diagnosis, based on observations and experience.

[59] Associate Professor Tibballs is a senior intensive care physician at the Royal Children's Hospital Melbourne, Australia.

[60] ADHB stated that viral causes were considered, and that testing for viruses does not influence management as there are no treatments for this apart from oseltamivir early in the course of influenza, and any possible minor benefit of oseltamivir is described when started early, and at the time of Miss A becoming severely unwell when testing was appropriate, she was more than ten days into her illness.

like other members of the healthcare teams, did not consider Mycoplasma or viral causes of pneumonia. He advised that PICU staff had the responsibility to diagnose the cause of Miss A's pneumonia, but further investigations, in particular the nasopharyngeal aspirate (7 Month2), did not occur until late in the course of her illness. He also noted that erythromycin (the antibiotic used to cover an atypical cause of pneumonia) was started "very late in the course of the illness".

124. Associate Professor Tibballs also considered that the IDS should have been consulted earlier during Miss A's PICU admission, from around 29 Month1.

125. ADHB acknowledged that there was a delay in the diagnosis of the non-bacterial causes of Miss A's pneumonia, including both Mycoplasma and influenza.

126. ADHB agreed that when Miss A returned to ADHB on 28 Month1 she had pneumonia that was not responding to treatment and was becoming more severe. It accepted that testing for viral and atypical causes, including a nasopharyngeal aspirate, should have occurred around 1–2 Month2, when her pneumonia was becoming more severe. It also accepted that alternative causes of Miss A's pneumonia should have been considered earlier.

127. ADHB advised:

> "Our previous review and Professor Tibballs' report raise important issues about testing for atypical and viral causes of severe pneumonia in patients not responding to treatment. We acknowledge this should have happened and we have improved our guidelines to assist this to occur."

128. ADHB noted that certain investigations — namely a tracheal aspirate and broncoalveolar lavage — could not have been done until Miss A was intubated (which occurred on 4 Month2). In response, Associate Professor Tibballs advised that the decision to intubate ought to have been taken earlier than it was, especially since the actual cause of the pneumonia was unknown and Miss A was deteriorating.

129. ADHB also agreed that it would have been better if the IDS had been consulted earlier, and also that all of their reviews should have been documented. ADHB advised that the IDS reviews all patients in PICU every day, and this occurred with Miss A.

Other expert opinions

130. I note the consensus of the expert reviewers, including HDC's expert advisors and the opinions sought through ADHB's review, which echo the above.

131. ADHB's review ultimately concluded that despite Miss A's worsening clinical condition and no identified cause, there were no further investigations undertaken to identify the cause of her pneumonia and, as a consequence, Miss A did not receive anti-viral or anti-Mycoplasma treatment until late in her illness.

132. I note the opinions of two of the reviewers for the DHB's internal review. IDS consultant Dr E noted that no investigations for atypical pathogens or viral pneumonia or influenza were

undertaken, and considered that testing for both atypical pathogens and viral pathogens should have been considered in the context of worsening disease, and progression to surgical intervention and intensive care. Paediatric consultant Dr F similarly noted that at Miss A's readmission, there was an opportunity to consider aetiology (cause of her illness) with more testing such as a nasopharyngeal swab and respiratory PCR panel.

133. In addition, I note the comments from my paediatric and nursing advisors. Dr Doran noted the delay in testing, that specific antiviral therapy was not started, and that treatment covering organisms associated with atypical pneumonia was not commenced empirically until 5 Month2. Mr Gorte commented that with the time of year, the threshold for considering influenza as a possible diagnosis, and consideration of initiating treatment, needs to be reflected upon.

Conclusion

134. The need to establish the cause of Miss A's illness was clearly identified, but testing and further investigations were delayed, resulting in a delay in diagnosing Mycoplasma pneumonia and influenza B. It follows that specific treatment for those conditions was also delayed. In addition, there was delay in starting empiric antibiotics, while a more definitive diagnosis was pending.

135. Based on the expert opinion and ADHB's responses, I conclude that further investigations for viral and atypical pneumonia, and broader empirical treatment, should have occurred when it became clear that Miss A was not responding to treatment and her pneumonia was becoming more severe. Miss A's deterioration was notable on her re-admission to ADHB when she required surgical insertion of a chest drain. Such further investigation/ treatment was indicated around this time (although it is not possible for me to conclude a specific date or time, but at the latest by 1–2 Month2). I also conclude that the IDS should have been asked to review Miss A earlier than 2 Month2, namely around 29 Month1 after her admission to PICU. This may have informed the need for, and scope of, further testing.

136. While I am unable to determine whether an earlier diagnosis and treatment would have altered the course of Miss A's condition, I am critical that Miss A did not receive timely investigations, and was prevented from being afforded appropriate treatment earlier.

137. I am also critical of the inadequate documentation of all IDS reviews that occurred.

138. In my view, these failures were not isolated incidents and did not involve only one or two staff members. During Miss A's admission, there were numerous missed opportunities by the services involved in her care to investigate more intensively and in a more timely way, and the cumulative effect of these missed opportunities demonstrates a concerning lack of critical thinking by ADHB staff, attributable to the DHB as the overall service provider.

139. In my opinion, by failing to investigate the cause of Miss A's illness adequately in a timely manner (with the resulting diagnostic and treatment delays), and by failing to start empiric antibiotics earlier, ADHB failed to provide services to Miss A with reasonable care and skill, and therefore breached Right 4(1) of the Code.

Guidelines in place at the time of events — adverse comment

140. There were also issues with guidelines in place at the time at ADHB, which impacted the care provided to Miss A.

141. I refer to the ADHB Clinical Guideline for Pneumonia summarised below under "Changes made". At the time of events, this did not include specific guidance for the investigations to be undertaken, and when, or guidance on which antibiotics to consider for illness with different clinical features. Associate Professor Tibballs advised that the guideline stated that a nasopharyngeal aspirate may be useful for diagnosing viral pneumonia, and indicated who may benefit from treatment with oseltamivir, but the advice was restricted to children aged under two years, which was erroneous.

142. ADHB advised: "[W]e agree that the ADHB guideline for severe pneumonia was not as clear as it could be in terms of testing for viral and atypical causes in children with severe pneumonia." ADHB stated:

> "[M]ost guidelines would suggest that tests for these causes should occur when pneumonia is severe and/or progressive despite standard antibiotic treatment. Our hospital clinical guidelines were not clear about this and have subsequently been amended. We sincerely apologise for this omission in her care."

143. ADHB's internal review made recommendations to review and update the ADHB Clinical Guideline for Pneumonia (the guideline current at the time of these events), and to include a pathway for investigation and management of children with severe progressive pneumonia, including detail on when and how to test for atypical pneumonia and viral pneumonia. The review also recommended adding circumstances for when empiric combination therapy with an antibiotic from the macrolide class (antibiotics that are used to treat Mycoplasma pneumonia) can be considered, and outlining clear indications for testing for influenza and use of oseltamivir in hospitalised children in both the Influenza Guideline and the Pneumonia Pathway. At the time, the latter was outlined only in the Influenza Guideline.

144. I am concerned that the guidelines in place at the time were not sufficiently clear to guide staff regarding further investigations and treatment in the context of a child with severe pneumonia. Nonetheless, guidelines should not replace clinical judgement and critical thinking. As outlined above, and regardless of the adequacy of the guidelines in place at the time, I am most concerned that further investigations/testing and starting of empiric antibiotics were not considered sooner. It is appropriate that ADHB has reviewed and amended the ADHB Clinical Guideline for Pneumonia.

Care provided by PICU service — adverse comment

145. Miss A was admitted to the PICU on 29 Month1, and remained there until her death.

146. Associate Professor Tibballs advised that in his view, "there were two major deficiencies in medical practice at [ADHB]" — the first relating to diagnosing and managing community-

acquired severe pneumonia by all services, including PICU; and the second being the management of the ECMO.

147. Associate Professor Tibballs considered that investigations and further documentation of Miss A's medical history could have been undertaken at her earlier ED presentation and earlier hospital admission. ADHB has disputed these criticisms.

148. I acknowledge the differing perspectives. In the circumstances, I have relied on the peer expert advisors for the initial ED presentation on 23 Month1, and the paediatric care and paediatric nursing care provided during Miss A's 25–27 Month1 admission. Relying on my other experts, I consider that aside from the exceptions already identified and as discussed earlier in this opinion, the care provided was appropriate. Of most concern to me is the delay in identifying the cause of Miss A's ongoing and serious illness during her 28 Month1 admission, discussed in detail above (under "Delay in identifying cause of Miss A's illness and in starting empiric antibiotics during 28 Month1 admission", in which I have relied on Associate Professor Tibballs' advice as the peer expert advisor).

149. However, Associate Professor Tibballs also identified a number of concerns with the care provided by the PICU service. ADHB has disputed several of these criticisms. I will address each issue in turn.

High Flow Nasal Prong (HFNP) therapy

150. Miss A was started on HFNP (respiratory support) on 29 Month1, and remained on this until her procedure on 4 Month2, when she was started on mechanical ventilation.

151. Associate Professor Tibballs advised that usually HFNP is given only for mild respiratory illness. He indicated that while it may be useful in avoiding invasive mechanical ventilation, in severe pneumonic disease (as Miss A had), it was not appropriate and below a reasonable standard of care.

152. ADHB disagreed. In its view, the use of HFNP was appropriate (as it had advantages over more invasive ventilation). It further contended that escalation to invasive ventilation also occurred at an appropriate time. ADHB stated that the timing of starting invasive ventilation requires clinical judgement balancing the risks and benefits, and is a dynamic decision based on frequent observation and review of real-time physiological parameters, and cannot be judged reliably from reading clinical notes.

153. Associate Professor Tibballs agreed in part with ADHB's response, but was critical that sound clinical judgement for the decision was not evident from the documentation.

154. Having considered ADHB's submission and my expert's advice, on balance I consider that in Miss A's case, the use of HFNP and the timing of invasive mechanical ventilation was not unreasonable. However, relying on Associate Professor Tibballs' advice, it would have been prudent for the clinical rationale to have been documented more clearly.

Consideration of High Frequency Oscillation Ventilation (HFOV) on 4 Month2

155. On 4 Month2, Miss A was started on mechanical ventilation. Associate Professor Tibballs commented that mechanical ventilation was continued in PICU, but emphysematous changes (rupturing of small air sacs) had been observed on the surface of the right lung at that time on 4 Month2, and surgical emphysema (presence of air in the tissues under the skin) appeared within 24 hours of the start of mechanical ventilation. He advised that this was the time to consider whether HFOV (an alternative type of mechanical ventilation), if available, ought to have been given. He considered that this was a less injurious form of mechanical ventilation.

156. ADHB disagreed with this advice, and advised that in its opinion, the most appropriate and least injurious form of ventilation was used. ADHB further commented that there is no evidence that HFOV is less injurious to the lungs than the type of mechanical ventilation that was used for Miss A. ADHB referred to various studies, and noted that in recent years HFOV has been used much less commonly.

157. In response, Associate Professor Tibballs did not wholly accept ADHB's comments, but acceded that there was debate about which technique was less injurious, and advanced that, at the least, it was worthwhile "trialling" HFOV.

158. What is evident to me from the exchange of views is that the choice of invasive ventilation appears to be an area where differing specialist opinion exists, and which, ultimately, is a matter of clinical judgement. In the circumstances, I accept that ADHB's choice of ventilation was reasonable.

Hypertension while on VV-ECMO (life-support machine)

159. Miss A was started on VV-ECMO on 7 Month2.[61]

160. Miss A had high blood pressure (hypertension) from around 4pm on 7 Month2, until 5pm on 8 Month2. Associate Professor Tibballs was particularly concerned that PICU staff permitted severe prolonged hypertension (high blood pressure), while there was a state, albeit relatively brief, of excessive anticoagulation (excessive blood thinning). Associate Professor Tibballs was also critical that PICU staff did not act in sufficient time or with appropriate medication to control the hypertension. He described the hypertension as "severe", and noted that it continued for more than 24 hours.

161. In response, ADHB stated that hypertension on ECMO is common, and that the hypertension Miss A experienced when first on ECMO was not severe. ADHB also claimed that the hypertension was treated appropriately.

162. Associate Professor Tibballs reviewed ADHB's response and stated that hypertension being common does not make it acceptable or untreatable, and maintained that in his opinion, the treatment of Miss A's hypertension was inadequate and delayed. He advised that milrinone is not the preferred agent to treat severe hypertension, while nitroprusside (the

[61] It should be noted that Associate Professor Tibballs' advice was initially based on the belief that Miss A was on a different kind of ECMO — namely VA-ECMO. ADHB has satisfied me that Miss A was on VV-ECMO.

appropriate treatment) was commenced belatedly after 18 hours of severe hypertension. He considered that the requirement would have been to restore the blood pressure to at least normal values as quickly as possible, and that ADHB's statement that the hypertension was not severe is erroneous.

163. Associate Professor Tibballs agreed that the necessary anticoagulation for ECMO, even when strictly controlled, is a high risk factor for cerebral haemorrhage. In his view, to minimise the additional risk that hypertension posed, strict upper limits for blood pressure should have been present and adhered to.

164. In Associate Professor Tibballs' opinion, inadequate attention was paid to combatting this important risk factor.

165. In response to the provisional opinion, ADHB stated that while there was one recording of significant hypertension and agreed that this needed to be treated, and that the charting of goals for blood pressure was inadequate, it maintained that this was not a hypertensive emergency as suggested by Professor Tibballs. ADHB referred to Miss A's hypertension as "moderate". It further commented that there is no literature to support that a moderate degree of hypertension is a cause of cerebral haemorrhage.

166. Relying on Associate Professor Tibballs' advice, I accept that an upper limit of blood pressure should have been identified while Miss A was on ECMO. There is, however, a difference in clinical opinion regarding how severe Miss A's hypertension was, which I am unable to resolve. Therefore, I do not propose to make any further comment on this matter.

Management of VV-ECMO — anticoagulation

167. Associate Professor Tibballs advised that anticoagulation was briefly excessive, putting Miss A at risk of bleeding, including at an increased risk that she might bleed into her brain.

168. ADHB asserted that the anticoagulation was appropriately managed in accordance with its applicable protocol. It stated that the clotting time often fluctuates while on ECMO, and, in response, the blood-thinning medication (heparin infusion) is adjusted accordingly. ADHB said that there was no deviation from its protocol in managing Miss A's heparin dosing, and indeed Miss A was at all times receiving a comparatively low dose for a child on ECMO. The result was too high at 3am, so heparin was reduced, and by 7am the ACT was coming back to the desired range. After that, all clotting times were at the lower end, or below the desired range, and remained so all the morning of 8 Month2.

169. Associate Professor Tibballs acknowledged that controlling anticoagulation is a difficult task. In the context of the anticoagulation being excessive for a brief period, he questioned the precision of the protocol. However, he also advised that the anticoagulation being excessive is not the main issue, and did not necessarily imply a standard of care below normal.

170. In its response to the provisional opinion, ADHB challenged Associate Professor Tibballs' comments on its protocol. It confirmed that its protocols are reviewed regularly, and that inevitably there are times when measurements will fall outside the goal range. The quality of a protocol is in how much this is minimised, not that it is prevented entirely. ADHB considered that in Miss A's case there was a single measurement, which was responded to appropriately.

171. Having considered the two views, I accept and acknowledge that this is a complex clinical issue. I note, however, that Associate Professor Tibballs does not imply a departure from the standard of care in this respect. I accept ADHB's submission that its protocol is reviewed regularly and it compared to others around the world. I am not critical of the adequacy or precision of the protocol.

28 Month1–11 Month2 — other comment

172. As outlined above, through the course of the investigation, clinical advice was sought, and a number of other concerns were identified (outlined in full in the appended reports). These included minor criticisms of the paediatric surgical care provided;[62] that discussion of organ donation did not occur; the standard of documentation of death; and causation of the brain bleed. There were also issues with the general standard of documentation during this admission (other than what has been specifically identified in this report). ADHB agreed that documentation could have been better in Miss A's case.

173. Death processes are outside the scope of this investigation.

174. My consideration of the complaint is not to assess whether the actions of any of the healthcare providers who cared for Miss A caused her death. Rather, my role is to assess whether, with the information available to Miss A's healthcare providers at the time of events, they acted appropriately and in accordance with accepted standards of practice. I have already identified those areas of care that fell below the standard of care.

175. I note further that there is conflicting opinion between Associate Professor Tibballs and ADHB regarding the underlying cause of Miss A's brain bleed. However, it is agreed that cerebral haemorrhage is a known risk with ECMO, and there is no suggestion that ECMO should not have been used in the clinical situation. As it is not my role to determine the underlying cause of Miss A's brain bleed, I do not propose to resolve that conflict in this opinion.

176. The other concerns raised by clinical advisors are not central to the issues in the assessment of Miss A's case, and therefore they have not been detailed or addressed fully in this report. However, these have been brought to the DHB's attention for its consideration.

[62] My paediatric surgical advisor, Professor Mark Stringer, identified two minor criticisms — that the course of urokinase therapy was not completed as prescribed, and that a chest CT scan was not done before undertaking the VATS procedure. Professor Stringer noted that he does not consider that either would have impacted on the progress of Miss A's disease. Professor Stringer advised that the surgical care was appropriate and there were no other surgical interventions that could have saved Miss A.

177. I also acknowledge that other aspects of the care provided to Miss A, namely the nursing care and paediatric surgical care, were largely appropriate.

Changes made

Pneumonia Guideline

178. The ADHB Clinical Guideline for Pneumonia (2010) that was in place at the time included:

> "**Investigations**
> …
>
> Sputum, throat swabs and NPA [naso-pharyngeal aspirate] for bacterial cultures do not help determine who should receive antibiotics. An NPA may be indicated for co-horting patients being admitted, for diagnosis of suspected viral pneumonia (< 2 years) and deciding who may benefit from antiviral medication such as oseltamivir.
>
> A blood culture is an insensitive test for bacterial pneumonia in children however blood cultures should be considered in the unwell child with pneumonia, especially the child suspected of having Staphylococcus aureus or complicated pneumonia. Fever magnitude, full blood count findings or CRP do not reliably differentiate viral from bacterial pneumonia …
>
> **Treatment**
> …
>
> **Oral antibiotics**
> Oral antibiotics will provide adequate coverage for most mild to moderate episodes of pneumonia. This may include some of those requiring admission …

Age	Antibiotic	Dose	Duration
…	…	…	…
≥ 5 years, Mycoplasma pneumonia suspected	Erythromycin	12.5mg/kg/dose QID	7–10 days
	OR, Roxithromycin (tablets only)	4mg/kg BD	

> …"

179. ADHB advised that following a review of the treatment provided to Miss A, the ADHB Clinical Guidelines were updated in 2018, including specific advice on when to consider

macrolide and/or anti-viral treatment. The updated version outlined the indications of severe pneumonia, further detail on investigations to consider and undertake, circumstances for when empiric combination therapy with a macrolide can be considered, and indications for testing for influenza and use of oseltamivir.

PEWS chart

180. ADHB further advised that in August 2018, the Patient Deterioration Clinical Governance Committee approved amendments to the "Recommended Actions" on the PEWS chart. The changes were based on feedback from ward clinical teams and the specialist nurse team that children may have an increase in PEWS to 6–7 that is readily resolved by attention to pain relief or fever management, with no ongoing clinical concerns regarding deterioration. They included statements to manage pain/fever and distress, as well as changes in review times for certain PEWS scores.

Recommendations

181. I recommend that ADHB:

a) Provide a written letter of apology to Mr and Mrs A for the aspects of care I have identified as deficient. The apology is to be sent to HDC within three weeks of the date of this report, for forwarding to Mr and Mrs A.

b) Provide HDC with an update on the implementation and effectiveness of the updated Pneumonia Guideline and PEWS chart, including whether any further changes have been made, within two months of the date of this report.

c) Perform an audit of its compliance with the updated Pneumonia Guideline and PEWS chart for a random three-month period within 2021, and provide HDC with the results of the audit within six months of the date of this report.

d) Use the findings of this complaint as a basis for training paediatric services staff in management of pneumonia at ADHB, in a way that maintains the anonymity of all parties involved, and provide evidence of that training within three months of the date of this report.

e) Communicate the changes made to the Pneumonia Guideline to other DHBs, within one month of the date of this report.

f) Consider the possibility of further systems improvement to prevent human error in relation to review prior to transfer, such as a nursing checkpoint for escalating the changing condition of a patient prior to transfer, and updating the nursing transfer letter template. ADHB is to provide HDC with the outcome of its consideration within two months of the date of this report.

g) Further consider the recommendations made by my paediatric intensivist advisor, and report back to HDC on the outcome of its consideration, within one month of the date of this report.

182. I also request that ADHB remind its staff about the importance of full and accurate documentation of clinical care.

Follow-up actions

183. A copy of this report with details identifying the parties removed, except Auckland District Health Board and the experts who advised on this case, will be sent to the Ministry of Health and the Health Quality & Safety Commission, and placed on the Health and Disability Commissioner website, www.hdc.org.nz, for educational purposes.

Appendix A: Independent advice to the Commissioner

The following expert advice was obtained from Dr John Bonning, an emergency medicine physician:

"**Case reference C18HDC00063 18th May 2019**

My name is Dr John Bonning. I am an emergency physician, working in the ED of a tertiary referral (Waikato) hospital.

I have been practising as an Emergency Medicine specialist for 15 years as a Fellow of the Australasian College for Emergency Medicine. I have been a doctor for 28 years. I am the President-Elect of the Australasian College for Emergency Medicine and a Director on the Board of ACEM.

I have been asked by the Health and Disability Commissioner to provide an opinion on case number C18HDC00063 regarding the care provided by [ADHB] to [Miss A] on 23 [Month1]. All further care [Miss A] was provided subsequently is being spoken to by other expert advisors.

I have read the HDC's guidelines for independent advisors and declare that I have no conflict of interest (regarding knowledge of the affected parties) in this case.

I have read the following documents

1. Letter of complaint …
2. ADHB's treatment review …
3. Copy of meeting minutes …
4. [ADHB's] clinical guidelines for Pneumonia at the time of treatment
5. [ADHB's] updated clinical guideline for Pneumonia
6. ADHB's response …
7. Clinical records from ADHB for 23 [Month1]

I would like to express my sincere sympathy to [Miss A's] parents [Mr and Mrs A] and her family for their loss.

On 23 [Month1], [Miss A] (aged 6 years) was taken to the Emergency Department (ED) of [ADHB], arriving at around 0815hrs. She was seen on arrival by the ED triage nurse and a history of three days of fever and cough was elicited. A set of vital signs was taken on arrival, indicating a low-grade fever of 37.9, a heart rate of 115, a respiratory rate of 28, and lack of respiratory distress was documented. Oxygen saturations were 98%, and normal capillary refill was noted.

[Miss A] was soon after seen by emergency doctor [Dr C] who confirmed the history obtained by the triage nurse. She noted that [Miss A] had been active until the day before presentation, a reduced oral intake, and fever despite Paracetamol and Ibuprofen. A few other pertinent (symptom) negatives were noted. She documented

the fact that [Miss A's] [sibling] had been unwell over the last 3 weeks with pneumonia and had just recovered. She noted that [Miss A's] parent(s) were anxious about a diagnosis of pneumonia and wished blood tests and a chest x-ray to be performed. [Miss A] was up to date with her immunisations.

Examination findings were documented including the vital signs, all within normal range except for the fever. A temperature of between 37.5 and 38 is commonly termed a 'low-grade' fever. [Miss A's] general demeanour and appearance of not being distressed by her illness and moving around the ED normally was documented. Chest examination revealed normal respiratory rate, normal work of breathing, some quieter breath sounds on the right side (compared to left) indicating some lung abnormality underlying, but no added breath sounds such as crepitations or wheeze.

A provisional diagnosis of a lower respiratory tract infection was made based on reduced breath sounds on the right and the fact that [Miss A's] [sibling] had recently had (x-ray-confirmed) pneumonia. The terms 'lower respiratory tract infection' and 'pneumonia' are often used interchangeably, but the differentiation of these terms is not a key issue in this case. A prescription for Amoxycillin, Paracetamol and Ibuprofen was given and (presumably verbal) advice as to when to seek medical advice subsequent to her discharge.

Dr … was noted as the Consultant on duty at that time with overall responsibility for the care provided to all patients in ED but it is not clear if [Dr C] spoke to Dr … or not. I would not expect an emergency registrar to (feel the need to) run this case past a senior doctor prior to discharge in this clinical setting.

I have been asked by the Commissioner to assess whether the care provided to [Miss A] by [ADHB] on 23rd [Month1] was reasonable in the circumstances and why. In particular I will address some concerns raised including by [Miss A's] parents, with my responses relating purely to this presentation:

- Was the clinical assessment appropriate
- Should investigations such as blood tests or a chest x-ray have been performed
- Should investigation to identify the causative organism have been undertaken
- Did [Miss A's] assessment and management conform to the hospital guidelines at that time
- The appropriateness of [Miss A's] discharge, whether admission was discussed and the adequacy of discharge information

Apart from a low-grade fever of 37.9 and the reduced breath sounds on the right side of her chest [Miss A's] examination was near normal. Her vital signs were within the normal range for her age and according to documentation by the triage nurse and [Dr C] [Miss A] did not look particularly unwell. The examination findings were that of a stable child. I believe that the performance and documentation of the clinical assessment was appropriate and clinically sound.

It is not routine to undertake further investigations in this clinical scenario (despite the parent's wishes for [Miss A] to have investigations) and I support [Dr C's] decision not to

perform blood tests or a chest x-ray. Neither were indicated according to the pneumonia guideline and in my opinion neither are likely to have made any change to the clinical plan of discharge with antibiotics. In addition the medical literature suggests that in the majority of cases intravenous antibiotics offer little advantage over oral antibiotics for all but the most severe of pneumonias.

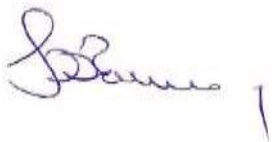
Tests to find causative organisms are rarely done in the majority of patients with (non-severe) community-acquired pneumonia, and when done they frequently do not find such organisms (in particular blood cultures and sputum samples) and rarely change management decisions. The medical literature on this matter states that blood tests including cultures do not improve clinical outcomes. Quite correctly the [ADHB] guidelines state that 'many children with pneumonia may be diagnosed and managed on clinical grounds alone. Radiology does not reliably distinguish bacterial from viral pneumonia so does not determine the need for antibiotics.'

'Return advice' is written in the discharge letter but the nature of this advice is not fully documented. Given the thoroughness of her documented assessment I would suggest that it is more likely than not that [Dr C] gave adequate instructions verbally. Even the very basic 'if ([Miss A]) gets sicker or does not get better, seek medical advice from either her GP or returning to ED' would probably be deemed sufficient. Since it is stated that [Miss A's] parent(s) requested investigations on this visit it is also likely that [Dr C] explained why she was not performing such tests and why she felt [Miss A] could be safely discharged.

Despite the tragedy of this case I do not believe accepted guidelines for assessment and management of community-acquired pneumonia need to be changed. Specifically, I strongly believe that routine testing for causative organisms (by GPs or emergency doctors) is not at all supported by the medical literature and should not be performed.

It is my opinion that [Miss A's] assessment and management (including being discharged on oral antibiotics) by medical and nursing staff at [ADHB] on 23 [Month1] was entirely appropriate given the well documented clinical findings at the time of this presentation and met the standard of care expected. Her care conforms to the [ADHB] guidelines both at the time of presentation and the revised guideline. The only issue that could be faulted was lack of written documentation of specific discharge advice, although the practice of stating that advice was given but not the specifics is very common and I do not view this as a departure from the standard of care.

I trust this report is of use to the Health and Disability Commissioner.



Dr John Bonning, BHB, MBChB, FACEM
Emergency Physician"

Appendix B: Independent advice to the Commissioner

The following expert advice was obtained from Dr John Doran, paediatrician:

"Review of complaint: [Miss A]/[ADHB] (Auckland District Health Board)

Ref: C18HDC00063

My name is John Doran and I am a General Paediatrician currently working in Taranaki Base Hospital where I have been as a paediatric consultant since 1991.

I qualified MBChB (Otago) in 1978 and gained my general paediatric fellowship in 1990 (FRACP).

I have reviewed the HDC's Guidelines for Independent Advisors.

This review is based on the documents provided by the office of the HDC which include:

1. Letter of Complaint and associated documents
2. Response from Auckland DHB … and Treatment review …
3. Clinical records from [DHB2] and Auckland DHB
4. [ADHB] Guidelines for pneumonia at time of treatment and updated post review
5. Copy of meeting minutes …

[Miss A] a 6 year old girl was taken to [the ED] at [ADHB] by her parents on 23 [Month1] having had three days of respiratory symptoms.

Clinical assessment at that time was consistent with a diagnosis of Lower respiratory tract infection and as she was assessed as being systemically well she was appropriately discharged home with oral antibiotics. The discharge summary, which I assume her parents were given on leaving [the] ED, included information and advice regarding signs of deterioration to be aware of and what to do if any ongoing concerns.

I have been asked to respond to a series of specific questions related to specific periods of her medical admission and the rest of the report is tailored to that end.

25 [Month1]–27 [Month1]

[Miss A] represented to … ED on the evening of 25 [Month1] at 2000hrs because of ongoing fever, irritability and cough with reduced oral fluid intake. Clinical assessment in conjunction with investigations were consistent with the primary admitting diagnosis of right sided Pneumonia.

Her treatment with intravenous (IV) fluids and IV antibiotics (Cefuroxime) was appropriate. The change of antibiotic from oral Amoxicillin to intravenous Cefuroxime was what would be expected as her symptoms had not responded to the high dose oral Amoxicillin and Cefuroxime offers a slightly broader range of antibacterial cover. It would not be usual at this point to routinely investigate for possible aetiological infecting organisms and the antibacterial treatment is aimed at the common pathogens.

Investigations such as nasopharyngeal swabs/aspirates for viral or atypical pneumonia are usually done when severe or persistent illness is not responding to standard therapy. In [Miss A's] situation at this time the clinical picture was still evolving.

The clinical team caring for her acted as would be expected in response to her symptoms and signs at this time and I would not expect advice from other paediatric subspecialist teams to be sought at this point.

27 [Month1]

From my review of the clinical notes provided the decision to transfer to [DHB2] was related to that being the DHB catchment area within which she and her family resided. It was recognised that she required ongoing hospital level care to manage her pneumonia and therefore required to be an inpatient.

In the written clinical notes documenting the morning ward round (0830, 27 [Month1]) it is reported that [Miss A] 'feels significantly better today'.

The presence and level of pleural effusion is not well documented in the clinical notes so I am unable to comment on that particular aspect of clinical assessment.

Her Paediatric Early Warning Score (PEWS) an assessment at that time (0855) was recorded as 5 having improved from 9 at 0230 that morning and 7 at 2100, 26 [Month1]. This supports that there was some clinical improvement with interventions which included oxygen and IV fluids.

[Miss A's] parents were obviously concerned about the planned transfer and wished her to remain in [ADHB] at **this time**.

Prior to her transfer which occurred at approximately 2pm there were nursing assessments which showed a PEWS = 7 so not substantially changed. There is no documented doctor assessment between the morning ward round and transfer. I do not think this was needed as condition relatively stable from nursing observations and assessment. However again her parents were worried that she wasn't as well and that she had deteriorated over the day.

The written information provided on transfer seems to be reasonable including copies of clinical notes and observation charts. I am unable to comment on the verbal handover which is the most critical part of transfer of patient care.

The decision to back transfer [Miss A] to [ADHB] was also appropriate as her deteriorating clinical condition was recognised, rapidly investigated (ultrasound chest), and further treatment initiated. I do not believe that the transfer to [DHB2] caused delay in more definitive treatment as the extent of the pleural effusion was rapidly diagnosed in [DHB2].

[Miss A] returned to [ADHB] on 28 [Month1] and had rapid surgical intervention (chest drain) for her effusion.

28 [Month1]–11 [Month2]

I am not a Paediatric Surgeon or Paediatric Intensive Care Specialist therefore I am unable to provide expert comment on these aspects of care but from my experience the interventions seem consistent with those of other children with pleural effusions and deteriorating respiratory function. These are clinical decisions that are very individual patient specific and responsive to the whole clinical picture as it unfolds at the time.

A range of paediatric subspecialists were very appropriately involved in [Miss A's] care trying to optimise all aspects of her care and senior medical staff were very much part of her management both in the decision making about various aspects and direct care.

It appears that the initial pleural aspirate (28 [Month1]) was sent for a number of investigations including mycoplasma pneumonia PCR but this was not reported on until 9 [Month2].

I note that treatment covering organisms associated with Atypical Pneumonia were not commenced empirically until 4 [Month2] and specific antiviral (Influenza virus) therapy was not started.

As discussed in the microbiology response these could have been considered earlier as intensity of intervention required progressed because of lack of expected response to standard antibacterial treatment. It is not possible to say whether these interventions would have altered the clinical course.

I note that one of the key recommendations is to consider atypical pneumonia or viral pneumonia when children are not responding to treatment of pneumonia as expected or if they are becoming more seriously unwell.

The recommendation goes further to say that treatment should be initiated empirically in clinical situations like [Miss A's] to ensure all possible infective causes are covered.

General

The recommendations suggested address the issues raised by the investigations into this complaint and the updated (February 2018) [ADHB] Clinical Guidelines reflect this.

This was a tragic outcome that would be very uncommon for paediatric Mycoplasma pneumonia which is often a self-limiting condition especially in a previously well child. Effusions do occur in mycoplasma pneumonia but only rarely require any intervention and clearly this case highlights that when rare events occur it can be difficult to identify the cause and therefore the recommendations advise acting without being absolutely certain about what the cause is, in this situation what the infecting cause is.

Dr John Doran FRACP
PAEDIATRICIAN"

The following further advice was obtained from Dr Doran:

"18 November 2019

…

Ref: 18HDC00063

Thank you for the opportunity to respond to the response from Auckland DHB dated 2 September 2019.

I am responding only to the comments relevant to my initial report. In particular these comments focussed on the documentation in the clinical notes about the presence and level of pleural effusion.

I acknowledge that this sad case has been reviewed by a number of experts now and that [Miss A] presented with pneumonia which was managed according to accepted practice and guidelines.

I agree that the pleural effusion was noted on the report of the initial chest x-ray and was signalled in the clinical notes.

Chest diagrams are part of the clinical notes reflecting examination findings on 26 [Month1] at several points (1025hrs, 1445hrs, 1630hrs) and again on 27 [Month1] at 0830. I had interpreted these as indicating the area of reduced air entry but accept that the diagrams also document the presence and level of the pleural effusion though that is not specifically stated.

These diagrams indicate a stable picture in terms of clinical examination findings.

I hope that answers the specific questions raised by the response.

Yours sincerely

Dr John Doran FRACP
PAEDIATRICIAN"

Appendix C: Independent advice to the Commissioner

The following expert advice was obtained from Mr Thomas Gorte, paediatric nurse:

"Thank you for the request for me to provide expert opinion and clinical advice in relation to the care provided to the child and family at [ADHB]. The periods of care being reviewed are from 25 [Month1] to 27 [Month1] and then from 28 [Month1] until 11 [Month2]. I have no personal or professional conflict of interest. I have read and agreed to follow the Commissioner's Guidelines for Independent Advisors. The majority of my advice is regarding the nursing care provided to the child and family. I have also inserted some information supported by evidenced-based literature regarding current knowledge surrounding chest infections in children.

I qualified as a Registered Sick Children's Nurse from Great Ormond Street Hospital in London in 1993. I have practised within various paediatric clinical settings in London, New Zealand and Barbados. I have also been employed in tertiary education since 2000 and was a senior Professional Clinician and acting Director of Undergraduate Nursing programme at Massey University for several years. I am currently employed within the hospital setting as a Nurse Educator within the Children Service. I have a Masters in Nursing and am also a qualified Health Service Auditor.

I have reviewed the following documentation: clinical records from ADHB from 23 [Month1] onwards; the letter of complaint and associated documents; ADHB treatment review; copy of the minutes …; [ADHB] clinical guidelines for pneumonia at the time of treatment; ADHB response …; and the clinical records from [DHB2] from 27 [Month1] to 28 [Month1]. I have only provided comment on the care provided by ADHB as that was what had been requested by the office of the Commissioner.

I have referred to [ADHB] policies as well as other evidenced-based information to support my perspectives. A short reference list is provided at the end.

25 [Month1]–27 [Month1]

Inconsistencies in assessment of work of breathing (WOB) identified.

At 0430hrs on the 26 [Month1], the nurse admitting the patient states that the WOB is moderate without including objective signs. The Respiratory Distress observation space has no recording which would have impacted upon the PEWS score and increased frequency of observations. From 0800hrs until 1400hrs, the chart recordings are that the patient has 'nil' respiratory distress, even though oxygen therapy is commenced at about 1000hrs with increased respiratory rate, heart rate and pyrexia of 39.0°C to 39.6°C. Based on these observations and the intervention, it is unlikely that there were no signs of accessory respiratory muscle use as physiological processes attempt to rectify the blood hypoxia.

At 0530hrs on 27 [Month1], the nurse records that the child was having abdominal indrawing and tracheal tug. At the age of six years, abdominal in-drawing is a moderate

sign of WOB. It is questionable whether the WOB assessments are accurate thereby leading to a lower PEWS and possible reduced time to escalate.

The issue with respiratory effort assessment is that there is a great deal of subjectivity. Both local and international guidelines do not have specific signs of respiratory effort that clearly differentiate the transition from mild to moderate to severe as the child's age increases. However, the 'nil' respiratory effort recording of a child with pneumonia, pyrexia and oxygen dependent is doubtful.

A Registered Nurse should have the skill to differentiate between normal and increased respiratory effort and accurately report on this. *My advice is that this is a moderate departure from the appropriate standard of nursing respiratory assessment.*

Utilisation of Paediatric Early Warning Score (PEWS)

About 20% of children who die in hospital have avoidable risk factors leading to death (Lambert et al., 2017). The introduction of PEWS is aimed at reducing this incidence by the initiation of early, timely intervention. Examination of the observation chart during the aforementioned timeframe shows several gaps with no PEWS recorded. Whilst an accurate PEWS might be impacted upon when a blood pressure has not been recorded, the lack of a PEWS score (of 4) at 1000hrs meant that the required escalation to the nurse in charge might not have been actioned. However, a review by the registrar at 1030hrs occurred which might have been requested by the nurse. At 1400hrs, the PEWS score is noted to be 9 whereby the RN escalates to the specialist nurse and house officer at 1400hrs which is appropriate and timely.

Following on from the specialist nurse team review, there is no evidence of increased recording of observations initially and senior doctor review within 30 minutes is not evident within the clinical record. The senior doctor review occurs two hours later. At 0230hrs on 27 [Month1], the specialist nurse appropriately escalates to senior medical staff as PEWS are 8+. The 5–11 years Clinical Observation Chart has recommended actions based on PEWS which were not consistently adhered to.

Between 2200hrs on 26 [Month1] to 0700hrs on 27 [Month1], the patient has had three specialist nurse reviews, hypotension with systolic readings of 81–93, tachypnoeic at 42–64 with increased WOB and slight pyrexia of 37.9°C. Whilst the heart rate has reduced from the earlier 130–145, the clinical picture is not one of overall improvement but one of static progress. From 0700hrs until the time of transfer at about 1300hrs, there is increased respiratory rate from 46 to 61 breaths per minute, increasing pulse rate from 105 to 144 beats/min, a reduced peripheral oxygen saturation (SpO_2) to 94% whilst still on 0.5L via nasal prongs but all with a normotensive blood pressure. All of these occurred synonymously with the pyrexial spike up to 39.7°C. A reduction in temperature to 37.6°C has synonymous reductions in heart rate to 112 but the respiratory rate is still 52, the SpO_2 is still reduced at 94% on 0.5L and the blood pressure is borderline hypotensive at 91 systolic. The PEWS is still high at about 8, so a senior doctor review needed to occur of which there is no evidence of this intervention within the clinical notes.

At 1100hrs, a specialist nurse team review is requested as the nurse records a PEWS of 9/10 with a respiratory rate of 61. The specialist nurse assessment obtains a PEWS score of 7 being recorded based on the following observations and associated PEWS score: RR — 50 (4), WOB — mild (1), oxygen requirement of 2L via nasal prongs (2) and BP 90/39 (1). This PEWS appears to be 8 instead of 7 which would require a senior doctor review. However, even with the PEWS score of 7, the increase in RR and oxygen requirement is concerning. At 1350hrs, an RN documents that the child has significant work with breathlessness whilst trying to drink and is lethargic. These signs are evidence of at least moderate respiratory effort and possible signs of deterioration.

Under competency 2.8 within the RN Scope of Practice, the level of effective nursing care required by the individual client was not consistent. *My advice is that the lack of consistent evaluation and timely escalation based on the PEWS score and the lack of improvement of the child is a moderate departure from the expected care delivered by a registered nurse.*

Appropriate Specialist Input

The RNs on the ward mostly maintained hourly observations and appropriately escalated to the specialist nurse team. Some of the suggestions from the specialist nurse team are sound such as changing of position, encouraging of airway exercises and the administration of regular analgesia for both comfort and to facilitate possible improved respiratory effort. What is missing is the further escalation to the senior medical doctors such as the registrar or consultant when PEWS scores are 8 or higher. In addition, the specialist nurse team should have encouraged increasing the frequency of observations as stipulated within the PEWS guidelines.

Specialist nurse team members are regarded as experts from which registered nurses look for support and guidance. There is a lack of consistent appropriate advice and lack of timely escalation which I advise to be a mild departure from the expected standard of care. The PEWS chart also contains guidance which the RNs on the floor can refer to.

Appropriateness of transfer

The patient is first seen on the 23 [Month1], and is diagnosed with a lower respiratory tract infection. She is commenced on oral amoxycillin and analgesics and is discharged home with sound advice. The child and family return to [ADHB] on 26 [Month1] and are admitted onto the ward at 0430hrs. The child is commenced on IV cefuroxime of which she receives five doses prior to transfer to [DHB2].

From admission at 0430hrs on 26 [Month1] through to 0700hrs on the 27 [Month1], the patient condition does not improve but instead remains static. This type of progress is concerning as children respond relatively rapidly to clinically effective therapies. Then, from about 0700hrs to 1300hrs, there are clinical signs of deterioration with further pyrexial spikes. This lack of improvement and deterioration occur despite the patient having received about four (4) days of antibiotics of which more than 24 hours of intravenous cefuroxime. Whilst the initial presentation could be regarded as a seemingly typical pneumonia, the lack of progress following four/five days of antibiotics

suggests that this issue is an atypical chest infection. The medical diagnosis on the 27 [Month1] is a right middle lobe (RML) with a possible effusion. Based on the Pneumonia and Pleural Effusion and Empyema Clinical Practice Guidelines from the Royal Children's Hospital Melbourne, red flags include 'breathlessness' and 'persistent fever in the setting of pneumonia despite 48 hours of antibiotics'. In addition, these guidelines suggest that the presence of pneumonia with an effusion should be cared for within a tertiary health facility (RCH, 2016).

Based on this clinical picture, it was not appropriate to transfer this patient to a facility with equivalent or less high dependency capability. Further still the transportation of the deteriorating patient carries significant risk. The transfer should have been cancelled with the team needing to increase the frequency of monitoring and clinically reviewing and reconsidering further investigations. *I advise that this is a severe departure from acceptable practice.*

Under Competency 4.1, there is inconsistent collaboration as there is a lack of formal referral from the specialist nurse and the senior doctor.

Tests, Scans and Assessments

As discussed earlier, increased frequency of observation and assessments had been indicated based on the PEWS scores. Whilst mostly the province of medicine, consideration of chest x-rays was indicated. Whilst the utilisation of chest x-rays does not alter the clinical management of the child with a typical pneumonia, the lack of response to antibiotics and the suggestion that [there is] a pleural effusion are criteria for strongly considering AP/PA erect chest film. In addition, an ultrasound of the chest might have determined the size of the effusion and possibly identified other complications. The other consideration with an atypical respiratory tract infection is to consider the context in terms of time of year. The occurrence of influenza-like illness in New Zealand over the last decade has been mostly [over the winter months]. The threshold for considering influenza as a possible diagnosis and consideration of initiating treatment needs to be reflected upon.

On 9 [Month2], the PCR from the pleural aspirate showed mycoplasma for which macrolide (clindamycin) therapy was commenced. There was no significant improvement after 48 hours. There are reports of significant complications associated with M. pneumonia and non-response of mycoplasma to macrolids where doxycycline is commenced (Patra & Thirunavukkarasu, 2013; Zhou et al., 2014). The literature suggests that there might be increasing incidence of mycoplasma pneumonia for which the clinical picture is more challenging to identify this causative organism.

Information provided to [DHB2]

The Nursing Transfer Letter provides some useful information that suggests that this is a mild to moderately unwell child with pneumonia who has low flow oxygen dependency which is not totally inaccurate. What is missing are the multiple nurse specialist team reviews that have been undertaken and the fact that the health of the child has deteriorated since first presentation on 23 [Month1]. *The clinical picture*

depicted within the transfer letter does not accurately reflect the condition of the client and I consider to be a moderate departure from the expected standard of care.

28 [Month1]–11 [Month2]

The nursing care provided within the surgical unit at [ADHB] is appropriate. Based on the elevated PEWS of 8–12 between 2030hrs on 28 [Month1] to 0500hrs on 29 [Month1], timely escalation to the specialist nurse team and the on-call house surgeon occurred with further escalation to both the medical and surgical registrars. Increased frequency of observations and interventions noted with the insertion of accurate fluid balance charts and regular administration of analgesics.

Similarly, based on the documentation, the nursing care within the PICU is clear, holistic and systematic. Assessments include physiological, psychological and family centred considerations all of which are timely and thorough. Documentation is thorough providing excellent depth of detail. The dislodging of the chest tube at about 0900hrs on 31 [Month1] does not lead to overt changes or deterioration based on the observations recorded during the following 12 hours. *The care by the RNs within the ICU is timely and of appropriate quality as it is responsive to the identified changes in acuity of the child. I feel that the nursing care is thorough even though the outcome was saddening.*

Recommendations

The specialist nurse team need to more consistently escalate to senior medical staff for review within 30 minutes if PEWS is 8 or higher, even if the interventions implemented appear to show improvement.

The specialist nurse team also need to stipulate that the RN caring for the patient with a PEWS of 8 or more need to increase the frequency of observations over a specified time frame.

Greater focus by the health team on observation trends as opposed to one or two sets of observations will provide the health practitioners with a deeper perspective of the success or inadequate response of treatment regimes.

A review of the knowledge and skills of the registered nurses regarding objective signs of work of breathing and subjectively ascertaining whether mild, moderate or severe work of breathing is needed.

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Date: 19 June 2019

Thomas Gorte RN BSc MN

Addendum to Case C18HDC0063

Independent Expert Paediatric Nurse Advice to the Commissioner

Inconsistencies in assessment of work of breathing (WOB) identified.

The clinical documentation by the 5th year medical student at 1025hrs and the PEWS chart states that the child does not have any work of breathing from 0800hrs to about 1300hrs on 26 [Month1]. The nursing notes (0430–0700) within the Assessment component of the SBARR tool state that on admission from … ED, the patient is alert and has moderate work of breathing which was not recorded on the PEWS observation chart. Whilst one might be able to attribute the increased WOB to increased temperature, the pyrexia is continuous even following the administration of paracetamol at 0440hrs and 0900hrs when the work of breathing was recorded as nil. Thus, there are inconsistencies with the recordings. However, whilst it is typical that the patient with pneumonia, pyrexia, significant tachypnoea and oxygen dependency would have accessory muscle use, it is appreciated that if two or more health professionals have recorded no increased work of breathing, then this observation should be respected.

With regard to the PEWS Respiratory Distress guide on the reverse side of the document, this tool is understandably limited in detail. Objective signs of increased respiratory effort such as intercostal, subcostal and abdominal recession are not

referred to but instead amalgamated under subjective terms 'mild', 'moderate' or 'severe' recession/indrawing. I alluded to the overall difficulty in differentiating the transition from mild to moderate to severe as the child age increases.

Based on the correlation amongst different health professionals within the documentation regarding the work of breathing, *I will revise my advice regarding the nursing respiratory assessment to be within acceptable nursing standards.*

Utilisation of Paediatric Early Warning Score (PEWS)

The PEWS scores within the documentation are not consistently correct. From admission, there are errors and a lack of PEWS scores when blood pressures are not measured. Whilst mandatory reporting is not a requirement and clinical judgement is to be respected, the history surrounding the introduction of the PEWS was in response to missed subtle changes and missed early intervention opportunities. Whilst an escalation secondary to the elevated PEWS might not have led to a change in circumstances, it is paramount that collaborative clinical review occurs. The case in hand shows a clinical picture of no improvement of a child with pneumonia even after having had greater than 48 hours of antibiotic therapy: continued pyrexial spikes, significant tachypnoea, increasing work of breathing and borderline hypotension. The previous report includes much greater detail to support the concerns raised.

Under competency 2.8 within the RN Scope of Practice, I maintain that the level of effective nursing care required by the individual client was not consistent. *My advice is that the lack of consistent evaluation and timely escalation based on the PEWS score and the lack of improvement of the child is a moderate departure from the expected care delivered by the registered nurse.*

Appropriate Specialist Input

I have read the response and reviewed the clinical documentation and observation charts which led to the referrals to the specialist nurse team and subsequent actions. The nurse specialists appear to have undergone rigorous training processes so are undoubtedly very qualified. Whilst one can appreciate that clinical judgement is of greater depth than a PEWS score and guidelines, I am concerned about the lack of improvement with borderline hypotension even after having several days of antibiotics inclusive of >24 hours of cephalosporins.

From the documentation, there were five reviews by various Specialist nurses from 1440hr on 26 [Month1] to 1125hrs on 17 [Month1]. The first four reviews are thorough with documentation demonstrating a clear plan of actions with some appropriate interventions. Based on the documentation, I maintain that it would have been appropriate for the specialist Nurses to provide some additional advice to the RNs on the floor such as increasing the monitoring frequency. Whilst there was appropriate escalation to the medical registrar following the reviews at 1440hrs and then at 0230hrs, there was no request to review by a senior doctor following the 1125hr review on 27 [Month1], even though there was an increased use of oxygen from 0.5L to 2L, a respiratory rate of 50 scoring 4 on the PEWS chart, and borderline hypotension of

90/39. The PEWS scores from 0200hrs until the transfer to [DHB2] were mostly 8+. Considering that the plan for transfer was on track for the early afternoon of that day, it would have been prudent for the specialist Nurse to request a senior medical review based on the increased evidence of work of breathing and possible circulatory compromise. Whilst I appreciate that this lack of escalation only occurred on this one instance, the importance of ensuring the stability of the patient prior to transfer is significant.

I believe that there is a lack of documentation of consistent supportive advice and lack of timely escalation which I advise to be a mild departure from the expected standard of care.

Appropriateness of transfer

The agreement by the nursing staff that this patient had no change in clinical condition is questionable. Based on a second review of the documentation, the observation charts and work of breathing from admission at 0430hrs on the 26 [Month1] through to the transfer to [DHB2] after 1300hrs on 27 [Month1] shows a patient that has made no improvement within the first 24 hours with increased work of breathing and increased respiratory rate, even with a reduction in temperature to 36.2°C. In addition, the patient has borderline hypotension for about 12 hours from about 1950hrs on 26 [Month1] to 0750hrs on 27 [Month1]. The PEWS score has increased from about 3 to 9. As stated in the previous review, which contains greater detail, this lack of improvement with some clinical signs of deterioration despite the patient having received about four (4) days of antibiotics inclusive of 24 hours of IV cefuroxime is concerning.

The frequency and timeliness of the nursing observations are of an appropriate standard. However, I disagree that there are no signs of deterioration. The issue being discussed is the appropriateness of transfer. As stated by the senior nursing staff, the decision to transfer was a senior medical decision, so I retract my statement that this decision was a severe departure from acceptable practice as I am not in a position to provide an expert perspective on medical practice. However, I am concerned about the lack of recognition of the deterioration of this patient from admission on the general paediatric ward at [ADHB] to the time of transfer to [DHB2].

Information provided to [DHB2]

As stated within the previous report, I maintain that the clinical picture within the transfer letter did not clearly reflect the condition of the client and consider this to be a moderate departure from the expected standard of care.

Recommendations

The information provided by the Nurse Director and Nurse Unit Manager surrounding the education, training and development of registered nurses working within [ADHB] is impressive. The orientation package, continuous development opportunities provided to RNs and the support of the PICU to equip the members of the specialist nurse team with the appropriate skills and knowledge appear rigorous.

The review of the PEWS chart and associated recommended actions are appropriate. There is good evidence of continual review processes aimed at RNs providing consistent high-quality care.

Based on the information provided, I do not have any further recommendations and feel that the overall training and professional development plans are commendable.

Signature:

Date: 10 November 2019”

Appendix D: Independent advice to the Commissioner

The following expert advice was obtained from paediatric surgeon Professor Mark Stringer:

"**Surgical Comment on the complaint: [ADHB] (Auckland District Health Board) re: [Miss A] (DOB …)**

Your ref: 18HDC00063

My comments relate to the surgical care provided by [ADHB] to [Miss A] (deceased) between 28 [Month1] to 11 [Month2]. I am a General Paediatric Surgeon with more than 20 years' experience as a Consultant Paediatric Surgeon. I have no conflict of interest to declare. My report is based on copies of the clinical notes, complaint and responses provided.

This is a very sad story and one which is devastating for [Miss A's] parents and family. Their questioning of certain aspects of [Miss A's] care is completely understandable and I acknowledge the detailed responses provided from [ADHB].

The complaints relate to multiple aspects of [Miss A's] medical care and not specifically to her surgical care. However, it is appropriate to review the latter.

Brief synopsis of clinical history relevant to [Miss A's] surgical care

[Miss A] was a six-year-old girl who presented to [ADHB] Emergency Department on 23 [Month1] with a three day history of fever and cough. She was discharged with a provisional diagnosis of lower respiratory tract infection. Two days later she re-presented to [ADHB] when she was admitted for treatment of pneumonia. Two days later (27 [Month1]) she was discharged to [DHB2]. Following a deterioration in her condition, she was found to have a right-sided pleural effusion (confirmed by ultrasound) and was transferred back to [ADHB] on the morning of 28 [Month1].

On 28 [Month1] an intercostal drain was placed by a paediatric surgical registrar for a right-sided parapneumonic effusion (p7 of supplementary notes). This was described as a simple effusion in the operation note dated 28 [Month1] and 400mls of 'straw coloured fluid' was drained. Intrapleural Urokinase treatment was prescribed. [Miss A] was transferred to the Paediatric Intensive Care Unit on 29 [Month1]. On 30 [Month1] intrapleural Urokinase was administered in PICU. The chest drain fell out 31 [Month1] (supplementary notes p47 and p50) and a repeat chest ultrasound scan later that day suggested only a small pleural effusion. A repeat chest ultrasound scan on 1 [Month2] reported underlying consolidated lung with only a small pleural effusion (maximum depth of 1.5cm over the right hemidiaphragm) (p280). However, by 3 [Month2] the pleural effusion had increased; a further ultrasound scan showed the effusion to be predominantly simple fluid with a few thin septations and an estimated volume of 146mls (p282).

On 4 [Month2] [Miss A] underwent a VATS procedure (video-assisted thoracoscopic surgery) (p417). At operation, the fibrin in her pleural fluid was described as 'very thin and easy to break down'. In addition, 'The visualised lung surface did not look grossly abnormal and certainly there was no areas that looked like hepatisation.' Two right-sided chest drains were placed (visible on the postoperative chest x-ray). Postoperatively, [Miss A] deteriorated and required ongoing ventilator support on PICU. She developed major surgical emphysema from a bronchopleural fistula. A CT scan of her chest on 5 [Month2] (p286) found that [Miss A's] right lung was almost completely consolidated with evidence of necrosis, extensive surgical emphysema, a possible right middle lobe bronchopleural fistula, small pneumatoceles in the right lower lobe and a loculated pleural collection anterior to the right lower lobe. There was consolidation and a pleural effusion around the left lung.

As [Miss A] deteriorated further, she was placed on veno-venous ECMO (7 [Month2]) but she developed multiple intracerebral haemorrhages and cerebral oedema and passed away on 11 [Month2]. Her pneumonia was attributed to Mycoplasma pneumonia and Influenza B infections (p7).

Comments

The surgical care provided by the paediatric surgical service was appropriate. There were no other surgical interventions that could have saved [Miss A]. In most cases, necrotising pneumonia responds to medical treatment with surgical intervention reserved for pleural drainage, empyema or rare complications such as a persistent bronchopleural fistula. The decision not to place a chest drain after the original drain fell out was reasonable and was appropriately guided by the results of ultrasound scans of the chest.

I have two minor criticisms of [Miss A's] surgical care. Neither would have impacted on the progress of her disease which was related to inexorable pulmonary parenchymal destruction.

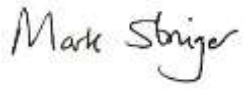
(i) There was confusion about the Urokinase therapy. This was prescribed (p206) after insertion of the first chest drain for the simple parapneumonic effusion. This decision would have been made by the surgical team. In the subsequent VATS operation note of 4 [Month2] it states 'For various reasons she [Miss A] did not receive appropriate Urokinase through this.' A dose of Urokinase was eventually administered through the chest drain in PICU on 30 [Month1] but the chest drain fell out on 31 [Month1] (supplementary notes p47 and p50) and no further doses were given.

In the presence of a simple parapneumonic effusion, some paediatric surgeons would not use Urokinase (a fibrinolytic agent) because fibrinous material was not evident on the chest ultrasound scan or in the pleural fluid. However, the [ADHB] Clinical Guidelines on Pneumonia (p33 of attached documents) state 'If the child is significantly compromised (high work of breathing, hypoxia, and/or persistent signs of sepsis), aren't making expected progress, or the effusion is very large, then additional intervention should be considered. This will usually be video-assisted thoracoscopic surgery (VATS)

with a chest drain or a chest drain with fibrinolytic therapy. Both of these interventions result in more rapid recovery than a chest drain or antibiotics alone.'

In [Miss A's] case, the Urokinase may not have been particularly beneficial but the fact is that it was intended to be given and was not and a VATS procedure was then undertaken one week later for what was considered to be a complex pleural effusion.

(ii) Unfortunately, the VATS procedure was never likely to make much of a difference to [Miss A's] condition, which was fundamentally one of necrotising parenchymal lung disease. However, the surgeons were faced with a severely ill and deteriorating child with a modest sized pleural effusion containing a few septations visible on ultrasound and so performing a VATS procedure was not unreasonable. Nevertheless, [Miss A] was not typical of the children we usually treat with an empyema. She had had a more protracted and complex clinical course. In such cases, a chest CT scan is often done *before* undertaking the VATS procedure to help gauge the significance of the pleural effusion and the severity of the parenchymal lung disease.



Professor Mark D Stringer MS FRCP FRCS FRACS
Paediatric Surgeon
Wellington Children's Hospital

Date: 24th August 2020"

Appendix E: Independent advice to the Commissioner

The following expert advice was obtained from paediatric intensivist Associate Professor James Tibballs:

"Your Reference: 18HDC00063

2/8/2020

Re: [ADHB] (Auckland District Health Board) ([Miss A] (dob …)

…

In your letter of 25th June 2020 you requested my opinion in relation to the care of [Miss A] at [ADHB] in [Month1] and [Month2], in particular whether the care provided in the Paediatric Intensive Care Unit (PICU) was reasonable.

In particular, you requested that I comment on:

1. Whether the care provided to [Miss A] between 29 [Month1] and 11 [Month2] was adequate/appropriate.
2. The timeliness of commencement of macrolide antibiotic treatment, and the appropriateness of the type of macrolide used.
3. The adequacy/appropriateness of testing and investigations, including for pneumonia and influenza.
4. Any other matters in this case that you consider warrant comment about the care provided by PICU.

In addition, you requested that for each question, please advise:

a. What is the standard of care/accepted practice?

b. If there has been a departure from the standard of care or accepted practice, how significant (mild, moderate, or severe) a departure do you consider this to be?

c. How would it be viewed by your peers?

d. Recommendations for improvement that may help to prevent a similar occurrence in future.

You also requested that if I note that there are different versions of events in the information provided, please provide your advice in the alternative. For example, whether the care was appropriate based on scenario (a), and whether it was appropriate based on scenario (b). While I note that you have requested my opinion of the care in PICU, I also note that the care provided by that service is a continuation of the care commenced elsewhere at [ADHB] and it is not possible to clearly separate and divide all responsibilities, but rather there are additional aspects exclusively the

responsibility of PICU. I also note that you have requested opinions from other paediatric specialties.

I believe that the matters fall within the scope of my medical practice. I am a full-time intensive care physician at the Royal Children's Hospital (RCH) Melbourne and have worked in that capacity since 1979. I am familiar with the principles of managing infections, including pneumonia, and of systems designed to warn staff of deterioration in the clinical condition of a child, having instituted a similar system for my hospital. This system, the Medical Emergency Team (MET) was published as the first world-wide for children and later adopted by the State of Victoria. In addition, I have been active in the formulation of the World guidelines for resuscitation by the International Liaison Committee on Resuscitation (ILCOR) and have published on the legal significance of clinical guidelines. I have furnished approximately 130 medicolegal opinions to legal representatives acting on behalf of plaintiffs or defendants in matters related to claims in deficiencies of health care.

I have the following academic and professional medical qualifications:

B Med Sci (Hons), 1970, Monash University, Melbourne.
MB BS, 1973, Monash University, Melbourne.
FANZCA, 1992–present, (Fellow of the Australian and New Zealand College of Anaesthetists)
FCICM (Fellow of the College of Intensive Care Medicine), 2010 (superseding FFICANZCA 1993 and FJFCIM 2002)
MEd, 1992, The University of Melbourne.
MBA, 1998, Deakin University.
MD, 1999, The University of Melbourne (Australian Venom Research Unit, Department of Pharmacology).
MHlth&MedLaw, 2006, (Master of Health and Medical Law), The University of Melbourne.
FACLM 2007 (Fellow of the Australian College of Legal Medicine).
PhD, 2019, The University of Melbourne.

SUMMARY OF MEDICAL MANAGEMENT

The following synopsis is my understanding of the course and treatment of [Miss A's] condition relevant to the complaint lodged by [Miss A's] family which according to the …Auckland District Health Board essentially is:

'If she had received earlier treatment for Mycoplasma, she would not have died.'

Some key physiological parameters for a child 5–11 years of age are the following:

Respiratory rate: 95th centile 34/minute; 99th centile 46/minute

Blood pressure (systolic): 95th centile 130 mmHg; mean blood pressure normal range approximately 60–90 mmHg

Heart rate: 95th centile 135/minute; 99th centile 150/minute

Emergency Department, [ADHB], 23 [Month1]

At [ADHB] on 23 [Month1] [Miss A], aged 6 years (22.5 kg), was diagnosed with a lower respiratory tract infection (pneumonia) on the basis of a history of cough and fever and on examination of reduced breath sounds in the lower right lung field. Her respiratory rate was 28 breaths/minute (a key index of severity of pneumonia) and percutaneous haemoglobin-oxygen saturation (SpO_2) 98% in room air (normal). It was noted that her mother was keen to have a chest X-ray performed because [Miss A's] sibling had had pneumonia over the previous 3 weeks, a diagnosis aided by a chest X-ray. [Miss A] was prescribed a course of oral amoxicillin but no chest X-ray was performed and no follow-up was arranged.

Comments:

1. It is noted that no history of the nature of the pneumonia and its treatment for [Miss A's] sibling was obtained. This is an error — it may have been important information to be used in guiding management considering that the siblings presumably shared common space at home and that [Miss A] may have contracted pneumonia from her sibling. Mycoplasma pneumonia (atypical pneumonia) is well-known for infecting specific communities such as a family with an incubation period of 2–3 weeks. It is not a rare cause of pneumonia among school-aged children. (Mycoplasma species are atypical bacteria in the sense that they do not have cell walls and thus not susceptible to medication which attacks bacterial cell walls, e.g., penicillins.)

2. By prescribing amoxicillin, it is evident that the doctor believed that [Miss A] had bacterial pneumonia, and that no other causative organism was considered. This is another error, since as stated it is difficult if not impossible to clinically differentiate pneumonia caused by bacteria, viruses and Mycoplasma in the early stages of infection.

3. No tests were performed to identify the infecting organism. It is stated in various places in the reviews that no test is available to identify the infecting organism. This is not exactly true. While the species of bacterium may not be reliably identifiable by non-invasive tests, a naso-pharyngeal aspirate may be used to identify viruses and Mycoplasma. This could have been done in the Emergency Department on 23 [Month1] with results made available within several days if not sooner.

4. A chest X-ray was not performed. While it is true that a chest X-ray is not always needed to diagnose pneumonia, as stated in the guidelines, it is problematic when a mother of a sick child requests that it be performed knowing that her other sibling had pneumonia with a diagnosis aided by chest X-ray. In other words, it is imprudent to dismiss the concern of a parent at any time, especially one who has informed knowledge.

Expert reviewers at [ADHB] stated that chest X-rays did not permit them to identify features which are diagnostic of Mycoplasma pneumonia. While Mycoplasma has the reputation of causing radiological features in excess of clinical symptoms, the

radiological findings in severe Mycoplasma pneumonia are so variable and nonspecific that it is impossible to differentiate it from pneumonia caused by viral and bacterial causes. In other words, the chest X-Ray cannot reliably confirm or exclude Mycoplasma pneumonia which means that Mycoplasma must always be considered as a possible cause of pneumonia. Some texts, e.g. Nelson's textbook of Pediatrics (20th edition, 2011, page 488), state that Mycoplasma accounts for up to 20% of all cases of pneumonia in children.

5. No follow-up was organised. This is another error. Pneumonia is a serious disease. If the presumptive causative organism is erroneous, the treatment will be ineffective and the pneumonia will progress. Sensibly, advice was issued to seek medical assistance if [Miss A's] condition worsened but failure to organise follow-up (with any service) was below a reasonable standard of care for diagnosed pneumonia.

Emergency Dept and Ward [ADHB], 25–27 [Month1]

At [ADHB] on 25 [Month1] [Miss A] represented with worsening symptoms and signs. She had a persistent cough and fever and with increased work of breathing (mild) consisting of subcostal retraction and respiratory rate 25–30 breaths/minute. Her SpO_2 was 93–95% (just below normal). She had vomited several times, associated with coughing. A chest X-ray revealed right middle lobe pneumonia with a small right pleural effusion. Her antibiotic treatment was changed to intravenous Cefuroxime and she was admitted to a ward.

Initially her respiratory rate remained in the 30's but at the ward round of the morning of 26 [Month1] it was recorded as 44/minute, her work of breathing had increased and she required a small amount of oxygen (0.5 L/min) to maintain the SpO_2 at around 95–97%. Her fever continued.

In the early afternoon of 26 [Month1], her paediatric early warning score (PEWS) was 9. She was attended by a nurse and then by a doctor. The latter at 14:45 hours recorded a respiratory rate of 48/minute, heart rate 145/minute and temperature 39°C. Intravenous fluid, which had ceased earlier in the day, was recommenced after a bolus and paracetamol was given but the antibiotic treatment was not changed. At 21:00 hours on 26 [Month1] when reviewed by a nurse, [Miss A's] respiratory rate was still 48/minute with mild work of breathing but her heart rate had decreased to 90/minute and her temperature was reduced. Her PEWS score was 7. At 02:30 hours on 27 [Month1] when again reviewed by a nurse, her PEWS was again 9, comprised of a respiratory rate of 52/minute and moderate work of breathing and still requiring oxygen at 0.5L/minute. At the end of the regular overnight nursing shift, the nurse recorded that at times her respiratory rate had been 55/minute during the night. However, by 05:30 on 27 [Month1] when examined by a doctor her respiratory rate had reduced to 30/minute but requirement for oxygen remained. Transfer to another hospital was organised and confirmed at 07:30 hours on 27 [Month1]. However, when followed up by the specialist nurse at 08:53 hours on 27 [Month1] [Miss A's] respiratory rate was 40/minute with a PEWS of 5 without the need for oxygen. However, at 11:25

hours on 27 [Month1] her respiratory rate was 50/minute with a PEWS of 7. The reason for review at the latter time was that her respiratory rate had been 61/minute with a PEWS of 10. Towards the end of the regular nursing shift, the nurse recorded at 13:15 hours on 27 [Month1] (and confirmed by the observation chart) that [Miss A's] respiratory rate had been up to 60/minute and her heart rate up to 144/minute during the late morning and early afternoon on 27 [Month1], and she was breathless and continued to require oxygen. After these observations she was transferred without further medical consultation to [DHB2] for on-going care.

Comments:

1. Again, no clinical personnel considered whether or not [Miss A] might be suffering from non-bacterial pneumonia. The antibiotic was changed from oral Amoxycillin to intravenous Cefuroxime with the underlying assumption that the pneumonia was bacterial. No test was conducted to look for other causes, neither viral nor Mycoplasma.

2. The deterioration in [Miss A's] condition in the mid-morning to early-afternoon on 27 [Month1] was not conveyed to medical staff. It is as if the nursing and medical teams were conducting separate parallel practices. The instructions to nursing staff are stated explicitly on the observation chart that if the PEWS is 8+, the doctor must review the patient within 15 minutes and the senior doctor must review within 30 minutes. Obviously, to effect such action, the doctors must be informed. The advice to nurses should be even more explicit than it is such as: 'Inform the doctor who must review within 15 minutes etc.' In this case, doctors do not appear to have been informed. This is a practice below standard. If they had been informed of [Miss A's] deterioration before her transfer the outcome may well have been different. It may have prompted them to wonder why [Miss A's] pneumonia had not responded to antibiotic treatment but rather had worsened.

3. It appears that [Miss A] was never examined by a senior doctor. Such was probably present on ward rounds but no notes have been entered into the medical record by a senior doctor.

Emergency Dept, Operating Theatre and Recovery Room, Ward

[Miss A] was returned to [ADHB] on 28 [Month1] after apparent recognition at [DHB2] (no notes provided) of a pleural effusion, and worsening respiratory status. In the Emergency Dept (ED) of [ADHB], high flow nasal prong (HFNP, 40L/minute) oxygen was required (it provides a small amount of continuous positive pressure as well as a higher inspired oxygen concentration) and ongoing fever. A chest X-ray was apparently performed at 09:15 hours on 28 [Month1] but its report is not included in the record. However the next chest X-Ray (at 18:39 hours on 27 [Month1]) refers to a previously identified 'large right pleural effusion', 'extensive right lung consolidation' and 'consolidation within the left upper zones'. A registrar from the Paediatric Intensive Care Unit (PICU) was consulted (with a view of admitting her to PICU) but decided to leave her in the ED. The antibiotic Clindamycin was added to the Cefuroxime. A plan

was made to insert a chest tube to drain the pleural effusion in the operating theatre under anaesthesia. In the meantime [Miss A] was managed in the resuscitation area of the ED.

The Infectious Disease (ID) service was consulted on 28 [Month1]. From this the paediatric registrar noted that it was important to subject pleural fluid to microscopic examination and testing for Pneumococcus (bacterial) antigen.

Comments:

1. Once again no-one in the Emergency Dept considered that [Miss A] might have non-bacterial pneumonia. No test was conducted to look for viruses or Mycoplasma.

2. Neither the PICU registrar nor the Infectious Disease representative wrote any notes. The PICU registrar probably did not review the antibiotic treatment while the ID representative conveyed information related only to the assumption that [Miss A] was suffering a bacterial pneumonia. Mycoplasma would not be detectable on the planned microscopic examination of stained pleural fluid.

3. These omissions represent another lost opportunity to treat advanced non-bacterial pneumonia which if it had been given at this late stage may well have prevented death. Early treatment of an infection is important. Antibiotic treatment, even when appropriate, does not necessarily eradicate infection when given late in the course of infection.

On 28 [Month1] in the early evening a right chest drain was inserted by a surgeon in the operating theatre under sedation administered by an anaesthetist. An ultrasound examination showed that the effusion was not loculated. Between 17:20 and 17:50 hours, approximately 420 mls of straw-coloured fluid was drained on insertion of the drain which was then left to drain freely. Some fluid was sent for microbiological and biochemical tests. Tests included PCR (Polymerase Chain Reaction) testing for DNA of causes of atypical pneumonia. The results of those tests were 'signed out' from the laboratory on 9th [Month2] (12–13 days later) and included positive detection of Mycoplasma pneumoniae (*vide infra*).

The anaesthetist administered, in addition to sedative medication, the antibiotic Cephazolin (a cephalosporin antibiotic). [Miss A] was then admitted to the recovery area before admission to PICU at 08:00 on 29 [Month1]. During the period in recovery, from approximately 19:00 hours on 27 [Month1] until 08:00 hours on 29 [Month1], [Miss A] had a persistently high respiratory rate (50–60/minute) with PEWS 8–12 and required oxygen. In other words, draining the pleural effusion did not improve her condition much, if at all. A chest X-ray performed at 18:39 hours on 28 [Month1] (in recovery room) showed that the 'large right pleural effusion and extensive right lung consolidation are unchanged'. Knowing that 420 mls had been drained away in the operating theatre within the previous hour, this must mean either that pleural fluid was forming rapidly or that the pleural effusion had only been partially drained. The latter is the probable explanation given that another 198 mls issued from the tube from 19:00

on 28 [Month1] to 07:00 hours on 29 [Month1]. The radiologist also reported that there was subcutaneous emphysema — a collection of air under the skin outside the chest, which is not a pneumothorax (air collected in the pleural space), implying that the surgeon had inadvertently allowed external air to gain access to the subcutaneous tissues but not the pleural space and had not damaged the lung surface on insertion of the drain.

Comments:

1. Neither the anaesthetist nor the surgeon wondered why [Miss A's] condition had not improved with antibacterial treatment, representing another lost opportunity to revise the diagnosis.

2. It is unknown if the results of the PCR testing for atypical pneumonia in the pleural fluid were all or partly available before 9th [Month2], and if they were, were the results conveyed urgently to the treating clinicians. I note that similar tests were conducted on pleural fluid collected on 4th [Month2] and again the results were signed out of the laboratory on the same date as the previous ones, that is, on 9th [Month2]. If the 'sign out' date means the date they were released, it is obvious that the results of tests from the fluid sample collected on 28 [Month1] could have been available considerably earlier than 9th [Month2]. It is almost needless to say, positive results ought to be conveyed to clinicians as soon as possible. I am accustomed at my institution to be made aware of PCR results after 2 days. Obviously, if the results had been known on say 30th [Month1], a change in antibiotic treatment would have been instituted earlier than 5th [Month2] when Erythromycin (active against Mycoplasma species) was added to the therapy but very late into the course of illness.

PICU, 29 [Month1]–11 [Month2]

[Miss A] was then admitted to PICU on 29 [Month1] for high flow nasal prong (HFNP) oxygen when her condition had worsened and she had developed some oedema and a maculopapular rash which were interpreted as components of a systemic inflammatory response syndrome (SIRS) in response to presumed bacterial infection. Another chest X-ray at 23:07 hours on 28 [Month1] revealed little change but another on 30 [Month1] revealed progressive left upper lobe consolidation and left pleural effusion, indicating that [Miss A] had severe bilateral lung involvement. In addition, the radiologist noted that the opacification of the right lung was extensive and had not changed since placement of the drain on 28 [Month1], and wondered whether the position of the drain was satisfactory. (Subsequently, the drain tube 'fell out' or dislodged somehow, *vide infra*). A chest X-ray on 31 [Month1] showed 'significant opacification of the right hemithorax … due to a combination of consolidation and pleural material'. A subsequent ultrasound study on 31 [Month1] showed 'a large amount of consolidated lung in the right chest' and 'a small–moderate amount of pleural fluid' while another study on 1 [Month2] showed a small volume of fluid around the right lung.

[Miss A's] parents were very concerned with her lack of progress. A PICU registrar recorded the following on 29 [Month1]:

'Explained natural course of pneumonias. Now looking back it might not feel as the correct management, however it is according to protocol.'

On the 29 [Month1] the medical staff had received some results from the pleural fluid examination which included a high white cell count, negative result for Streptococcus pneumoniae antigen and the absence of organisms on a Gram-staining and culture results were not yet available. A blood culture from the 28 [Month1] was negative. On clinical examination [Miss A's] respiratory rate was 55–65/minute and SpO_2 95% while being given 40L/minute of 55% oxygen via HFNP.

On 30 [Month1], consolidation of the left upper lobe was evident along with previously known small amount of left pleural fluid.

On 31 [Month1] [Miss A's] condition remained static with respiratory rate of 45–65/minute but her work of breathing as described by various staff was moderate to severe. The chest drain became dislodged on mobilisation. Since only a small amount of fluid was present in the right pleural space, as detected by ultrasound (repeated over ensuing days), a decision was made not to replace it.

On 1 [Month2], the oxygen therapy was changed to low flow of 100%. [Miss A's] respiratory rate remained at around 60/minute and the SpO_2 was around 91% with increased work of breathing. HFNP at 40L/minute was re-instituted. Respiratory rate sometimes exceeded 70/minute. Alternative HFNP oxygen or low flow oxygen were administered according to SpO_2, respiratory rate and work of breathing. The rash which had been recorded days previously, and had been described as polymorphous, was now global and mottled.

On 2 [Month2], staff knew that the pleural fluid, collected on 28 [Month1], had not grown any organisms. A surgical representative proposed discussion with the infectious disease (ID) department whose representatives (two) suggested doing a pneumococcal antigen test on the pleural aspirate. Whether this was meant to be a repeated test (it had already been done twice on 28 [Month1] and 28 [Month1] and results released on 29 [Month1] and 2 [Month2] respectively) and was known to be negative or whether this was proposed as a new test, third test (which was negative) is not declared. One of the ID physicians opined that the rash was due to a drug reaction to the Cefuroxime which was then ceased with continuation of Clindamycin alone. A trial of oxygen administration by Hudson mask was tried on this day but abandoned due to resultant increased work of breathing.

On 3 [Month2], on another review by the infectious disease department, the ongoing fever, raised CRP (C-reactive protein), respiratory distress, oxygen requirement did not stimulate a reconsideration of the diagnosis. The widespread rash was still considered due to a drug reaction and it appears no consideration was given to other possible causes which include the known phenomenon of a rash associated with Mycoplasma pneumoniae infection. On 3 [Month2], another ultrasound examination and a chest X-ray showed an increase in the right pleural effusion (estimated volume 146 mls) with

continued extensive consolidation of the right lung. Respiratory therapy at this time was HFNP 50% oxygen at 35L/minute while [Miss A's] respiratory rate was 60–80 minute, SpO_2 85% and above, and work of breathing described as moderate with her fever reaching 40°C.

Overnight 3 [Month2]–4 [Month2], [Miss A's] respiratory rate varied between 40 and 80 breaths/minute. The PICU consultant acknowledged the difficult situation of not knowing the aetiology of the pneumonia which was worsening. Although nutrition had been adequate up to this point, it was to be facilitated with planned insertion of a nasogastric tube under anaesthesia for the video-assisted thoracoscopic (VATS) procedure with drainage of the pleural fluid on 4 [Month2]. At that procedure, around 400 mls of turbid fluid was removed from the right pleural space. The surface of the lung had early emphysematous changes. The procedure was performed under general anaesthesia with endotracheal intubation. Mechanical ventilation was continued on [Miss A's] transfer back to PICU. The surgeon recommended consideration of an empirical change of antibiotics given the lack of improvement. This implies that the results of DNA testing of pleural fluid collected on 28th [Month1], which was positive for Mycoplasma, were either not yet available, or had not been conveyed to clinical staff, or if they had been conveyed they had not been heeded.

On return from the operating theatre, mechanical ventilation was continued with a CPAP pressure-assisted mode initially with pressures (16/8) with 65% oxygen yielding a tidal volume of 120 mls (5–6 ml/kg) and SpO_2 92%. The plan was to reduce ventilation according to lung function ('to wean the ventilation') and extubate the next day. However, [Miss A's] condition appeared to deteriorate further overnight 4–5 [Month1] during which additional mechanical ventilation was required (pressures 26–30/10) while a vasopressor (noradrenaline) and boluses of fluid were required to treat hypotension (mean pressure 45 mmHg). The cause of this was considered to be worsening sepsis.

On 5 [Month2] on review by the infectious disease team, it was recognised that the antibacterial cover provided by the monotherapy with Clindamycin did not include activity against Mycoplasma but which was considered to be an 'unusual cause of such severe disease' but 'in view of lack of progress a macrolide antibiotic should be considered'. For this purpose, Erythromycin was commenced on 5 [Month2], which is very late into the course of illness.

Throughout the 5 [Month2], [Miss A's] condition continued to deteriorate. Increasing ventilator pressures and oxygen were required and subcutaneous emphysema appeared and became widespread. A CT scan of the chest revealed only a small amount of air leak into the pleural space but the right middle lobe appeared necrotic with a pneumatocele (intrapulmonary cyst of air). The air leak from the lung was regarded as a broncho-pleural fistula. In an effort to reduce air leakage from the lung, the ventilator pressures were reduced to 15/5 and respiratory acidosis permitted (permissive hypercapnia, $PaCO_2$ around 90–105 mmHg) but oxygenation was poor (PaO_2 60–70

mmHg) even with very high inspired oxygen (95%). Extra-corporeal membrane oxygenation (artificial heart and/or lung support, ECMO) was discussed.

Over the course of 6 [Month2] [Miss A's] lung function deteriorated further with $PaCO_2$ approximating 130 mmHg despite mechanical ventilation at pressures 23/5. A decision was made to commence veno-venous ECMO.

In the morning of 7 [Month2], a trial of inhaled nitric oxide was conducted but it did not improve gas exchange and was discontinued. In the afternoon of 7 [Month2], a surgeon inserted cannula for ECMO. According to the 'ECMO Data Sheet' (page 431), one cannula was inserted into the 'jugular vein' and another into the 'right femoral artery'. By definition, this placement of cannulae constitutes a set-up for veno-arterial ECMO (V-A ECMO), not veno-venous ECMO (V-V ECMO). However, every nursing and medical entry to the record after insertion of the cannulae refers to [Miss A] being given veno-venous ECMO and all recordings related to ECMO were recorded on an 'adult V-V ECMO chart'. (For both types of ECMO, the same anti-coagulation therapy is required to prevent formation of thrombosis (clots) in the circuit but exposes the patient to the considerable risk of spontaneous haemorrhage). Up to commencement of ECMO, an infusion of noradrenaline had been required with the aim of maintaining the mean arterial blood pressure (MAP) above 55 mmHg. During institution of ECMO the noradrenaline was temporarily increased and after its institution, it was reduced and then ceased in the afternoon of 7 [Month2] but the MAP was still 90–100 mmHg (hypertensive). Additional sedation and then a vasodilator (Milrinone) by infusion were given to control the hypertension but this was unsuccessful (MAP still 91 mmHg) and it was replaced by an infusion of sodium nitroprusside in the afternoon of 8 [Month2]. However, with an infusion of SNP at 2.5 mcg/kg/minute, the MAP had (only) decreased to 85–90 mmHg.

Anticoagulation therapy with heparin was administered with the aim of maintaining the activated clotting time (ACT) appropriately between 160–180 seconds. Overnight (07–08 [Month2]) when the ACT was excessive (316 seconds) the heparin was reduced from 13.8 units/kg/hr to 10 units/kg/hr but the ACT was still high at 231 units/kg/hr. According to the nursing entry in the records, heparin was given 'according to the protocol'.

After institution of ECMO, mechanical ventilation was appropriately reduced to 'lung rest' settings to allow spontaneous resolution of the disease process and recovery of lung tissue. A naso-pharyngeal aspirate (NPA) was collected and a broncho-alveolar lavage was performed on 7 [Month2] for pathogen detection.

On 8 [Month2], on a ward round of the infectious disease team, it was acknowledged that [Miss A's] [sibling] had recently been unwell for 2 weeks with pneumonia, sore throat and a rash about one month previously. Their pneumonia had been diagnosed with the aid of a chest X-Ray and they had recovered with the aid of antibiotics whose identities were unknown. In addition, [Miss A] had become unwell about one week

after her [sibling]. On this basis, the team opined that with the [sibling's] illness, [Miss A's] illness was 'highly suspicious of Mycoplasma'.

In the morning of 8 [Month2], [Miss A's] pupils were of normal size and reactive to light but in the afternoon of 8 [Month2], they were dilated and unreactive. A CT of the brain at around 17:00 hours on 8 [Month2] subsequently revealed significant intracerebral haemorrhages. After this care was directed at comfort but ECMO was continued while waiting for [Miss A's] extended family to arrive. Excessive urine output suggested onset of diabetes insipidus (a sign of lack of pituitary gland function).

Overnight 9–10 [Month2], PICU medical staff acknowledged the report of Mycoplasma in specimens collected on 28 [Month1] and 4 [Month2] and of Influenza B from the nasopharyngeal aspirate collected on 7 [Month2].

In explanation given to [Miss A's] parents on 9 [Month2], the cause of [Miss A's] pneumonia appeared to have centred only on Influenza as the cause of [Miss A's] pneumonia.

Clinical testing for brain death was conducted (in the presence of sedatives) by two physicians and each found no evidence of activity. The ECMO was ceased on 11 [Month2].

COMMENTS

The four specific questions that you posed:

'1. Whether the care provided to [Miss A] between 29 [Month1] and 11 [Month2] was adequate/appropriate;

2. The timeliness of commencement of macrolide antibiotic treatment, and the appropriateness of the type of macrolide used;

3. The adequacy/appropriateness of testing and investigations, including for pneumonia and influenza;

4. Any other matters in this case that you consider warrant comment about the care provided by PICU'

are addressed among the following multiple questions concerning [Miss A's] management in PICU.

1. Why was non-invasive respiratory support continued for so long before intubation with mechanical ventilation was instituted?

High Flow Nasal Prong (HFNP) oxygen treatment is usually only given for mild respiratory illness. It provides, in addition to oxygen, a small amount of continuous positive airway pressure (CPAP). It may be useful in avoiding invasive mechanical ventilation but in severe pneumonic disease, as [Miss A] had, it is not appropriate and below a reasonable standard of care. Of course mechanical ventilation is not without risk, and the use of HFNP may be used to avoid risks but the use of HFNP in severe lung

disease will not avoid hypercapnia, may not avoid hypoxaemia and leave the patient with the discomfort of respiratory distress. If mechanical ventilation had been given earlier, it may have limited the progression of lung disease. In addition, it would have permitted better clearance of secretions from the lungs (which were copious) and the opportunity to take tracheal aspirate or bronchial lavage samples for analysis to make the appropriate diagnosis. This is not to say that mechanical ventilation is without risk, one of the most important being leakage of air from the lungs, but that risk is probably proportional to the amount of positive pressure given and in [Miss A's] case, by the time it was given the degree of lung disease necessitated considerable pressure making air leak more likely.

2. Why was there not more effort in trying to diagnose the cause of pneumonia?

The PICU staff, like all other medical and surgical staff before admission to PICU, had the responsibility to diagnose the cause of [Miss A's] pneumonia. They did not take a nasopharyngeal aspirate until the 7 [Month2] (9 days after admission to PICU), did not do a tracheal aspirate until 6 [Month2] and did not do a broncho-alveolar lavage until 8 [Month2] (no result recorded). They did not seek the opinion of the Infectious Disease department until 2 [Month2], and if there was a respiratory medicine service at the hospital, it appears that they were never consulted.

3. Why didn't the staff consider non-bacterial causes of pneumonia?

The PICU staff, like other members of the healthcare teams, did not consider Mycoplasma or viral causes of pneumonia. This is not only the province of the infectious diseases department but is the responsibility of every clinician tasked with diagnosing the cause of pneumonia.

4. Why weren't the results of tests for atypical pneumonia reported and/or pursued earlier?

It is incongruous that the results of the tests for atypical pneumonia taken on 28 [Month1] and 4 [Month2] were both acknowledged on the same day, 9 [Month2]. This implies that the result of the tests on 28 [Month1] would have been available earlier than 9 [Month2], no later than 2 [Month2]. Of course, there are many opportunities for information to get lost or not conveyed to the staff responsible for ordering treatment. Multiple possibilities exist, some examples are:

(i) The test on the sample may have been postponed in the laboratory to be done among a batch with others;

(ii) The result may not have been communicated to the PICU staff;

(iii) The result may not have been regarded as important

(iv) The result was not pursued by PICU staff.

The last named is a real possibility because even when Mycoplasma had been identified, the staff spoke to [Miss A's] parents mentioning only Influenza infection. In

one Social Worker's note, it was actually [Miss A's] parents who enquired about Mycoplasma.

Mycoplasma is detectable by PCR testing over a number of hours. In this case it appears that a minimum of 5 days was the detection time. This practice is below an acceptable standard of care and requires investigation.

5. Why wasn't Mycoplasma considered earlier to be a likely diagnosis and treated earlier?

For many days, the PICU staff seemed bereft of ideas when presented with a case of inexplicable pneumonia. One can only speculate about their knowledge of the causes of community acquired pneumonia, as with the staff who had encountered [Miss A] before admission to PICU. This is particularly so when the patient has not responded to antibiotic treatment aimed at bacterial causes.

A considerable amount of circumstantial evidence suggested that [Miss A] could be suffering from Mycoplasma pneumonia:

(i) The pneumonia was probably acquired from a sibling in a family setting

(ii) The pneumonia was not responding to usual anti-bacterial treatment

(iii) A rash was consistent with Mycoplasma infection

(iv) The presence of cold agglutinins detected by Full Blood Count examinations on 2 [Month2], 4 [Month2], 5 [Month2], 6 [Month2], 7 [Month2], 8 [Month2] (3 times) is consistent with Mycoplasma infection

(v) Absence of bacteria detected on microscopy of pleural fluid on 29 [Month1] and 4 [Month2]

(vi) Repeated absence of bacteria in blood cultures and in pleural fluid.

The diagnosis of Mycoplasma pneumonia was confirmed late by tests conducted late and reported late. The consequent late administration of an appropriate antibiotic (e.g., Erythromycin) is a standard of care below reasonable.

6. Why weren't viral causes considered earlier to be the causes of pneumonia?

The same may be said about viral causes as about Mycoplasma. If the patient is not responding, and indeed whose condition is ingravescent in spite of antibacterial treatment, a search must be made for other pathogens, including respiratory viruses but this was not done until 7 [Month2] when a nasopharyngeal aspirate was taken and was positive for Influenza B.

6. Why wasn't the Infectious Diseases team consulted earlier?

They should have been consulted earlier during the PICU admission when it was recognised that [Miss A's] condition was not responding to the antibacterial treatment being administered. Admission to PICU on 29 [Month1] might have been an appropriate

time to get them re-involved. Again, there was an acceptance that the cause of pneumonia could only have been bacterial.

7. Why didn't the Infectious Diseases Team consider Mycoplasma as the likely diagnosis earlier than they did?

This is an oversight or omission on their behalf, as well as by the Emergency Dept, attending ward physicians, surgeons and PICU staff.

8. Why weren't less injurious forms of mechanical ventilation considered?

After mechanical ventilation was started in the operating theatre for the purpose of a VATS on 4 [Month2], mechanical ventilation was continued in PICU, but emphysematous changes had been observed on the surface of the right lung at that time on 4 [Month2] and surgical emphysema appeared within 24 hours of the start of mechanical ventilation. That was the time to consider for example whether high frequency oscillation ventilation (HFOV) if available ought to have been given. But that was not considered (and then by a physiotherapist) until 5 [Month2] and never used. Although HFOV is another form of positive pressure ventilation, the peak pressures required are less than with conventional mechanical ventilation and may have posed less risk for causation of a bronchopleural fistula and widespread emphysema and the subsequent need for ECMO.

9. Were there any consequences of the documentation that cannulae placed in vessels would be defined as V-A ECMO and not V-V ECMO?

Probably not. Either there was a mistake in documentation (see page 431 ECMO data sheet) or the PICU team has made an error in assuming V-V ECMO was being given when in fact it was V-A ECMO. There does not appear to be surgical record of the cannulation. Someone has probably mistakenly written 'femoral artery' when actually the femoral vein was cannulated or the PICU staff wrongly assumed that V-V ECMO was being given instead of V-A ECMO. If there were a mistake in documentation on the ECMO data sheet concerning the location of the femoral cannula (artery vs vein), it has not been later corrected.

I note that the radiologist's report of the chest X-ray of 7 [Month2] states that a cannula was located in the right femoral artery and in the report of the subsequent X-ray no correction has been made.

If PICU staff assumed they were giving V-V ECMO when in fact V-A ECMO was being given, there would have been no serious consequences during the performance of ECMO if it was run according to desired achievements in gas exchange and haemodynamic performance. However, there may have been a perception that there is no need to specify BP and haemodynamic targets during V-V ECMO because the ECMO is provided to substitute for lung function, not heart function. In contrast, it is essential to specify blood pressure and other haemodynamic targets during V-A ECMO because the ECMO is substituting for both lung and heart function. It appears that orders for V-V ECMO at [ADHB] do not usually contain an upper limit for blood pressure but they do

contain appropriate target ranges for pump flow, blood pH, blood PCO_2 and haemoglobin-oxygen saturation (SO_2), ACT range, platelet count and HCT (haematocrit). On 7 [Month2], the orders contained no hemodynamic targets (apart from flow) whereas on 8 [Month2] there was a lower limit of BP and a minimum venous pressure but no upper limit of blood pressure. The cerebral haemorrhage occurred during this time.

At the bedside, differentiation between V-V ECMO and V-A ECMO is readily made, provided that each is applied to appropriate circumstances. For example, during V-A ECMO given to substitute for poor or no native heart function, with the ECMO blood flow provided being non-pulsatile, there are poor or no palpable pulses and little or low pulse pressures (difference between systolic and diastolic pressures) until the heart recovers function. During V-V ECMO when the heart is functioning adequately or normally from the outset, there should be palpable pulses and easily discernible pulse pressures.

If V-A ECMO is given when the heart is functioning normally ([Miss A] had normal heart function with some support with noradrenaline), the flow provided by the ECMO would add to the flow created by normal heart contraction. In this situation, there would be palpable pulses and pulse pressures, as is usually observed when V-V ECMO is given appropriately for lung failure alone. In other words, the presence of pulses and pulse pressures is always observed in appropriately applied V-V ECMO but these may also be observed when V-A ECMO is applied erroneously. The question arises: Were the staff duped into believing that they were managing V-V ECMO rather than V-A ECMO?

There may be several important consequences if V-A ECMO is given erroneously when the heart is functioning normally:

(i) The excessive blood flow due to ECMO blood flow plus the native cardiac output may cause hypertension (as was observed).

(ii) The oxygenated blood pumped by the ECMO machine into the aorta via the femoral artery will run against (counter to) the flow of poorly oxygenated blood pumped into the aorta by the heart. This confrontation of flows may result in inadequate supply of oxygenated blood supply into the coronary and carotid arteries resulting in hypoxaemia of the heart itself and of the brain.

(iii) A large cannula located in a femoral artery for V-A ECMO, which directs ECMO flow towards the heart, obstructs blood flow to that leg. This problem is overcome as a matter of routine by providing a smaller cannula directing some flow distally, that is, down the leg. Since this was not provided in [Miss A's] case, it is a strong reason to believe that the ECMO provided was not V-A ECMO, but truly V-V ECMO. Moreover, no observations over the period 7–11 [Month2] suggest that there was different blood supply to the right and left legs.

(iv) A problem will arise when the patient's lung function begins to recover and the clinicians want to wean off the ECMO that they believe erroneously to be V-V ECMO.

With V-V ECMO, weaning is accomplished by reducing the gas flow to the membrane oxygenator but if this is done while on V-A ECMO a state of hypoxaemia may be caused because the ECMO then functions as a shunt, bypassing lung function. With V-A ECMO, weaning is accomplished by gradually reducing the blood flow as the native heart function improves.

Importantly, the anticoagulation is the same with both V-A ECMO and V-V ECMO.

In the absence of surgical documentation of the femoral cannulation, I suspect that there was an error in documentation of the cannula placed in a femoral vessel, that is I believe that V-V ECMO was being given. V-A ECMO is usually given for heart failure or both heart and lung failure alone whereas V-V ECMO is given for lung failure alone (for additional reasons beyond the scope of this report) and was appropriate in [Miss A's] situation. However, a tinge of doubt remains that the ECMO was truly veno-venous.

10. Why was anticoagulation on ECMO excessive?

Unfortunately anticoagulation was (briefly) excessive causing the ACT to be very prolonged for several hours. I do not have enough information at hand to be able to opine why this occurred. That is, whether or not there was any management below a reasonable standard. According to entries to the record, the anti-coagulation was managed 'according to protocol' but that cannot be strictly true or the protocol is deficient since it did permit excessive anti-coagulation. The protocol for heparin administration is not provided, that is for example how much to increase/decrease the dose of heparin when ACT measurements are outside target ranges.

The increases in heparin therapy on 7 [Month2] do appear appropriate (page 432), e.g.: from 10 to 11 (u/kg/hr) when ACT was 123 (secs), from 11 to 12 when ACT was 101; from 12 to 13.8 when ACT was 111 and reductions in heparin from 13.8 to 10 when ACT was 316. However ACT remained beyond the desired range (160–180 secs) for about 6 hours at a dose of 10 u/kg/hr from 03:00 to 09:00 hrs on 8 [Month2]. During this time the platelet count was adequate (204 at 22:00 7 [Month2], 202 at 03:00 hrs 8 [Month2]), but the APPT (activated partial thromboplastin time) was beyond the therapeutic range (50–80 seconds) at 96 seconds at 03:00 hrs on 8 [Month2].

11. Why was hypertension permitted to endure on ECMO?

Before institution of ECMO, a vasopressor (noradrenaline) was required to support blood pressure whereas after institution of ECMO the noradrenaline was associated with hypertension. Indeed, a vasodilator to lower blood pressure was required not long after institution of ECMO which was at 13:26 hrs on 7 [Month2]. It seems that the vasodilator (Milrinone) was inadequate in dose and a switch was made to sodium nitroprusside. Since the hypertension lasted a considerable number of hours from approximately 16:00 hrs on 7 [Month2] until the performance of a brain CT scan at 17:00 hrs on 8 [Month2], it is fair to say that the PICU staff did not act in sufficient time or with appropriate medication to control hypertension. Before institution of ECMO (at 13:26 hrs on 7 [Month2]) the range of blood pressure (BP) from 02:00 to 13:00 hrs on 7 [Month2] was

90–95/40–45 (systolic range/diastolic range) mmHg with a mean blood pressure (MAP) 55–65 mmHg, whereas after institution of ECMO from 16:00 hrs on 7 [Month2] to 17:00 hrs on 8 [Month2] (when the CT brain was performed) the BP was 110–140/65–95 with an hourly MAP average of 97 mmHg. This period is more than 24 hours of severe hypertension. Noradrenaline (vasopressor) therapy was ceased at 17:00 hrs on 7 [Month2] while Milrinone (vasodilator) therapy was commenced at 20:00 hrs on 7 [Month2] and sodium nitroprusside (vasodilator) commenced at 11:00 hrs on 8 [Month2]. These measures were not sufficient to control hypertension. During these periods there were no medical orders containing upper limits of blood pressure while on ECMO.

In fluid systems, pressure, flow and resistance are linked by Ohm's equation: Pressure = Flow x Resistance. In order to reduce blood pressure, apart from better vasodilator therapy to decrease resistance, the blood flow provided by ECMO could have been reduced. The range of specified flow in the parameters to be followed (protocol, medical orders) was 2.5–3.0 L/minute on 7 [Month2] and 8 [Month2] (but there are no ECMO orders for the following three days). The actual average hourly flow delivered during the period 16:00 hrs on 7 [Month 2] to 07:00 hrs on 8 [Month2] was 2.92 L/minute and from 08:00 hrs to 17:00 hrs on 8 [Month2] (when a brain CT was performed) was 2.84 L/minute. In other words, there was no attempt to decrease blood flow provided by ECMO even though there was provision in the medical orders for the flow to be reduced. If there had been a medical order specifying an upper limit of blood pressure the flow may have been reduced. The protocol, inadequate as it was, was not in any case followed.

Thus hypertension occurred because there was inadequate anti-hypertensive therapy and because the protocol for ECMO parameters contained no upper limit for blood pressure and the flows were not reduced when it was permitted to do so (*vide infra*).

12. Did the combination of excessive anti-coagulation and hypertension cause the cerebral haemorrhage?

Probably, yes. The risk of haemorrhage with the anticoagulation required for ECMO even when managed well is not inconsiderable. A figure of 10% was said by one of the PICU consultants to have been quoted to [Miss A's] parents before ECMO was instituted. Actually the risk is probably up to 30% in all degrees of severity, assuming that the anticoagulation is managed appropriately. However, in [Miss A's] case the anticoagulation was excessive for a short period of time and that unfortunately coincided with the hypertension which together would have increased the risk of spontaneous haemorrhage and more likely than not is the reason why cerebral haemorrhage occurred.

13. Why didn't the medical staff consider [Miss A] as a possible organ donor when they had diagnosed brain death?

Unknown. It was indeed a very tragic circumstance that [Miss A] suffered a severe brain haemorrhage as a complication of anti-coagulation and hypertension during ECMO. In

other words, this complication was an unfortunate iatrogenic consequence of treatment. It would have been very difficult for PICU staff to discuss organ donation with [Miss A's] parents, and they were apparently unable to do so. One can only speculate why they did not discuss organ donation as a duty but it was quite evident that [Miss A's] parents were devastated by events and that the staff may have thought that [Miss A's] parents may not have been able to sustain additional stress. That said, it is sometimes beneficial for the parents of a stricken child to agree to organ donation or at least to have broached the subject. If proceeding to organ donation, the parents may feel that at least some benefit to others may have occurred. After all, two senior members of the PICU performed the tests for brain death which they confirmed, and they documented (in part) their findings on a form used as an aid for diagnosing brain death before organ donation is undertaken. That said, the performance of such tests on a patient in whom sedative effects of medication could not be excluded does not conform strictly to [ADHB's] guideline for diagnosing brain death. However, it would have been better for the intensivists to state why organ donation, and there may have been good reasons, why this matter was not pursued. Of course, the intensivists may have performed the brain function tests (flawed as stated) with the intention of verifying that continuation of treatment was futile and that withdrawal of therapy was in any case proper.

13. Are there any matters related to the documentation of death?

There are minor issues but they have no bearing on the causation of [Miss A's] death. One of the two intensivists who diagnosed 'brain death' did so despite acknowledgement on the 'Brain Death Assessment' form (page 442) that the effects of sedative and neuromuscular blocking drugs could not be excluded, while the other intensivist diagnosed brain death without supplying any documentation and stated on the 'Record of Death' (page 446) and on the 'Medical Certificate of Cause of Death' (page 448) that brain stem herniation had occurred. While brain stem herniation may well have occurred by the declared date of death (10 [Month2]), tonsillar herniation was reported as not present on the brain CT scan performed on 8 [Month2]. There is no doubt however that it was appropriate to withdraw care and allow cessation of cardiac function but to diagnose brain death without excluding effects of sedatives and neuromuscular blocking medication and to state that brain death occurred without adequate documentation is suggestive of a degree of laxity.

It is puzzling why [Miss A's] death was not regarded as necessitating report to the Coroner. Her death was, *prima facie*, a death as a consequence of prolonged inadequate medical treatment. It is uncertain what information was provided to be accepted by the Coroner that reporting and an enquiry were deemed unnecessary. Death is not usually expected from Mycoplasma pneumonia.

14. Does the reliance upon protocols/guidelines ensure delivery of an acceptable standard of care?

No, protocols and guidelines and the like do not necessarily guarantee delivery of appropriate care. They cannot apply to every clinical circumstance and do not replace sensible clinical judgment. While adherence to a guideline is not sufficient defence

against accusations of inadequate treatment, non-adherence is equally not proof of inadequate treatment.

In [Miss A's] case, there are claims that management was 'according to protocol/ guidelines'. It can be stated that either those protocols/guidelines were not followed or if they were they did not benefit [Miss A].

1. The guideline concerning management of pneumonia created by clinicians of [ADHB] was not followed. It clearly states that *Mycoplasma pneumoniae* and various viruses (<15%) including Influenza B are possible causes of pneumonia in her age group (6–18 years) along with *Streptococcus pneumoniae* (up to 30%) and *Chlamydia pneumoniae* as the predominant causes. Treatment with Erythromycin or Roxithromycin is recommended if Mycoplasma is the suspected cause. Although differentiation between bacteria, Mycoplasma and viral causes is difficult, no effort was made to accomplish such differentiation in the Emergency department (or anywhere else until it was too late). The guideline also states that a nasopharyngeal aspirate may be useful for diagnosing viral pneumonia and indicating who may benefit from treatment with Oseltamavir (sic) but the advice is restricted to children less than 2 years of age. This is erroneous.

2. The protocol for anticoagulation therapy during ECMO is not provided. However, if indeed it was followed it did not prevent excessive anti-coagulation and exposed [Miss A] to a greater risk of haemorrhage than with strictly controlled anticoagulation.

3. The protocol for prescription of haemodynamic targets for V-V ECMO was not complete on 7 [Month2] and was insufficient on 8 [Month2] and absent for the following three days. Arguably, an upper limit of blood pressure should be specified in addition to a lower limit. As it was, the parameters within to vary blood flow were not followed thus contributing to severe hypertension.

CONCLUSIONS

Unfortunately, there were many lacunae in the medical management of [Miss A] ranging from history-taking to investigations, chasing up and reporting results, prescribing and applying therapy, and avoiding complications.

The ultimate cause of death, cerebral haemorrhage, was a complication of anticoagulation therapy and inadequately managed hypertension. However, the fundamental error in medical management, across the whole institution, was the failure to consider Mycoplasma and viruses earlier as possible causes of community acquired pneumonia. Had it not been for this failure, to treat Mycoplasma especially, [Miss A] would probably have survived. It is not possible to disagree with the apparent claim by [Miss A's] parents and paraphrased by the … of the Auckland District Health Board: 'If she had received earlier treatment for Mycoplasma, she would not have died'. In other words, failure to diagnose and treat Mycoplasma pneumonia was the direct and ultimate root cause of [Miss A's] death. Numerous services at [ADHB] were in position to consider the aetiological diagnosis of pneumonia, but failing to do so in time provided care below a reasonable and acceptable standard.

SUGGESTIONS for IMPROVEMENTS

From the discussion above, it may be obvious that many issues could be addressed. The following are several areas of suggestions:

1. Consideration of the diagnosis and treatment of community acquired pneumonia, especially when the patient is not responding to first-line treatment.
2. Performance of testing for viruses and Mycoplasma.
3. Importance of rapid reporting and chasing results.
4. Documentation of surgical and medical procedures and consultations. (Obviously a patient is entitled to know what treatment was given, or not given.)
5. Consideration of the indications for the use of non-invasive respiratory support and invasive ventilation in severe pneumonia.
6. Revision of the haemodynamic protocol for V-V ECMO including an upper limit of blood pressure and examination of the anticoagulation protocol. Enforcement of the PEWS protocol.
7. Requirements for the diagnosis of brain death.
8. Need to consider organ donation in brain dead patients.
9. Reporting of deaths to the Coroner.

Yours sincerely,

Associate Professor James Tibballs"

The following further advice was received on 10 January 2021:

"10/1/2021

…

I reply to criticisms of my report of 2/8/2020 lodged by [Dr J] on 5th November 2020.

Sight should not be lost of the following fundamental irrefutable facts:

1. [Miss A] had Mycoplasma +/- Influenza infection
2. Neither of those diseases were treated until too late
3. [Miss A] had been anticoagulated for ECMO
4. [Miss A] had inadequately treated hypertension
5. [Miss A] sustained a fatal intracranial haemorrhage which was associated with anti-coagulation for ECMO and inadequately treated hypertension.

Below are specific replies to [Dr J's] multiple comments and criticisms. I have introduced different numeration.

1. **[Dr J's] Comment**. 'This response is based on review of the independent reports and the clinical record by a number of clinicians who were not directly or primarily involved

in [Miss A's] care. This includes … (Emergency Department), [Dr F] ([General Paediatrics]), [Dr E] (Paediatric Infectious Diseases), [Dr G] (PICU), [Dr J] ([PICU]).'

My Reply. The claim that there were independent impartial reports of [Miss A's] management is untenable. At least four of this group were from departments involved in [Miss A's] (mis)management. Moreover, the review did not address any of the events in PICU which led to [Miss A's] death.

2. **[Dr J's] Comment:** 'We acknowledge that there was a delay in the diagnosis of the non-bacterial causes of [Miss A's] pneumonia, including both mycoplasma and influenza. Most guidelines would suggest that tests for these causes should occur when pneumonia is severe and/or progressive despite standard antibiotic treatment. Our hospital clinical guidelines were not clear about this and have subsequently been amended. We sincerely apologise for this omission in her care.'

My Reply: [Dr J] acknowledges that there was delay in diagnosis. In fact the diagnosis was far too late.

3. **[Dr J's] comment:** 'We accept that there was a failure to escalate a degree of clinical deterioration on 27 [Month1]. Had this occurred [Miss A] may not have been transferred to [DHB2]. While this would have avoided a transfer to and from [DHB2], it did not have any impact on [Miss A's] outcome. She was at most moderately unwell on return from [DHB2] and the transfer itself would not have impacted on her clinical course. It is highly speculative for Professor Tibballs to suggest that, had [Miss A] stayed at [ADHB], the team may have been prompted to manage her differently.'

My Reply. It was not at all speculative but a view shared by a senior clinician within [ADHB] who considered similarly. In the 'Meeting Summary — [Miss A] family Meeting' (…) [Dr F] ([General Paediatrics]) acknowledged that '… the timing of her transfer is unfortunate and if her deterioration had occurred earlier in the day, transfer to [DHB2] is unlikely to have been done.'

4. **[Dr J's] comment**. 'We strongly disagree with Professor Tibballs' assertion that, had [Miss A] been treated earlier with a macrolide antibiotic for her mycoplasma, she would have survived. There is considerable uncertainty as to whether treatment with macrolides for mycoplasma improves outcome at all and even less evidence regarding increased survival with severe infection. With respect to treatment of mycoplasma, conclusions in (i) the 2015 Cochrane review (Gardner SJ, Gavrench JB, Chang AB), (ii) a 2014 systematic review of over 4000 patients (Treatment of mycoplasma pneumonia: a systematic review; Biondi E et al; Paediatrics, 2014), and (iii) the most recent version of UpToDate, all consistently conclude that 'there is insufficient evidence to draw any specific conclusions about the efficacy of antibiotics for this condition (although one trial suggests macrolides may be efficacious in some children with lower respiratory infection secondary [LRTI] to mycoplasma).'

My reply. I agree that administration of even the appropriate antibiotic does not guarantee survival from serious infection, and that Mycoplasma pneumonia is not very

susceptible to appropriate treatment. However that does not mean that no appropriate antibiotic at all should be given and that survival is not possible. In [Miss A's] case, the failure to give an appropriate antibiotic deprived her of even a chance of survival.

5. **[Dr J's] comment**. 'The use of antibiotics has to be balanced with possible adverse events.'

My reply. I agree, but there was no weighing of the benefits with adverse events in [Miss A's] case. [Miss A] had severe pneumonia and an appropriate antibiotic was not given until far too late. The weight of possible adverse effects of antibiotic against a high risk of dying from pneumonia is negligible.

6. **[Dr J's] comment**. 'There is still a need for high quality double-blinded RCTs to assess the efficacy and safety of antibiotics for LRTI secondary to M. pneumoniae in children'.

My reply. I agree but that does not mean that no treatment at all should be given when strongly indicated.

7. **[Dr J's] comment**. 'Treatment is certainly recommended because of the possibility of some benefit but this is very different from suggesting that earlier treatment would have prevented [Miss A] from dying.'

My reply. [Dr J] concedes that treatment should have been given. Contrary to [Dr J's] claim, earlier treatment may well have prevented her death (absent the mishap with inadequate treatment of hypertension — *vide infra*). In the end, PICU staff administered an appropriate antibiotic against Mycoplasma, meaning they must have considered it worthwhile, thus defeating all [Dr J's] protests that treatment was not likely to be effective in preventing [Miss A's] death. (Why give treatment if thought to be totally ineffective?)

8. **[Dr J's] comment.** 'We strongly disagree with Professor Tibballs' assertion that treatment with oseltamivir would have improved [Miss A's] outcome. Oseltamivir for influenza has only very weak evidence for a clinical benefit. This evidence shows that the duration of symptoms may be reduced by half to one day over a typical week long illness. However potential treatment benefit is mainly seen when treatment is started within the first 48 hours of symptoms, which would not have been either possible or indicated in [Miss A's] case.'

My reply. I agree that Oseltamivir does not have an outstanding therapeutic benefit but it is not negligible. This treatment would indeed have been possible — if staff had tested for influenza on [Miss A's] presentation rather than blithely assume that the cause of her pneumonia could only have been bacterial.

8. **[Dr J's] comment.** 'We strongly disagree with many of the criticisms of care made by Professor Tibballs. Professor Tibballs is a Paediatric Intensivist who has considerable expertise in medical emergency teams, early warning scores and resuscitation. However we understand that Professor Tibballs has never worked as part of the ECMO team at

Royal Children's Hospital in Melbourne managing ECMO support in patients. He does not therefore have sufficient current experience of this highly specialised aspect of intensive care treatment to assess the adequacy of the ECMO care provided to [Miss A]. He also has no expertise in paediatric emergency medicine or general paediatrics. Many of his comments reflect lack of expertise in all of these areas and a good number of them are inconsistent with standard international evidence based guidelines (including at the Royal Children's Hospital [RCH], Melbourne where he works and whose guidelines are widely used around the world). Some of Professor Tibballs' comments also do not reflect contemporary clinical practice in Paediatric Intensive Care.'

My Reply. [Dr J's] understanding that I have never worked as part of the ECMO team at the Royal Children's Hospital (RCH) in Melbourne is erroneous. I have a long history associated with the development of ECMO at RCH and considerable experience in its management. I was Acting Director of the Intensive Care Unit when the ECMO programme was commenced by a colleague at RCH in 1988. From that time until 2015 over a period of 27 years I managed hundreds of infants and children on V-A ECMO, V-V ECMO and VAD (ventricular assist devices) in a team with cardiac surgeons, junior intensive care medical staff, perfusionists, nurses and technologists. I contributed to the development of protocols for the institution and running of ECMO. I continue to work in a unit where ECMO is commonly used and is discussed daily. I am commonly in a situation where I am required to discuss the commencement of ECMO with a child's parents and to inform them of the risks and benefits of this intervention, including the incidence of cerebral haemorrhage and the measures to be taken to minimise its risk.

From this experience and from my training I can fairly say that I have expertise in the management of ECMO and this permits me to evaluate the standard of the management of ECMO provided to [Miss A].

It behoves me to point out that it does not take the World's foremost expert in ECMO to know that it is of extreme importance to control the anticoagulation required for ECMO and to prevent hypertension which together with anticoagulation (of any degree) for ECMO may be foreseen as a potent cause of cerebral haemorrhage.

9. **[Dr J's] comment.** 'We would respectfully ask that the Commissioner consider seeking further advice from specialists in Emergency Medicine and General Paediatrics and a Paediatric Intensivist who is familiar with contemporary clinical practice in ECMO. We would be happy to suggest a number of suitable clinicians in Australia to choose from.'

My reply. If [Dr J] is suggesting that a review of clinical practices in [ADHB] be instigated, in particular of the management of ECMO in PICU, I suggest that the Commissioner seeks advice from units not associated with [ADHB] rather than rely upon [Dr J's] recommendations, in order to gain independent advice.

I am not questioning the general competency of PICU at [ADHB] to manage severe pneumonia and to run ECMO. Principally, I am rather drawing attention to one instance comprising two major deficiencies in medical practice at [ADHB]: (1) to diagnose and to

manage community acquired severe pneumonia across the whole organisation, including PICU; (2) to manage ECMO in PICU in terms of minimising the risk factor of hypertension for cerebral haemorrhage.

10. **[Dr J's] comment.** 'Professor Tibballs' opinion is notable for a high degree of hindsight bias and a level of certainty which is not justified and does not reflect the uncertainties and ambiguities of clinical practice that are faced by clinicians undertaking their work every day in a complex system.'

My reply. I acknowledge that the tasks involved in managing a seriously ill child are complex and that uncertainties and ambiguities of clinical practice exist. However, my original report focused mainly on fundamental errors — errors which even [Miss A's] parents, as non-medical witnesses, perceived. There is no escaping the realities that no-one considered non-bacterial causes of [Miss A's] pneumonia until it was too late, that the medical history lacked important documentation, that the management of artificial respiratory support was highly questionable and that the management of ECMO was problematic.

It is obvious that my opinion is a retrospective one, but it is also the nature of the opinion of every person involved in this case, including [Dr J's]. From a contemporary viewpoint, it would have been prudent to reconsider the cause of [Miss A's] pneumonia well before the diagnosis was belatedly made, and to better control hypertension.

11. **[Dr J's] comment.** 'We agree that there are instances where [Miss A] was reviewed by various medical staff without adequate documentation of those reviews being made.'

My reply. Consultations **must** be documented.

12. **[Dr J's] comment.** 'Further investigating the nature of [Miss A's] [sibling's] pneumonia would not have changed the initial decision making for [Miss A].'

My reply. Merely knowing (by taking a sufficient medical history) that a sibling had recently contracted pneumonia may have prompted clinicians to consider non-bacterial causes. Taking an adequate medical history is a fundamental medical skill, and that especially includes illnesses in the siblings of a child.

13. **[Dr J's] comment.** 'Mycoplasma does cause infection in families and communities. The same is equally true of respiratory viral infections and these are the most common cause of respiratory illness in children and spread very easily. Because it is not possible to clinically determine the cause of pneumonia, a more detailed history of the nature of pneumonia in [Miss A's] [sibling] would not have helped to define the likely cause of [Miss A's] illness.'

My reply. These comments are contradictory to [Dr J's] claims that it was not indicated to test for non-bacterial causes of pneumonia. It is indeed possible to determine the non-bacterial causes of pneumonia — if the appropriate clinical tests are performed,

but in [Miss A's] case they weren't. It would have been helpful to have known that [Miss A's] [sibling] had had pneumonia and as [Dr J] admits, Mycoplasma does indeed cause infection in families.

14. **[Dr J's] comment:** 'It is incorrect to say that by prescribing amoxicillin it is evident that the doctor believed that [Miss A] had bacterial pneumonia, and no other causative organism was considered. A doctor managing community acquired pneumonia (CAP) in a child will normally consider the range of possible causes.'

My reply. Yes, a doctor would **normally** consider a range of causes but there is no evidence that this was so in [Miss A's] case anywhere in the medical record until finally when it was too late. Why else would a doctor prescribe an antibiotic active against bacterial pneumonia if it was thought that the infecting organism was not bacterial? This is an example of illogical analysis.

15. **[Dr J's] comment.** 'Viral pneumonias do not respond to antibiotics but cannot be distinguished from bacterial pneumonia on history, examination or radiographic imaging findings. In the face of this uncertainty, it is prudent to prescribe oral antibiotics while acknowledging that, for a large proportion of cases, the antibiotics have no benefit. Oral amoxicillin is the recommended treatment in most international guidelines (including the British Thoracic Society and the Infectious Diseases Society of America) and the guidelines for [ADHB] and RCH for all children with mild to moderate pneumonia, whether treated as an outpatient or admitted to hospital. This was not an error and follows recommended best practice.'

My Reply: I have no problem with the prescription of amoxicillin, in case the causative pathogen is bacterial. However, no other pathogens were ever considered until it was too late and when it was plainly obvious that [Miss A's] condition was not responding to antibiotics. It is not true that guidelines of various hospitals including [ADHB] and RCH, do not consider causes other than bacteria.

16. **[Dr J's] comment.** 'The criticism that tests should have been done in the Emergency Department to identify the infecting organism is both incorrect and opposed to both local and international guidelines. No tests were performed because none were indicated. This follows recommended best practice. As stated in the RCH guidelines 'investigations including chest x-ray, are not recommended routinely for CAP particularly in those with mild disease who are expected to be managed as an outpatient'. In reference to atypical pneumonia, it is also stated that 'testing for atypical pneumonia (including mycoplasma) rarely influences management, as it does not differentiate infection from asymptomatic carriage. There is no proven benefit from treatment with macrolides alone or in combination with β-lactams in children with suspected or confirmed atypical pneumonia. The only exception in practice is in cases of severe pneumonia — azithromycin may be considered.'

My reply. If a medical history had been taken that a sibling had recently had pneumonia, it may have prompted clinical staff to consider non-bacterial causes. Since the right questions were not asked, ignorance prevailed and no-one considered

alternative causes and treatment. Even when it was evident that [Miss A] had severe pneumonia, azithromycin was still not considered.

17. **[Dr J's] comment.** 'Most international guidelines recommend against investigations because they do not influence management or treatment choices for mild to moderate pneumonia. ADHB's internal review stated that no test was available which would provide a result within the relevant time frame for decision making in the Emergency Department.'

My reply. That's not quite true. If a follow-up had been organised the results would probably have been available. If the fact that a sibling had had pneumonia was given attention, testing for non-bacterial causes may have been performed.

18. **[Dr J's] comment.** 'We did not suggest that no test was available because clearly tests are available.'

My reply. This is a non-sequitur, and contradicting his claim in comment 16.

19. **[Dr J's] comment.** 'However they are not available within the time frame required for Emergency Department treatment.'

My reply. That **may** be so, but could be helpful on re-presentation or in retrospect. If follow up had been organised (which it wasn't) the results would have been available.

20. **[Dr J's] comment**. '… but more importantly such tests are not recommended because they do not alter clinical management'

My reply. Is [Dr J] saying that Mycoplasma should not ever be tested for and not ever treated? If so, that is plainly erroneous. Eventually (too late) it was tested for and delayed appropriate treatment (too late) was given.

21. **[Dr J's] comment**. 'We agree that a chest x-ray is not always required to make a diagnosis of CAP in a child who is well enough to be managed at home. A chest x-ray would usually be performed for a child if their condition is severe enough to require admission to hospital. We can assume that discussion occurred around the helpfulness or otherwise of a chest x-ray at the time of [Miss A's] first presentation since [Miss A's] mother raised this.'

My reply. That is an unjustified assumption based on no documentation.

22. **[Dr J's] comment**. 'The decision not to perform an x-ray does not mean that any concerns raised by [Miss A's] mother were dismissed or not taken into account when making this decision.'

My reply. They must have been dismissed because it did not happen.

23. **[Dr J's] comment.** 'The lack of testing (including chest X-ray) raised by Professor Tibballs reflects an incorrect view that these are standard practice and would have led

to treatment with a macrolide at this point. However, as mentioned, tests and treatment with a macrolide are not recommended for children being managed as an outpatient with CAP in any international guidelines. This is because the test cannot distinguish asymptomatic carriage in a case of mycoplasma and because there is no evidence that the treatment with macrolides improves outcome, especially in those children with mild to moderate pneumonia where the condition is self-limiting.

My reply. This reasoning contains more instances of *non sequitur*. We are not considering asymptomatic carriage with a self-limiting condition but real pneumonia which led to death. In spite of the claim, there is some evidence that macrolides improves outcome, but [Miss A] was never given that chance. I agree that a chest X-ray cannot distinguish causes of pneumonia but it can indeed diagnose pneumonia.

24. **[Dr J's] comment**. 'It is also incorrect to state that it is an error that no follow up was organised in [Miss A's] case. Most children with CAP discharged on oral antibiotic from the emergency department recover uneventfully. In a small proportion of cases a child's condition may become worse regardless of the presumptive causative organism. Discharge information is copied to the child's GP electronically at the time of discharge with a paper copy provided to their parents. It is policy and practice to give clear return advice to parents of children discharged from the Emergency Department with any acute illness. In [Miss A's] case this advice was given and her parents very appropriately returned to [ADHB] when they were unhappy with her progress. [Miss A's] management and discharge advice was standard good practice. It is also not clear how other follow up would have changed the course of events.'

My reply. I could not find proof that follow-up had been arranged. It was patently an error not to arrange follow-up. If follow-up had been arranged in the context of community acquired pneumonia with a sibling similarly affected and tests conducted searching for the cause, it is likely that the outcome would have been very different.

25. **[Dr J's] comment**. 'We do not agree that alternative non-bacterial causes of pneumonia were not considered. The change from oral amoxicillin to intravenous cefuroxime was appropriate and in line with [ADHB] and other international guidelines. No other testing was indicated at that time as [Miss A] quite clearly had mild to moderate uncomplicated pneumonia.'

My reply. A change from amoxicillin to cefuroxime is still in line with the rigid thought process that the cause was a bacterial one.

26. **[Dr J's] comment**. 'We agree that deterioration on the ward on 27 [Month1] was not conveyed to medical staff and it should have been. With the benefit of hindsight, if this had been escalated [Miss A] may well not have been transferred to [DHB2].'

My reply. I agree. She deteriorated just before transfer from [ADHB] but this was not reported to medical staff. [Dr J's] statement contradicts that in comment 27.

27. **[Dr J's] comment**. 'However she was transferred to another appropriate inpatient facility where her deterioration was appropriately detected and acted upon. The time to surgical intervention was not compromised by the transfer to [DHB2] and back. No other changes in her management would have occurred if she had remained at [ADHB].'

My reply. That's speculative. If she had remained and continued to worsen while remaining under surveillance of [ADHB] staff, they may have been prompted to consider causes of pneumonia other than bacterial.

28. **[Dr J's] comment.** 'The facility she was transferred to is very experienced at managing moderate severity pneumonia.'

My reply. That is not questioned, and indeed staff at that institution recognised that [Miss A] was not responding to treatment but may have assumed that [ADHB] would determine why that was so.

29. **[Dr J's] comment.** 'There are multiple comments about missed opportunities to diagnosis mycoplasma and viral pneumonia. We agree that when [Miss A] returned to [ADHB] she had pneumonia that was not responding to treatment and was becoming more severe.'

My reply. Was it not then appropriate to consider causes of pneumonia other than bacterial?

30. **[Dr J's] comment.** 'We accept that testing for viral and atypical causes should have occurred around 1–2 [Month2] when her pneumonia was becoming more severe.'

My reply. This is contradictory to his claims that such testing was not warranted.

31. **[Dr J's] comment.** 'The PCR testing on the pleural fluid taken on the 29 [Month1] was requested when [Miss A] was being reviewed by an Infectious Diseases specialist on 7 [Month2]. That is, it was requested retrospectively from a sample taken nine days earlier. The pleural fluid sample was initially tested for bacteria and underwent microscopy for cell counts. The mycoplasma PCR test was not ordered at the time the pleural fluid was taken. There was therefore no failure to check results. The ID service documented reviews on 2nd, 3rd, 5th and 8th [Month2]. We agree that there should have been earlier ID consultation and also that there were multiple verbal discussions with Infectious Diseases clinicians that were not documented in the clinical record. The ID service reviews all patients with infections in PICU daily but documentation of these reviews did not always occur.'

My reply. A patent contradiction exists between: '… there should have been earlier ID consultation' and 'The ID service reviews all patients with infections daily …'

From the viewpoint of a third party examining the record, it may be fairly assumed that if something was not documented, it did not happen.

If a respiratory service exists at [ADHB], it should have been consulted early in [Miss A's] admission to the PICU.

32. [Dr J's] comment: 'The retrospective request to add an atypical panel to the pleural fluid is documented by the PICU consultant in the clinical notes on the 11 [Month2] when cold agglutinins were detected.'

My reply. Cold agglutinins were first detected on 2 [Month2]. If detection of cold agglutinins was the trigger for PICU staff to consider Mycoplasma infection, it should have been considered on 2 [Month2].

PICU Management

Non Invasive ventilation

33. **[Dr J's] comment**. 'Professor Tibballs' comments are not correct and do not reflect current intensive care practice. Both high flow nasal prong (HFNP) oxygen and invasive ventilation have advantages and disadvantages. It is incorrect to say that HFNP is not appropriate in severe pneumonia. The use of HFNP has expanded considerably in recent years and is often used in severe pneumonia. It has major advantages over invasive ventilation which include improve[d] airway clearance if the patient has a strong cough, minimizing sedation, improved ventilation/perfusion matching, avoidance of positive intrathoracic pressure and the avoidance of ventilator induced lung injury. Invasive ventilation also has specific advantages including reduction of work of breathing (preventing patient exhaustion), secretion clearance and also to facilitate taking of samples for analysis from the lungs. The timing of institution of invasive ventilation requires clinical judgement and is typically based around balancing the risk and benefits. It is a dynamic decision based on frequent observation and review of the patient's physiologic parameters in real time and cannot be reliably judged from reading clinical notes. The use of HFNP was appropriate and we believe escalation to invasive ventilation also occurred at an appropriate time.'

My reply. I agree that whenever possible it is better to avoid invasive ventilation for all the reasons stated by [Dr J], and agree that the timing of institution of invasive ventilation requires sound clinical judgement. However, sound judgement was not evident and I cannot agree that the decision making process cannot be reliably judged from the clinical notes. If that is the case, ie 'cannot be judged from the clinical notes', the clinical notes are inadequate.

Consideration of other causes of [Miss A's] pneumonia (also comment 3)

34. **[Dr J's] comment**. 'We agree that a nasopharyngeal aspirate looking for viral and atypical causes of pneumonia should have been done once [Miss A's] pneumonia became severe and complicated. This was around 1–2 [Month2]. A tracheal aspirate and bronchoalveolar lavage could not have been done until she was intubated which occurred on 4 [Month2].'

My reply. The decision to intubate ought to have been taken earlier than it was, especially since the actual cause of the pneumonia was unknown and [Miss A] was deteriorating. It was important to establish the cause of the pneumonia. Earlier intubation would have enabled earlier testing for bronchoalveolar lavage specimens to be examined.

Pursuing results of test

35. **[Dr J's] comment**. 'As noted above, the mycoplasma PCR on the pleural fluid collected on 28 [Month1] was asked for retrospectively by the ID consultant reviewing [Miss A] on 7 [Month2]. This test was therefore neither performed nor available until after 7 [Month2]. All other tests were followed up in a timely way.'

My reply. It ought to have been requested on 28 [Month1], as suggested below by [Dr J] (comment 36).

Consideration of Mycoplasma earlier

36. **[Dr J's] comment**. 'We agree that alternative causes of [Miss A's] pneumonia should have been considered earlier. However we do not agree that the circumstantial evidence, as quoted by Professor Tibballs, strongly suggests that [Miss A] could be suffering from mycoplasma pneumonia.'

My Reply. The infectious diseases specialist (eventually) thought the same — the circumstances of the infection with pneumonia in a child and occurring in a sibling are indeed suggestive of mycoplasma infection.

37. **[Dr J's] comment**. 'We suggest that Professor Tibballs' characterisation of this "circumstantial evidence" further demonstrates that his opinion is significantly impacted by hindsight bias. There are a number of more likely explanations for these matters, namely:

i. Sibling contact and spread could more likely have been ascribed to respiratory viral infection'

My reply. I agree, viral infection was more likely but Mycoplasma was not unlikely.

ii. Lack of response to anti-bacterial treatment occurs with viral pneumonias and also bacterial and necrotizing pneumonias

My reply. I agree but that category also includes Mycoplasma.

iii. A rash also occurs with viral infection and can be related to antibiotics

My reply. I agree.

iv. Cold agglutinins occurred from 2 [Month2] at which point we agree that mycoplasma testing should have been done. This conflicts with [Dr J's] comment 32 implying that the detection of cold agglutinins on 11 [Month2] was the trigger factor to test for

mycoplasma. Why wasn't the detection of cold agglutinins on 2 [Month2] the trigger factor?

My reply. This statement by [Dr J] is a tantamount admission that testing was unduly delayed.

v. Absence of bacteria on microscopy of pleural fluid is not indicative of other causes. Culture-negative empyema, particularly with prior antibiotic is typically found in up to 50% of children with bacterial pneumonia.

My reply. I agree that bacteria are not always identifiable in pleural fluid even when the cause of the pneumonia is bacterial. However that is not the only explanation — the cause may be from an organism not detectable with the tests conducted.

vi. Absence of bacteria in blood cultures occurs in the majority of cases of bacterial pneumonia.

My reply. I agree that blood cultures may not be positive in cases of bacterial pneumonia, but that result should not exclude consideration of causes other than bacterial.

Lack of consideration of viral causes

38. **[Dr J's] comment**. 'It is not correct that viral causes for [Miss A's] condition were not considered by the [ADHB] clinicians. In fact it was assumed that this was likely and/or that there may have been a bacterial infection in addition to a viral infection. Viral causes or secondary bacterial infection on top of a viral infection are all common. Viral and bacterial causes are far more likely than mycoplasma to cause severe necrotising pneumonia, which [Miss A] developed. Testing for viruses does not influence management as there are no treatments for this apart from oseltamivir early in the course of influenza. Any possible minor benefit of oseltamivir is described when started early (within the first 2–3 days). At the time of [Miss A] becoming severely unwell, when testing was appropriate, she was more than 10 days into her illness.'

My reply. There is no documentation at all that other causes were considered until the infectious disease consultant belatedly suggested testing for Mycoplasma.

Earlier consultation of the ID team

39. **[Dr J's] comment.** 'The ID team were consulted on 2 [Month2] and saw [Miss A] on every day after that. We agree that it would have been better if they had been consulted earlier and also that all of their reviews should have documented. The ID team review all patients in PICU every day and this occurred with [Miss A]. However documentations of these reviews in the clinical record did not always occur.'

My reply. Again, if a consultation was not documented, a third party examining the medical record may fairly assume it did not happen.

Why weren't less injurious forms of mechanical ventilation considered?

40. **[Dr J's] comment.** 'Professor Tibballs' comments regarding ventilation are incorrect and do not reflect current intensive care practice. The most appropriate and least injurious form of ventilation was used. The initial plan was to facilitate [Miss A] breathing by herself as soon as possible as this would help limit any injury caused by the ventilator. However due to the nature and progression of her disease this was not possible, so the aim of ventilation was to use the lowest possible pressures. This included using airway pressures as low as possible to limit lung damage and tolerating high levels of carbon dioxide. There is no evidence that HFOV is less injurious to the lung than IPPV. Several relatively recent large randomized control trials in adults have shown either no benefit to HFOV or that it increases mortality. Cohort studies in children have also shown that it potentially increases mortality. As a result HFOV has become much less commonly used in recent years and many ICUs do not use it at all or only very rarely and for very specific indications.'

My reply. I agree that it is not entirely clear which form of invasive ventilation is superior in terms of limiting lung damage. All forms of positive pressure artificial ventilation have the potential to injure the lungs. Even so-called 'non-invasive ventilation' and high flow intranasal flow oxygen can cause lung damage (as occurred in [Miss A]) and other complications. It is debatable whether conventional mechanical ventilation (CMV) or high frequency oscillatory ventilation (HFOV) is less injurious. However, when lung disease is severe and gas exchange is precarious it is worth at least trialling HFOV in lieu of CMV to determine if less positive pressure can achieve the same or better gas exchange. No such trial was conducted. In spite of [Dr J's] claim, HFOV remains a valuable tool in the treatment of severe lung disease and is used around the World. Over 200 scientific articles are to be found in the medical literature concerning HFOV in groups of children with severe lung disease. The majority are in favour of using HFOV compared with CMV, while a few are contrary. However, in an individual patient, only a trial of such therapy can indicate which is better.

Documentation and possible failure to recognise V-A as compared to V-V ECMO

41. **[Dr J's] comment.** 'The mode of support used for [Miss A] was V-V ECMO and there is no doubt about this. All of the team caring for [Miss A] knew what mode of ECMO was being used and how to manage both the circuit and the patient. There was a simple error made by the perfusionist in documenting where the cannula was placed. This should have been recognised and corrected. However it was not recorded in a place which is routinely accessed and so was not discovered at the time she was receiving care.'

My reply. As I said in my original report, on balance it was evident that V-V ECMO was being used, but that doesn't excuse errors in documentation.

42. **[Dr J's] comment**. 'Professor Tibballs writes at some length about the differences between V-V and V-A ECMO, whether there could have been any confusion as to which mode was being used and any possible consequences if these assumptions were incorrect. Professor Tibballs' comments reflect a theoretical knowledge of ECMO and

not those of someone with practical experience of managing patients on ECMO. The team were all aware that she was receiving V-V ECMO, she was managed appropriately on V-V ECMO and we all agree that the management of this is very different than V-A ECMO.'

My reply. I acknowledged that in my original report that I thought there had been an error in documentation of the location of the femoral cannula.

[Dr J's] claims that my knowledge of ECMO is (merely) theoretical and he believes that I have not managed ECMO. As above, (reply to comment 8) that belief is a misunderstanding on his behalf.

I disagree that management of the ECMO (V-V) was appropriate. The staff permitted severe prolonged hypertension to go unchecked while there was a state, albeit relatively brief, of excessive anticoagulation. The two conditions: a state of anticoagulation even when appropriately controlled together with hypertension are a dangerous combination for cerebral haemorrhage.

Excessive anticoagulation on ECMO

43. **[Dr J's] comment**. 'Again we strongly disagree with Professor Tibballs' assertion that anticoagulation on ECMO was excessive and his comments about this issue. Anticoagulation was managed by our protocol which is evidence-based and similar to that used in other ECMO centres in Australasia. Activated clotting time (ACT) levels often fluctuate while on ECMO and the heparin infusion is adjusted accordingly. This is common especially in the first day or two on ECMO, where ACT levels outside the desired range occur not uncommonly. There was no deviation from our protocol in managing [Miss A's] heparin dosing. [Miss A] was at all times receiving a comparatively low dose of heparin for a child on ECMO. Most of her ACTs and activated partial thromboplastin times (APPTs) (both measures of heparin effect but also influenced by other coagulation abnormalities) were much lower than desired. She started heparin at a dose of 10u/kg/hr at around 1700 on 7 [Month2]. When the ACTs were persistently low the dose was increased to 11 (after the 2100 sample), 12 (after the 2300 sample) and then 13.8 (after the 0100 sample). The result at 0300 was 316, which is too high and so the heparin was reduced back to 10. By 0700 the ACT was coming back in to the desired range and after that all of the samples showed results that were at the lower end or below the desired range. This was true throughout the morning of 8 [Month2] and when [Miss A] developed fixed and dilated pupils at 1600. Doses of heparin of 15–35u/kg/hr are commonly required on ECMO, averaging around 18–20u/kg/hr in this age. Many ECMO programmes never reduce the heparin dose below 10 because of the risk of blood clots forming in the ECMO circuit.'

My reply. I agree that controlling the anticoagulation is a difficult task but the fact remains it was excessive for a brief period. If it is true that the protocol was followed but it occurred nonetheless, it must imply that the protocol was imprecise despite even the best efforts to refine it. That the anticoagulation was excessive is not the main issue and does not necessarily imply a standard of care below normal.

Hypertension on ECMO

44. **[Dr J's] comment.** 'Hypertension on ECMO is common. The hypertension that [Miss A] experienced when first on ECMO was not severe and was treated appropriately with escalation of anti-hypertensive agents as required. [Miss A] was initially started on milrinone for her hypertension, with reduction in her blood pressure. When the hypertension became worse again she was started on sodium nitroprusside.'

My reply. Hypertension being common doesn't make it acceptable or untreatable. Treatment of hypertension was inadequate and delayed. As discussed in my original report, the hypertension was indeed severe and was present for at least 24 hours. Treatment was attempted but was inadequate. Milrinone is not the preferred agent to treat severe hypertension while nitroprusside, an appropriate treatment, was commenced belatedly after 18 hours of severe hypertension. The cerebral haemorrhaging probably occurred during the treatment with Milrinone. Contrary to [Dr J's] claim, treatment was not given as required. The requirement would have been to restore the blood pressure to at least normal values as quickly as possible. The statement that it was not severe is erroneous — see my original report of its extent and duration with reference to normal values.

45. **[Dr J's] comment.** 'Professor Tibballs' discussion about Ohm's equation and adjusting the ECMO flow to modify the blood pressure is totally incorrect. It would apply to a patient on V-A ECMO but not to a patient on V-V ECMO.'

My reply. It would apply if as [Dr J] confirms V-A ECMO is used, but as in my original report and I state again, I believe that [Miss A] was on V-V ECMO despite the errors in medical notes which could lead a third party to wonder whether V-A ECMO was used.

46. **[Dr J's] comment.** 'In V-V ECMO blood is drained and returned to the venous side of the circulation and so does not directly contribute to alterations in cardiac output in the way that Professor Tibballs describes. Reducing ECMO flow would have no effect on the blood pressure but would have reduced the amount of supplementary oxygenated blood being circulated to [Miss A] and would have therefore resulted in lower blood oxygen levels. This would have been both dangerous and ineffective for the purposes he described. Cardiac output does often increase on ECMO because some of the negative effects of mechanical ventilation on the circulatory system are reduced which allows improved return of blood to the heart and a higher cardiac output.'

My reply. I agree the discussion about 'Ohm's equation' pertains only to V-A ECMO. However, as discussed in my original report the surgical documentation about the location of the femoral cannula (artery) and X-ray report conflict with the clinical records. I accept that the ECMO was truly V-V ECMO; which I said in my original report. However, the question remains of why were such errors in documentation permitted to go uncorrected by PICU clinicians?

Did the combination of excessive anti-coagulation and hypertension cause the cerebral haemorrhage?

47. **[Dr J's] comments**. 'Again we strongly disagree with this assertion. Cerebral haemorrhage occurring in patients on ECMO is most commonly due to bleeding into areas of abnormal brain. Brain abnormalities are most commonly due to either ischaemia (when there has been a period of cardiac arrest or very low cardiac output) or inflammation/infection. Both mycoplasma and influenza can cause inflammation of the brain. This is well described as a cause of cerebral haemorrhage in patients on ECMO and is something we have observed in previous patients. The fact that the haemorrhages were multi-focal makes this the most likely cause. Professor Tibballs' suggestion that this was due to severe hypertension in the presence of excessive anti-coagulation is very unlikely to be the cause. If hypertension was the mechanism it would lead to rupture of an intracerebral vessel and be much more likely to lead to a single large haemorrhage rather than multi-focal haemorrhages.'

My reply. [Dr J's] reasoning is faulty. Hypertension is a well-known cause of cerebral haemorrhage in all age-groups. Severe hypertension is a medical emergency and must be treated urgently in order to prevent a catastrophic cerebral haemorrhage.

[Dr J's] supposition that the cerebral haemorrhage being due to cerebral inflammation associated with underlying mycoplasma or influenza infection is a self-defeating argument. If such infection was present predisposing to cerebral haemorrhage, it would have been all the more important to control the hypertension.

48. **[Dr J's] comment**: 'The risk of intracranial haemorrhage on ECMO is not around 30%. This reflects very old data. In current practice the internationally accepted rate is 6–8% and this is the rate in our own programme at [ADHB]. The rate is higher in patients on V-A ECMO and lower in patients on V-V ECMO. The most important factor is that the rate is considerably increased when there are any brain abnormalities.'

My reply. [Dr J] has misinterpreted comments in my original report and I am not aware of any 'very old data' as he is that the incidence of cerebral haemorrhage was at one time 30%. I did not say, as [Dr J] claims, that the risk of cerebral haemorrhage was around 30%. Careful reading of my report shows that I said the total incidence of haemorrhage was around 30%. This figure was derived from a chapter in a publication of the Extracorporeal Life Support Organisation (ELSO) 2017: Bratton SL, Workman J. 'Comorbidities among pediatric patients with respiratory failure on ECLS' In: Brogan TV, Lequier L, Lorusso R, MacLaren G, Peek, G. (eds) p255–265. This chapter stated that the risk of brain haemorrhage was 6%, cannulation site bleeding 18% and surgical site bleeding 13%. IF it is assumed that brain haemorrhage and cannulation site bleeding or cannulation site bleeding occur together, the total incidence is around 30% (as stated in my original report), thus justifying my original statement. The ELSO data is based upon haemorrhage as being defined as a fall in haemoglobin of at least 2g/dL over 24 hours or a loss of blood as more than 20mL/kg body weight over 24 hours or a transfusion requirement of more than 10 mL/kg body weight over 24 hours. This data does not include other sites of haemorrhage which if taken into account indicates that overall incidence of haemorrhage is actually higher. For example at RCH up until 2015 (data supplied by Dr … at RCH), when all degrees of haemorrhage are considered, the incidence of cerebral haemorrhage

during ECMO given for respiratory illness was 7.4%, cannulation site haemorrhage 17.9%, surgical site haemorrhage 7.4%, gastrointestinal haemorrhage 5.3% and pulmonary haemorrhage 3.2%.

Organ donation consideration

49. [Dr J's] comment. '[Miss A] had both active influenza B and mycoplasma and this may have made her unsuitable for donation of some of her organs. However we agree that this should have been discussed with the organ donation service to see whether any of [her] organs could have been used. If this was possible, discussion with the family should have occurred.'

My reply. I concur.

Matters related to the documentation of death

50. **[Dr J's] comment**. 'Many of Professor Tibballs' comments in this section are incorrect or appear to be based on a misinterpretation of the clinical notes. The clinical examination confirming brain death was documented on the "Procedures for Certification of Brain Death" form on 10 [Month2]. The intensivist has recorded "yes" to confirm that potentially reversible causes for the patient's condition have been adequately excluded, including depressant drugs and neuromuscular blocking agents. Professor Tibballs may have interpreted this documentation incorrectly because the intensivist has written over the first two columns, correcting the "no" to "yes". [Miss A] had not received any sedative or neuromuscular blocking drugs for more than 36 hours before brain death testing had occurred and therefore there is no possibility that the intensivist performing brain death testing had concluded that [Miss A] was brain dead with reversible causes still present. We agree that documentation of the brain death assessment was not adequate. However as [Miss A] had ceased all sedatives and neuromuscular blocking drugs for more than 36 hours before the testing was done and, as she had normal renal and liver function, the intensivist was appropriately certain that this was not affecting the results.'

My reply. No further comment.

51. **[Dr J's] comment.** '[Miss A's] case was discussed with the coroner, as is our usual practice. The Coroner is often guided by what the family wanted, and [Miss A's] family did not want a post-mortem to take place. All such deaths are discussed with the Coroner, but the final decision about a Coroner's post-mortem and whether to open an enquiry are made by the Coroner.'

My reply. Coroners can be guided by what is conveyed to them. There is no record of what information was conveyed. If it had been conveyed that the diagnosis (Mycoplasma pneumonia) was missed and/or that the cerebral haemorrhage was associated with uncontrolled hypertension with possible brain Mycoplasma infection, the Coroner may have decided on a different course of enquiry.

Reliance of protocols.

52. **[Dr J's] comment**. 'We agree that protocols and guidelines do not necessarily guarantee delivery of appropriate care. We agree that the [ADHB] guideline for severe pneumonia was not as clear as it could be in terms of testing for viral and atypical causes in children with severe pneumonia. The guideline has been updated and considerable effort has been put in to education about this.'

My reply. Accepted, but note that this concession mainly involves other departments in the hospital, rather than PICU primarily.

53. **[Dr J's] comment**. 'Professor Tibballs' comments about protocols for anti-coagulation on ECMO and also for prescription of haemodynamic targets on V-V ECMO are incorrect, as has been noted above.'

My reply. Although I take [Dr J's] assurance that it was in fact V-V ECMO being used, there is still no acknowledgement that hypertension was permitted to exist and went inadequately treated. No upper targets for blood pressure appear to exist in the ECMO protocol.

54. **[Dr J's] comment**. 'In terms of the nine improvements suggested by Professor Tibballs we agree with the first and fourth and these were identified in our previous internal review and report regarding [Miss A's] case. We agree that consideration of the diagnosis and treatment of severe CAP not responding to first line treatment should include testing for influenza and mycoplasma. We also agree that documentation of surgical and medical procedures and consultations could have been better in [Miss A's] case.'

My reply. It is not a matter of whether or not documentation could have been better, it was in fact substandard. A patient, their family and any representative have the right to know what treatment was given. If treatment given is not recorded, a third party may fairly conclude it was not given.

55. **[Dr J's] comment**. 'All of Professor Tibballs' other suggestions are either inconsistent with current evidence and practice or reflect an interpretation of events in this case that is factually incorrect. We have reviewed our own experience with ECMO for influenza and mycoplasma pneumonia. We have also discussed with the ECMO team at the Royal Children's Hospital in Melbourne their experience with influenza and mycoplasma pneumonia. This confirms that ECMO is rarely required, especially for mycoplasma pneumonia. For both, it is most commonly (>90% of the time) required early (within 36–48hrs of admission) for rapidly progressive fulminant disease. For influenza, it is occasionally required later, often because of a secondary bacterial infection. We believe that the most likely explanation for [Miss A's] illness was combined mycoplasma and influenza pneumonia, with the protracted course most likely due to the influenza and her late decline due to either the influenza alone or, more likely, secondary bacterial infection. It is not uncommon to fail to isolate bacteria when antibiotics are being given. It is also impossible for anyone to be certain about the precise contribution of the various infections. Unfortunately her disease progressed to the point that she needed ECMO support. She died from a cerebral haemorrhage, most

likely related to the need for anticoagulation on ECMO causing her to bleed in to areas of brain affected by encephalitis caused by either influenza, mycoplasma or both.'

My reply. I agree that the necessary anticoagulation even when strictly controlled is a high risk factor for cerebral haemorrhage. However, strict upper limits for blood pressure (hypertension) must be present and adhered to. This was not done. It is of no interest to a patient to know that their need for ECMO is 'rarely required' but it would be of great interest to know that the intensive care unit in charge of ECMO would have protocols to combat other risk factors within their capability, such as the control of hypertension, which [ADHB] did not have.

If, as [Dr J] claims, [Miss A] died from haemorrhage into the brain made susceptible by 'encephalitis caused by either influenza, mycoplasma or both' it surely would have been all the more important to control hypertension, but inadequate attention was paid to combatting this important risk factor for cerebral haemorrhage.

56. **[Dr J's] comment.** 'All of the teams involved in [Miss A's] care while she was in [ADHB] have reviewed and reflected on her care and have learnt from, or made changes to their guidelines, to improve the care of children in the future. Our previous review and Professor Tibballs' report raise important issues about testing for atypical and viral causes of severe pneumonia in patients not responding to treatment. We acknowledge this should have happened and we have improved our guidelines to assist this to occur.'

My reply. I am assured that guidelines have been modified to manage pneumonia not responding to antibiotics, but I am not at all assured by a continued refusal to accept that hypertension is an important risk factor for cerebral haemorrhage in a patient anticoagulated necessarily for ECMO. Importantly, there has been no presentation of data by [Dr J] to prove that hypertension was not present. I must assume therefore that [Dr J] accepts that hypertension was present as detailed in my previous report. This factor which probably caused the cerebral haemorrhage in association with anticoagulation has not been rebutted.

57. **[Dr J's] comment**. 'However because of the uncertainty about the efficacy of treatment improving outcome for these conditions, we do not agree that it could be said with any confidence that earlier macrolide treatment would have altered the outcome in [Miss A's] case.'

My reply. That is nonsensical. That can only be concluded if the correct treatment was applied and [Miss A] died nonetheless. There can be no chance of survival if the correct treatment was not applied at all.

58. **[Dr J's] comment**. 'Once again we extend our deepest sympathies to [Miss A's] parents and family.'

My reply. Of course, it goes without saying that sympathies are extended to the family for [Miss A's] death but the discussion should have gone a lot further. An admission of

an adverse event should have been made and an apology extended. This does not mean that this would have been an admission of liability — that would be for a Court to determine. However, failure to admit that an adverse event has occurred and failure to offer an apology are poor reflections on the culture in the unit and are potent causes for a patient's representative to seek legal compensation.

CONCLUSIONS

1. [Dr J's] commentary contains multiple contradictory statements the most significant of which are two comprise self-defeating arguments:

(a) Testing for Mycoplasma was not warranted yet should have been done earlier, and;

(b) Mycoplasma treatment was not warranted but caused [Miss A's] cerebral haemorrhage.

2. [Dr J's] commentary overall shows a lack of preparedness to acknowledge that serious adverse events occurred in PICU, which is of concern in the context of a requirement for open disclosure, apology (while not admitting liability) and action to prevent recurrence. While acknowledging that medical management of [Miss A's] condition could have been better by other departments of [ADHB], [Dr J] is unable to perceive that management could have been better in the intensive care unit related to the treatment of hypertension which was clearly inadequate and on the balance of probabilities the cause of the cerebral haemorrhage from which [Miss A] died. The inability to admit that an adverse event has occurred does not reflect well on the prevailing culture of the unit.

3. Unfortunately [Dr J] has resorted to some extent to inappropriate arguments *ad hominem* derived from erroneous information and to unreasoned denial of facts.

Yours sincerely,

James Tibballs"

Appendix F: Summary of ADHB review

ADHB review of the treatment provided to Miss A

ADHB conducted a review of the treatment provided to Miss A by ADHB in Month1 and Month2, with input from specialists from each ADHB service involved, but who were not involved directly in her clinical care. ADHB stated that the reviews were written using verbal interviews with the medical staff involved.

The review identified that the treatment Miss A received was in keeping with the current ADHB Clinical Guidelines and appropriate decision making occurred throughout her admission. However, the review did, in retrospect, identify areas of potential modification or improvement to ADHB's practice, although it was the team's view that this would have been very unlikely to have changed Miss A's outcome. The areas were:

> "1. Despite worsening clinical condition and no cause identified there were no further investigations undertaken to identify the cause of her pneumonia.
>
> 2. Subsequent to 1 above, she did not receive anti-viral nor anti-mycoplasma treatment. It was uncertain whether this impacted on her final outcome."

The review made recommendations to review and update the current ADHB Clinical Guideline for Pneumonia, and the creation and inclusion of a pathway for investigation and management of those with severe progressive pneumonia, including detail on when and how to test for atypical pneumonia and viral pneumonia. It also recommended:

> "a) For those with severe or deteriorating course of pneumonia, to include consideration of NP swab for atypical PCR panel and respiratory virus panel.
>
> b) Empiric combination therapy with a macrolide (oral or parenteral), in addition to a beta-lactam[1] antibiotic can be considered/prescribed for the hospitalised child for whom M. pneumonia and C. pneumoniae are significant considerations OR with severe or progressive disease where no other causative pathogen is found OR for children with presumed bacterial CAP[2] who do not have radiographic evidence that distinguishes bacterial from atypical CAP.
>
> c) Outline clearly indications for testing for influenza and use of oseltamivir[3] in hospitalised children in both the Influenza guideline and Pneumonia pathway (noting that this was currently only outlined in the Influenza guideline).
>
> d) Create links between relevant guidelines including ADHB Empyema guideline, Pneumonia guideline, and Influenza guideline.
>
> e) Communication of these changes to all ADHB clinical staff."

[1] Beta-lactam antibiotics are a class of antibiotics.
[2] Community acquired pneumonia.
[3] Oseltamivir is an antiviral medication used to treat influenza.

The specialist reviews of the services are outlined below.

Children's ED
Dr I, a paediatric emergency medicine specialist, considered that on 23 Month1, clinically there were no signs that this was more likely to be mycoplasma. He noted that the majority of pneumonias in Miss A's age group are likely to be viral, which they expect to recover spontaneously and for which antibiotics will make no difference. He advised that antibiotics are normally prescribed since it is difficult to determine which children with pneumonia have a bacterial cause on clinical, radiological, or laboratory criteria. Amoxicillin is the first-line oral antibiotic as it is well tolerated and covers the most frequent bacterial causes.

Dr I considered that at Miss A's re-presentation on 25 Month1, it was not routine to take nasopharyngeal aspirates/swabs in the children's ED as these do not provide results within the period required for initial treatment decisions and rarely change management. He noted that sputum samples are rarely indicated and are not readily obtained from young children. He advised that cefuroxime is the recommended antibiotic for complicated pneumonia in the ADHB Guidelines. Miss A's full blood count results were unremarkable and did not change her initial management. The initial attempt to obtain electrolytes and CRP measurements were unsuccessful since the specimen was haemolysed. This is usually due to a difficult specimen draw, is not unusual, and does not prevent initiating appropriate treatment for pneumonia.

General paediatrics
Dr F, a paediatrician, considered that the care provided to Miss A during her 25–27 Month1 admission was appropriate and consistent with ADHB guidelines. At the time of decision to transfer to DHB2, Miss A appeared clinically stable, and it appeared to be a reasonable decision.

He considered that Miss A deteriorated soon after transfer, which was unfortunate timing, and if the deterioration had occurred prior to transfer, then Miss A was likely to have been kept in ADHB. He considered that the clinical course would have been similar whether she had stayed or was transferred.

Dr F considered that at her readmission to ADHB on 28 Month1, there was an opportunity to consider the cause of her illness with more testing such as nasopharyngeal swab and respiratory PCR. He said that it was unclear whether earlier identification of mycoplasma and influenza would have changed the clinical course, although may have changed antimicrobial[4] choice once in PICU. He stated that the decision to transfer to PICU on 29 Month1 was appropriate.

Infectious Diseases
Dr E, an infectious diseases specialist, considered that the oral amoxicillin prescribed at Miss A's first presentation was appropriate and consistent with the ADHB Pneumonia

[4] Antimicrobial refers to an agent that inhibits the growth of microorganisms.

Guideline at the time of events. She noted also that the ADHB Guidelines remain consistent with international recommendations.

Dr E noted that there was a lack of specific testing at Miss A's first and second presentations, and lack of consideration for testing for influenza or atypical pneumonia pathogens in community-acquired pneumonia at her second presentation and admission. However, Dr E considered that testing was consistent with the ADHB Clinical Guidelines. She noted that a chest X-ray, blood cultures, and CRP were performed at the second presentation, the chest X-ray changes were consistent with lobar pneumonia, and intravenous cefuroxime was appropriately selected in view of the failed response to amoxicillin.

Dr E noted that the general consensus is to treat influenza once diagnosed for hospitalised children with influenza or with risk factors for severe disease. She noted that there was no investigation for influenza in this case. She noted that there is conflicting evidence worldwide that use of oseltamivir can alter the course of influenza, therefore it is not widely recommended in outpatient or mild pneumonia to either test or use oseltamivir for ambulatory patients (first presentation).

Dr E noted that there was no investigation for atypical pathogens nor viral pneumonia, although she noted that mycoplasma is often a self-resolving pneumonia and there is conflicting evidence that macrolide antibiotics can alter its course.

Dr E identified that there was a lack of "atypical" presentation (such as wheeze, chest X-ray changes, or arthralgia) that would lead attending clinicians to consider investigation of atypical pathogens, addition of macrolide antibiotics, or investigation for influenza. She considered that the lobar appearance on X-ray, very high inflammatory markers, development of dense consolidation, and progression of the fluid build-up (effusion) clinically typical of bacterial pneumonia and empyema with classic causes, were covered by appropriate antibiotics and in concordance with guidelines.

Dr E considered that testing for both atypical pathogens and viral pathogens should have been considered in the context of worsening disease and progression to surgical intervention and intensive care.

Paediatric surgery

Dr J, a paediatric surgeon, reviewed the placement of the intercostal chest drain on 28 Month1, the VATS on 4 Month2, and the decision not to operate prior to commencement of ECMO on 7 Month2.

Dr J agreed with the decision to place a chest drain on 28 Month1 and the timing of the procedure. He noted that the postoperative chest X-ray looked satisfactory and drainage of simple fluid was achieved. Fluid was sent from theatre for appropriate laboratory analysis. Although there is a comment in the clinical notes that the chest drain was positioned high in the chest, he did not think the position was important and subsequent imaging amply demonstrated satisfactory resolution of the effusion.

Dr J noted that the surgical team who had placed the chest drain requested postoperative urokinase instillation, and documented the protocol for fibrinolysis in the case notes, but the first dose of urokinase was not instilled until 30 Month1 and the drain fell out the next day, and therefore an incomplete course of urokinase was administered.

However, he stated that looking at the radiological imaging at the time of chest drain insertion and subsequent imaging up to the point of chest drain displacement, he did not feel that urokinase treatment was necessary, as the fluid was not complex and satisfactory drainage occurred without fibrinolysis.[5] He therefore felt that the failure to complete a course of fibrinolysis did not compromise the patient's outcome, and in fact he would not have prescribed urokinase himself.

Dr J agreed with the decision not to resite the chest drain after displacement, as there were no clinical or radiological indicators to suggest that any further surgical intervention was necessary at that stage.

In relation to reaccumulation of pleural fluid which showed some septation on 3 Month2, while he noted that it is difficult to predict the benefit of drainage at that stage, he generally agreed that the size and clinical status justified drainage. He also noted that while the benefit of VATS was unclear and simple drainage may have been effective, there was radiological evidence of septation and their practice is to place chest drains under general anaesthetic, and he agreed with the decision to perform VATS at that stage and believed the procedure was conducted in a safe and judicious manner.

He said it should be noted that a "minimal" decortication[6] was performed, and the operation note states that simple fibrinous strands were broken down and a limited washout was performed. This was not an extensive, long procedure and was likely beneficial in optimising drainage.

Dr J noted that Miss A's condition deteriorated postoperatively, with poorer oxygenation and hypotension[7] needing inotropic support.[8] He noted that a bronchial blocker[9] could not be deployed by the anaesthetists in theatre because of instability. Because of this, it is conceivable that some contamination of the left lung could have occurred intraoperatively due to spillage from the right bronchial tree, but the judicious approach to decortication described suggests that appropriate care was taken to minimise this risk. This risk is not unavoidable, but he believed the perceived benefits of the procedure undertaken at that stage justified the risk.

Dr J noted that it was unclear when exactly the surgical emphysema developed. He considered it unlikely that the air leak, which was most likely from a bronchopleural fistula

[5] Fibrinolysis refers to the process of breaking down fibrin in blood clots or other body fluids including pulmonary effusions.
[6] Decortication refers to surgical removal of the surface layer of the lung.
[7] Low blood pressure.
[8] Inotropes are medications that change the force of the heart's contractions.
[9] A bronchial blocker is a device that can be inserted into the main bronchus of the lung requiring isolation and the cuff intermittently inflated to block gas flow.

in the middle lobe, was caused by the VATS procedure. In his view it was caused by the underlying lung disease. The required high ventilator pressures resulted in the development of surgical emphysema as the air "vented" into the chest wall along a chest drain track. It should be noted that the case records suggest that air seemed not to accumulate further in the body wall once it was noted, and there was no bubbling via the chest drains at any time despite them being flushed and demonstrated to be patent. Therefore the bronchopleural fistula would appear to have been controlled by the ventilator strategy utilised at this stage.

Dr J noted that Miss A's condition continued to deteriorate and she was commenced on ECMO on 7 Month2. He noted that considering whether surgery is appropriate in this setting is an extremely difficult clinical decision and he supported the decision not to perform a thoracotomy at this stage because:

- Miss A had extensive bilateral consolidation, which would not have been improved by surgery, and surgery could have made this worse.
- It was unclear whether the resection of necrotic lung would have resulted in improvement of her clinical status even were it to have been achieved safely.
- Lung resection in this setting is an extremely challenging undertaking. An attempt at resection/lobectomy may have resulted in difficult or impossible to manage air leaks, life-threatening bleeding, pneumonectomy, and even death.

In his opinion the decision to proceed to ECMO without surgical resection was therefore appropriate.

Dr J noted that the surgical approaches used and decision-making strategies were consistent with many other cases they have managed successfully over the years with extensive necrotising pneumonias. In summary, he considered that the timing of surgery, surgical approaches employed, and the decision-making about when to operate and when not to were all appropriate. He considered that from a surgical perspective the outcome here was unexpected but could not reasonably have been avoided.

Paediatric intensive care
Dr G provided an overview of the care provided to Miss A in the PICU but did not provide an opinion.

Meeting minutes from … meeting with Miss A's family
A meeting between ADHB staff and Miss A's family occurred on …, to discuss ADHB's review and the family's concerns. Dr G and Dr I were also present.

Dr G was noted to have stated that if Miss A had been given erythromycin earlier it may not have made any difference to her outcome. Dr I noted that Miss A's case was very unusual and her influenza B was not diagnosed in the initial stages, but that treatment would not have changed if it had been known earlier. Influenza B was not suspected initially, so tests were not performed, in keeping with the current ADHB guidelines, and, at the time of diagnosis, treatment with antiviral medication was not indicated.