Health Centre

Medical Officer, Dr B

A Report by the
Health and Disability Commissioner

(Case 17HDC01683)

Health and Disability Commissioner
Te Toihau Hauora, Hauātanga

Contents

Executive summary

1. This report concerns the services provided to a man in his eighties at a community health centre (the health centre) during his final stay there. The Commissioner found Dr B in breach of Right 4(1) of the Code for providing suboptimal care to the man, and found the health centre vicariously liable for Dr B's breach.

2. The man displayed a number of concerning signs. An X-ray of his chest showed a slight abnormality, his vital signs had deteriorated, his white blood cell count was low, and his C-reactive protein levels were high.

3. The medical officer did not change the man's medical instructions in response to these signs.

4. The following day, another medical officer diagnosed the man with pneumonia and a urinary tract infection (UTI). She prescribed trimethoprim for his UTI and considered increasing his steroid dosage, but judged that it would be futile.

Findings

5. The Commissioner found that the care provided by Dr B fell below the appropriate standard in the following respects:

- He omitted to synthesise his observation of the chest X-ray with further information that subsequently came to light.
- He omitted to collect blood for culture when it was reported that the man's vital signs had deteriorated.
- Despite a number of convincing factors, he failed to identify that the man had developed sepsis, with the consequence that he did not provide antibiotics to the man in accordance with accepted practice.

6. The Commissioner found that the health centre had not taken such steps as were reasonably practicable to prevent Dr B's omissions, and that it was vicariously liable for his breach.

7. The Commissioner criticised the second medical officer for prescribing trimethoprim when ciprofloxacin would have been more appropriate, and for not increasing the man's steroid dosage when he was deteriorating.

Recommendations

8. The Commissioner recommended that both medical officers reflect on their failings and report to HDC on their reflections and changes to practice.

9. The Commissioner recommended that the health centre review the effectiveness of the changes it made following these events, and report to HDC on the outcome of an audit it performed.

10. The Commissioner recommended that the medical officers and the health centre apologise to the family.

Complaint and investigation

11. The Health and Disability Commissioner (HDC) received a complaint about the services provided to Mr A by Dr B and the health centre. The following issues were identified for investigation:

- *Whether the health centre provided Mr A with an appropriate standard of care in Month3 and Month4 2016.*
- *Whether Dr B provided Mr A with an appropriate standard of care in Month3 and Month4 2016.*

12. This report is the opinion of the Health and Disability Commissioner, Anthony Hill.

13. The parties directly involved in the investigation were:

Complainant/Mr A's son	
Community health centre/provider	
Dr B	Medical officer/provider
Dr C	Medical officer/provider

14. Also mentioned in this report:

RN D	Registered nurse

15. Further information was received from the Office of the Coroner, the Medical Council of New Zealand, and the District Health Board.

16. Independent expert advice was obtained from Dr Sarah Clarke, a rural medicine specialist (Appendix A).

Information gathered during investigation

Introduction

17. Mr A was in his eighties at the time of these events. He suffered from a number of health issues, including vasculitis,[1] hypertension,[2] type 2 diabetes,[3] chronic renal failure,[4] and systemic lupus erythematosus.[5] This opinion relates to the services he received at the health centre between 27 Month3[6] and 8 Month4, when he died of a chest infection.

[1] Inflammation of the blood vessels.
[2] High blood pressure.
[3] A condition where the body cannot regulate its blood sugar levels appropriately.
[4] Loss of kidney function.
[5] A long-term autoimmune disease.
[6] Relevant months are referred to as Months 1–4 to protect privacy.

Public hospital — treatment and discharge

18. Between Month1 and Month3, Mr A received treatment at the public hospital for a variety of health issues related to his gastrointestinal tract.[7] On 25 Month1, Mr A underwent a laparotomy,[8] a sigmoid resection,[9] and a colostomy.[10] On 12 Month2, an evisceration of his small bowel[11] was reduced.

19. On 27 Month3, the public hospital discharged Mr A to a community health centre that operates a small rural hospital. The discharge summary noted that Mr A's "[u]rinary culture showed growth of Enterobacter cloacae[12] group sensitive to trimethoprim[13]", and also that his creatinine[14] levels had "risen to 484[15] likely secondary to a combination of dehydration and trimethoprim treatment".

20. The discharge summary indicated the public hospital's plan for the team at the health centre to take over care of Mr A, including administration of his medication and assistance with his rehabilitation. The plan set out an extensive medication programme, including a direction that Mr A be given two 5mg prednisone[16] tablets per day until 3 Month4, and then one 5mg prednisone tablet per day thereafter. Accordingly, health centre staff provided Mr A with 10mg of prednisone per day until 3 Month4, and 5mg per day on 4, 5, and 6 Month4.

Health centre — 6 Month4

21. Between 7pm and 12am on 6 Month4, RN D recorded Mr A's vital signs as "B/P 172/90, RR18, Temp 36.3, O2 sats 99% on air. BGL 7.1mmol/l".[17] She noted that Mr A had a cough and had vomited a "moderate amount", and that he had complained of abdominal discomfort. She administered Mr A 4mg of ondansetron,[18] following which he reported that his abdominal discomfort had resolved.

First call to Dr B — early morning, 7 Month4

22. At 1.30am on 7 Month4, RN D reviewed Mr A's vital signs and noted a deterioration to "B/P 170/100, HR 120 irreg, Temp 37.7, RR28, BGL 12.5mmol/l, O2 sats 86% on air". RN D recorded that Mr A had "vomited [a] small amount" and that his cough was "persistent"

[7] Digestive tract.
[8] Surgical examination of the abdominal organs.
[9] Removal of the part of the colon.
[10] Creation of an artificial opening in the colon to divert waste through the abdominal wall.
[11] A herniation of the small bowel.
[12] A species of bacteria that can cause several different types of infection.
[13] An antibiotic used to treat and prevent urinary tract infections.
[14] A waste product caused by wear and tear to the muscles.
[15] Higher than normal — a high creatinine output can indicate issues with the kidneys.
[16] A steroid medication used to suppress the immune system and decrease inflammation.
[17] Blood pressure 172/90mmHg, respiratory rate 18 breaths per minute, temperature 36.3°C, blood oxygen saturation 99%, and blood glucose level 7.1 millimoles per litre.
[18] A medication used to prevent and treat nausea and vomiting.

and "increasingly moist sounding". She telephoned the on-call medical officer, Dr B, who instructed her to provide Mr A with 15mg of codeine and 25mg of cyclizine.[19]

23. RN D observed Mr A throughout the night and made several further entries in his clinical notes. Mr A's vital signs are not referred to again, but the notes record that he was feeling better, his vomiting had stopped, and his coughing was decreasing. RN D noted that a urine specimen had been tested and showed "leukocytes[20] present".

Personal review by Dr B — morning, 7 Month4

24. Dr B reviewed Mr A at 9.50am on 7 Month4. Dr B noted: "[Mr A's] main problem today is SOB.[21] He had some vomiting last evening followed by SOB." Dr B recorded Mr A's temperature as 37.3°C, his oxygen saturation as 86%, and his respiratory rate as 28 breaths per minute. Dr B told HDC that his "differential diagnosis included aspiration pneumonia,[22] congestive heart failure and UTI[23]".

25. Dr B also reviewed an X-ray of Mr A's chest taken that morning, and recorded that it showed "minor congestion,[24] no obvious consolidation[25]".

26. Dr B decided to stop Mr A's subcutaneous hydration[26] and raise his bed by 45 degrees, and instructed that Mr A be given extra oxygen if his blood oxygen saturation dropped below 93%.

27. Dr B requested blood tests, which were reported at 2.06pm as white blood cells "2.0 L" and neutrophils "1.6 L".[27] This was a decrease from his white blood cell count of 9.0 per litre and his neutrophil count of 8.0 per litre from his blood test results on 5 Month4.

28. Mr A's creatinine level was reported as 456. This was a decrease from his 5 Month4 level of 482, but an increase from his 1 Month4 level of 402. Mr A's C-Reactive protein (CRP) level[28] was reported as 76, which was an increase from the last reported result of 9 on 1 Month4.[29]

Afternoon, 7 Month4

29. At 3.20pm, a nurse recorded Mr A's vital signs as "BP 127/75, HR 92, RR 24, T 37.9, O2 sats 90% 1L O2 via NP".[30] This was an improvement on Mr A's earlier signs at 1.30am and

[19] A medication used to prevent and treat nausea and vomiting.
[20] White blood cells. High numbers in urine can indicate an infection.
[21] Shortness of breath.
[22] Lung infection.
[23] Urinary tract infection.
[24] A build-up of mucus and fluid in the lungs.
[25] A swelling or hardening of a part of the lung because of the presence of fluid.
[26] Insertion of fluid into the area between the skin and muscle.
[27] 2,000 white blood cells per microlitre of blood, and 1,600 neutrophils per microlitre (compared to the normal range of 4,000 to 11,000 white blood cells and 1,900 to 7,500 neutrophils).
[28] A substance produced by the liver in response to inflammation.
[29] 76 milligrams and 9 milligrams per litre of blood. A result of over 10 milligrams is associated with (but not necessarily indicative of) inflammation.
[30] His blood oxygen saturation was 90% while receiving a litre of oxygen via nasal prongs.

9.50am earlier that day, with the exception of his temperature, which had increased. The nurse withheld Mr A's daily dose of 5mg of prednisone because he was not eating.

Second call to Dr B — evening, 7 Month4

30. At 8pm, RN D noted Mr A's vital signs as "Temp 38, B/P 129/75, HR 103, RR28, EWS=2,[31] O2 sats 85% on 1 litre O2 via NP". She increased Mr A's oxygen to two litres per minute and telephoned Dr B (who was on call) to inform him of Mr A's vital signs and the results of the blood test that had been reported earlier that day. Dr B did not change Mr A's medical instructions.

31. Dr B did not ask RN D to collect a blood sample for culture. He told HDC:

> "Unfortunately, I forgot to ask for blood culture when [Mr A] had fever and I agree that was a mistake and was not my usual practice ... I accept it was a mild departure from the applicable standard of care."

32. Dr B stated that his omission to collect a blood sample for culture "had no impact on [Mr A's] care", but "in hindsight, and now knowing the cause of death, starting empiric antibiotics would have been a better choice". He submitted that his omission was not a severe departure from the standard of care because of the complexity of Mr A's medical condition. Dr B stated:

> "[Mr A] was a complex patient and I did not want to cause him harm by making the wrong decision. [Mr A] presented to me as a patient experiencing [congestive heart failure] and, again with hindsight, that is what I focused on. I was also mindful of the potential involvement of his recent suspected bowel obstruction."

33. Dr B told HDC that a number of factors influenced his decision not to start Mr A on antibiotics at this time, including the following:

a) Several of Mr A's exhibited symptoms were as indicative of heart failure as they were of sepsis,[32] including shortness of breath, a heart rate higher than 100 beats per minute, fatigue and weakness, and nausea.

b) Although Mr A's CRP had increased to 34, it "had been fluctuating and could have been linked to his prior suspected bowel obstruction. As an inflammatory marker it is not a very specific indicator."

c) Mr A's temperature was never recorded as exceeding 38°C, and in fact decreased to 36.4°C by the morning of 8 Month4.

d) At 1,600 neutrophils per microlitre, Mr A "was not technically neutropenic[33] based on guidelines from [Regional] Health Pathways" (which set 1,000 to 1,500 neutrophils per microlitre as the spectrum of mild neutropenia).

[31] Early Warning Score — a nationally standardised scoring tool used to escalate patients' treatment based on deteriorating vital signs. The scores range from 0 to 10, with higher scores indicating a higher priority for escalation. Mr A's EWS was 2.

[32] A condition where the organs are endangered by the body's strong reaction to infection.

e) He "was worried about [Mr A's] kidney injury experienced during his long hospital admissions. With 'first, do no harm' in mind, [he] decided not to start with antibiotics until [he] had more evidence."

Review by Dr C — morning, 8 Month4

34. Dr C, a medical officer, reviewed Mr A at 10.30am on 8 Month4. She recorded: "[Mr A] continues to be unwell and deteriorating clinically, with minimal food intake, looks SOBE,[34] moist cough … looks very very tired, tachypnoea.[35]" Dr C noted Mr A's vital signs as "BP=103/67. HR=94, RR=28, T=36.4, O2 sats=92% 1l".

35. Dr C also reviewed the chest X-ray that Dr B had reviewed the previous day. She recorded: "[C]hest X-ray done yesterday showed heart not enlarged, diffuse increased interstitial markings, no obvious consolidation, but WBC[36] was low (official report pending)."

36. Based on these observations, Dr C recorded her impression that Mr A was "[u]nwell with … clinically hospital acquired pneumonia left side" and a recurrence of the Enterobacter UTI observed by the public hospital's medical team in Month3. She spoke to an infectious diseases consultant, who advised her to treat Mr A with piperacillin and tazobactam.[37]

37. Dr C told HDC that the infectious diseases consultant advised her "not to start cotrimoxazole[38] despite the risk of pneumocystis pneumonia (PCP)[39]" because "both the dose of Trimethoprim component (higher than for other indications) required to treat PCP and the duration of the treatment (at least 3 weeks for PCP) pose a significant risk of worsening the renal function", and because Mr A's "chance of surviving even with treatment was poor".

38. Dr C considered that Mr A's prognosis was "poor", and informed one of his children about his deteriorating situation. She told HDC that she stopped Mr A's mycophenolate,[40] and instructed that he be provided with piperacillin and tazobactam for his pneumonia, and trimethoprim for his UTI.

39. Dr C stated:

> "At the time of the discussion with the ID [Infectious Diseases] consultant the results of the urine culture were not available. When they became available, the only options for treatment of UTI were trimethoprim, ciprofloxacin or gentamycin. Gentamycin was not an option I felt due to the poor renal function. The duration and dose of trimethoprim needed for UTI infection was less than for PCP. I did not feel that my

[33] An abnormally low neutrophil count.
[34] Shortness of breath on exertion.
[35] Abnormally rapid breathing.
[36] White blood cell count.
[37] Two antibiotics that are often prescribed alongside each other for a stronger effect.
[38] An antibiotic containing trimethoprim.
[39] A type of chest infection caused by a fungus.
[40] An immunosuppressive agent that is used to prevent the immune system from attacking kidney transplants.

> decision to treat UTI with trimethoprim would pose the same risk of renal function deterioration. I did not feel that I went against the advice of the ID consultant. The Medsafe datasheet for trimethoprim states that where the eGFR[41] is 10 or above, a standard dose may be used but plasma levels should be monitored after approximately 3 days of treatment. [Mr A's] eGFR was 10."

Afternoon, 8 Month4

40. Later that day, Mr A's urine culture became available. This revealed "a growth of Enterobacter species" susceptible to ciprofloxacin, co-trimoxazole, gentamicin, norfloxacin, and trimethoprim.[42]

41. At 2.30pm, a nurse recorded that Mr A was unable to swallow his trimethoprim tablet. Consequently, Dr C instructed that his trimethoprim be replaced with intravenous ciprofloxacin.

42. Dr C told HDC that she "did consider increasing the prednisone" being given to Mr A, but she "felt it was not mandatory to give a stress dose[43] [of prednisone]", and that increasing the amount of Mr A's prednisone "would be futile, as [Mr A] had deteriorated significantly during the day and was unlikely to survive much longer".

Deterioration — late afternoon, 8 Month4

43. Dr C recorded at 5pm that Mr A had deteriorated rapidly and was "now basically not responsive to voice". She noted that he "look[ed] quite terminal", and that she had told his son that this was "likely a terminal event". She recorded that she would give Mr A another measure of piperacillin and tazobactam, and that his situation would be reviewed again "later during the day".

44. At around 7.10pm, Mr A's son informed staff that Mr A had stopped breathing. Dr C reported on the health centre's "Record of Death" form that Mr A died at 7.20pm. She wrote: "Became unwell last 48h — ? hospital acquired pneumonia ? heart failure ? fungal chest infection. Died despite large spectrum antibiotic started only today." Dr C circled "without known cause" in relation to Mr A's death.

Coronial autopsy report

45. Because Dr C was unclear about the cause of Mr A's death, she referred his death to the Coroner. She told the Coroner:

> "It was my impression that [Mr A] died of an infectious complication. However, he did not respond to broad base antibiotic treatment. I was wondering if the cause of death was not a fungal infection, or pneumocystis pneumonia. This would have explained

[41] Estimated Glomerular Filtration Rate. This is a measure of the kidney's functionality, with a score of 90 to 100 indicating a healthy kidney, and a score of less than 15 indicating kidney failure.
[42] Various types of antibiotic.
[43] A large dose of steroids given to a patient to compensate for the body's inability to produce sufficient hormones in response to an illness.

> the lack of response to antibiotics. It is finally possible that the cause of death was a bacterial infection, but treated too late or a pulmonary embolism or other event."

46. A pathologist reviewed Mr A's body and prepared a coronial autopsy report. She summarised:

> "From the following detailed examination, I am of the opinion that death was due to septicaemia[44] following a febrile illness involving urinary tract infection and aspiration pneumonia. The deceased was immunocompromised to fight this infection due to diabetes, diabetic nephropathy,[45] and treatment for lupus erythematosus."

47. Under the heading "Cause of Death", the pathologist reported the direct cause as "[s]epticaemia", the antecedent cause as a "[u]rinary tract infection", and relevant underlying conditions as "[i]mmunosuppression, diabetes and aspiration pneumonia". The report stated: "Other significant conditions contributing to the death, but not related to the disease or condition causing it [were] [a]dvanced age and debilitation." The Coroner accepted the pathologist's report and found that Mr A had died of septicaemia caused by a UTI.

Further information

Posthumous review of chest X-ray

48. On 19 Month4, a consultant radiologist reviewed the X-ray of Mr A's chest that Dr B had reviewed on 7 Month4. Allowing for the fact that "[t]he patient was not able to take a deep breath", she found the following:

> "[T]he heart size and pulmonary vessels are normal. There is a little patchy change at the left lung base medially. No other focal abnormality seen in the lungs. There is an old healed fracture of the right seventh rib."

Dr B

49. Dr B told HDC:

> "I am currently under the GPEP [General Practice Educational Programme] and I am in my 3rd year of training. I work in a very supportive practice with an excellent mentor with weekly teaching sessions. I also attend a weekly peer meeting at practice and a monthly peer group with other registrars."

50. Dr B stated:

> "Since these events, I have researched and reflected on materials including:
>
> - Overview of neutropenic fever syndrome.
> - Evaluation and approach to adults with undifferentiated hypotension[46] and shock.

[44] Blood infection.
[45] Loss of kidney function caused by diabetes.
[46] Abnormally low blood pressure.

- Treatment of acute decompensated heart failure.
- Diagnosis and treatment of sepsis."

51. He also said: "Since these events, I have been especially careful not to miss any requests for blood culture for febrile patients and to consider starting antibiotics."

Health centre

52. The health centre told HDC that subsequently it made the following changes:

- It introduced a Nurse Initiated Sepsis Pathway/Sepsis Triage Flow Chart, which is used for the assessment and initial clinical management by nursing staff of patients clinically symptomatic of sepsis. The health centre said that "[t]he flow chart facilitates the early obtaining of a number of relevant diagnostic tests and for the empirical prescribing and administration of antibiotics".
- It incorporated an "Early Warning Score System" into the patient observation chart.
- The RHM Specialists provided an education session to nursing and medical staff on the topic of sepsis.
- Currently it is undertaking an audit to review the time from assessment/provisional diagnosis of sepsis to when antibiotics are administered. The audit will compare those interventional treatment times to those post-introduction of the Nurse Initiated Sepsis Pathway/Sepsis Triage Flow Chart.

Responses to provisional opinion

53. Mr A's son was given an opportunity to respond to the "information gathered" section of my provisional report, and had no comments to make.

54. The health centre, Dr B, and Dr C were given the opportunity to respond to the relevant sections of my provisional report. Their responses have been incorporated into the report as appropriate.

55. The health centre told HDC that its "Nurse Initiated Sepsis Pathway" (implemented after these events) was "designed to raise awareness of sepsis and to emphasise the need for early administration of intravenous antibiotics on a patient's initial presentation", rather than "to be a 'standing order' for nurses to administer antibiotic therapy prior to discussion with the ward doctor". The health centre submitted that it "was not entirely convinced" that the Nurse Initiated Sepsis Pathway would have "prevented the incorrect treatment trajectory provided to [Mr A]" if it had been in place at the time.

56. Dr B told HDC that he was "presented with a complex clinical picture", and that he "was only ever motivated by what he thought was best for [Mr A]". Dr B also stated that he recognised "with hindsight, that aspects of the treatment provided should have been done differently", and has since "worked hard to educate and upskill himself to ensure he would act appropriately if faced with the same or a similar situation in the future".

57. Dr C told HDC that she had "no comments to make on HDC's provisional opinion", and stated: "I accept the Commissioner's conclusion and recommendations."

Opinion: Dr B — breach

Standard of care provided — breach

58. Several aspects of the care provided by Dr B are concerning. These include:

- Dr B's morning and evening interpretations of Mr A's chest X-ray taken on 7 Month4.
- Dr B's omission to request a blood culture when Mr A developed a fever in the evening of 7 Month4.
- Dr B's decision not to provide antibiotics to Mr A in the evening of 7 Month4.

59. HDC obtained independent clinical advice from Dr Sarah Clarke, a rural medicine specialist, on whether the care that the health centre and its staff provided to Mr A was reasonable in the circumstances.

Interpretation of chest X-ray

60. Dr Clarke advised:

> "[Dr B's] interpretation of the chest x-ray was of 'mild congestion' where-as the later formal report stated 'There is a little patchy change at the left lung base' and the post mortem examination revealed resolving and acute aspiration pneumonia, both suggesting that [Dr B's] initial interpretation of the chest x-ray was incorrect. Interpretation of investigations must be performed with consideration as to the clinical context."

61. Dr Clarke noted:

> "At the time that chest x-ray was performed [Mr A] had suffered an acute deterioration from the night before of his respiratory rate (18 to 28) and his oxygen saturation (99% on room air to 86% on room air) which are both suggestive of an acute respiratory compromise. At that time, given the context that [Mr A] had not developed a fever, this misreading of the chest x-ray represents a mild departure from standard of care, and would be considered a reasonable error of judgment by my peers."

62. However, concerning Dr B's evening interpretation of the chest X-ray on 7 Month4 2016, Dr Clarke advised:

> "At the time that [Mr A] developed a fever and the blood test results were available showing a leukopenia[47] and elevation of C-reactive protein [Dr B] should have had a high index of suspicion that the changes he'd seen on the chest x-ray and described as 'congestion' may have represented infection, and as such his interpretation that 'there was no significant consolidation on the chest x-ray to represent pneumonia' would represent a moderate departure from standard of care and the decision to 'rule out' respiratory infection based on the x-ray interpretation would be viewed as unreasonable by a group of my peers."

63. Dr Clarke further advised:

> "Tests are performed to add information to a diagnostic picture and should not be interpreted in isolation. [Dr B's] interpretation of the chest xray as showing 'mild congestion' indicates that he recognised that the xray was not normal, and in considering this within the clinical context of tachypnoea and hypoxia it is reasonable to consider this abnormality to be related to heart failure, while infection should still have remained on the differential."

64. Dr B submitted that at the time, he interpreted the chest X-ray correctly as showing no obvious consolidation. I have considered the fact that Dr C also interpreted the X-ray as showing no obvious consolidation.

65. However, as my expert advisor notes, X-rays should not be interpreted in isolation. I am critical of Dr B's apparent omission to synthesise this observation with further information that subsequently came to light, including Mr A's deteriorating vital signs and reduced white blood cell count. I accept Dr Clarke's advice that against the background of that further information, Dr B "should have had a high index of suspicion that the changes he'd seen on the chest x-ray and described as 'congestion' may have represented infection".

Omission to request blood culture

66. Dr Clarke advised:

> "Indications for blood culture include:
>
> 1. Clinical features of sepsis including tachycardia,[48] tachypnoea, increased or subnormal temperature and change in sensorium,[49] hypotension or prostration
> 2. Suspicion of infective endocarditis[50]
> 3. Pyrexia[51] of unknown origin
> 4. Unexplained leukocytosis[52] or leukopenia

[47] An abnormally low white blood cell count.
[48] An abnormally high heart rate.
[49] The human senses.
[50] Infection of the inner lining of the heart chambers or heart valves.
[51] Raised body temperature.
[52] An abnormally high white blood cell count.

5. Systemic and localised infections including suspected meningitis, osteomyelitis,[53] septic arthritis, acute untreated bacterial pneumonia or other possible bacterial infection (Ntusi, Aubin, Oliver, Whitelaw & Mendelson, 2010).

At the time that [Mr A] developed a temperature of 38 his other observations were recorded as blood pressure 129/75, heart rate 103, respiratory rate 28 and oxygen saturations of 85% on 1 litre/minute of supplementary oxygen. As such he had many of the above criteria for the clinical features of sepsis including at least tachycardia, tachypnoea and increased temperature, and he was known to have leukopenia with likely 'possible bacterial infection' based on a known leukocytosis in the urine and an abnormal chest x-ray with hypoxia, tachypnoea and unilateral clinical chest findings that could be consistent with pneumonia. In this situation the accepted practice would be to collect blood for culture. Failure to do so was a moderate departure from the standard and this would be viewed by my peers as unreasonable."

67. Dr Clarke noted that she considered this a moderate departure "because the collection of blood for culture per se does not immediately impact upon the treatment of the patient's condition as positive results are often non available for many hours to days, though may direct antimicrobial therapy once available".

68. Dr B agreed that his omission to ask RN D to obtain a blood culture "was a mistake". However, he noted that this "was not [his] usual practice" and "had no impact on Mr A's care". Dr B submitted that this was a "mild departure from the applicable standard of care".

69. I accept Dr Clarke's advice that the standard was for Dr B to have requested a blood culture. Notwithstanding whether this omission was an exception to Dr B's usual practice or whether it affected Mr A's care, I am critical of Dr B for omitting to request the blood culture in these circumstances.

Decision not to provide antibiotics

70. Dr Clarke provided HDC with a table of the diagnostic criteria for sepsis, and advised that at the time of RN D's telephone call to Dr B on the evening of 7 Month4:

"[Mr A] certainly met the above criteria for sepsis namely he had suspected infection (urine, possibly chest), tachypnoea, tachycardia, likely hypoxaemia, leukopenia, elevated C-reactive protein and a decrease of >40mmHg systolic blood pressure compared to his pre-deterioration BP (172 to 129), the latter of which would further characterise [Mr A] as having severe sepsis (Dellinger, et al., 2013)."

71. Dr Clarke stated:

"While any one of these abnormalities (each individual observation, or each individual blood test result) can be explained by alternative reasoning, the constellation of clinical signs and biochemical markers together points to a picture of sepsis, a

[53] Bone infection.

diagnosis that should have been in the forefront of [Dr B's] mind in this scenario and one that should have been emergently acted upon."

72. Dr Clarke further advised HDC:

"Accepted practice in the setting of sepsis is to prescribe antibiotics (Dellinger, et al., 2013). Failure to identify sepsis and/or prescribe antibiotics at this time represents a severe departure from the standard of care and would be seen by a group of my peers as unacceptable."

73. Dr B accepted that "in hindsight, and now knowing the cause of death, starting empiric antibiotics would have been a better choice" following RN D's telephone call on the evening of 7 Month4. However, he disagreed with Dr Clarke's assessment that his decision was a severe departure from the standard of care. He stated:

"[Mr A] was a complex patient and I did not want to cause him harm by making the wrong decision. [Mr A] presented to me as a patient experiencing [congestive heart failure] and, again with hindsight, that is what I focused on. I was also mindful of the potential involvement of his recent suspected bowel obstruction."

74. Dr B told HDC that several factors influenced his decision not to start Mr A on antibiotics, including the following:

a) Several of Mr A's exhibited symptoms were as indicative of heart failure as they were of sepsis, including shortness of breath, a heart rate higher than 100 beats per minute, fatigue and weakness, and nausea.

b) Although Mr A's CRP had increased, it "had been fluctuating and could have been linked to his prior suspected bowel obstruction. As an inflammatory marker it is not a very specific indicator."

c) Mr A's temperature was never recorded as exceeding 38°C, and in fact decreased to 36.4°C by the morning of 8 Month4.

d) At 1,600 neutrophils per microlitre, Mr A "was not technically neutropenic based on guidelines from [Regional] Health Pathways" (which set 1,000 to 1,500 neutrophils per microlitre as the spectrum of mild neutropenia).

e) He "was worried about [Mr A's] kidney injury experienced during his long hospital admissions. With 'first, do no harm' in mind, [he] decided not to start with antibiotics until [he] had more evidence."

75. However, Dr Clarke advised HDC:

"While I accept that heart failure and sepsis can co-exist in a patient, the relationship between heart failure and other morbidities is complex and the overarching issue in this case is that [Dr B] was 'focused on' a diagnosis of heart failure and failed to identify sepsis as a critical part of [Mr A's] deterioration. [Dr B] did not make the diagnosis of sepsis, and neither did he act upon his own suspicion of infection as

> described in his differential diagnosis that included UTI and aspiration pneumonia. I maintain that failure to prescribe antibiotics in this case was a severe departure from standard care."

76. Dr Clarke noted:

> "The reference in the discharge summary from the public hospital is to trimethoprim, an antibiotic which is known to cause and/or potentiate kidney injury (Fraser, Avellaneda, Graviss, & Musher, 2012), and would not preclude the use of other non-nephrotoxic antibiotic therapies and as such need not have been a consideration in the decision of whether or not to commence antibiotic therapy."

77. She advised:

> "I do not accept the concern regarding nephrotoxicity of antibiotics as a valid reason to withhold antibiotics in a patient with renal failure who meets sepsis criteria as options that are safe for this situation do exist. I also do not accept a concern regarding bowel obstruction to be relevant to withholding antibiotics in this context."

78. I accept Dr Clarke's advice that the information available to Dr B collectively indicated that Mr A had developed sepsis. Although it may have been reasonable for Dr B to suspect other contributing causes of Mr A's deterioration (such as heart failure), I would expect a practitioner of Dr B's experience to recognise sepsis in these circumstances, and I am critical that he did not do so.

79. I further accept Dr Clarke's advice that "[a]ccepted practice in the setting of sepsis is to prescribe antibiotics", and that Dr B's concerns about Mr A's kidney injury need not have prevented him from abiding by this practice. Dr B could have provided non-nephrotoxic antibiotics to Mr A. In the absence of a good reason for withholding non-nephrotoxic antibiotics from Mr A, I am critical of Dr B for not commencing antibiotic therapy.

Conclusion

80. Dr B provided care to Mr A that fell below the appropriate standard in the following respects:

- Dr B omitted to synthesise his observation of Mr A's chest X-ray with further information that subsequently came to light.
- Dr B omitted to collect blood for culture when it was reported that Mr A's vital signs had deteriorated.
- Dr B failed to identify that Mr A had developed sepsis despite a number of convincing factors indicating that he had, with the consequence that Dr B did not provide antibiotics to Mr A in accordance with accepted practice.

81. As a result of these omissions, opportunities were missed to treat Mr A's deteriorating condition appropriately. For these reasons, I find that Dr B breached Right 4(1) of the Code of Health and Disability Services Consumers' Rights (the Code).[54]

Decision not to review Mr A personally — other comment

82. Dr Clarke also commented on Dr B's decision not to review Mr A personally following the telephone call he received from a nurse around 1.30am on 7 Month4. Dr Clarke noted:

> "[Dr B] was made aware of [Mr A's] deteriorating status including tachypnoea, tachycardia and significant hypoxia, at 0130 on 07 [Month4] and managed this deterioration by telephone with oral medications. Further observations are not recorded in the nursing note for that shift.
>
> Had [Mr A] improved with the therapy given by phone and his heart rate, respiratory rate and hypoxia returned to normal levels then it may be reasonable that [Dr B] did not review [Mr A] in person at that time. If, however, [Mr A] remained tachycardic, tachypnoeic and hypoxic despite the therapies instituted by telephone order (which seems likely given his next reported set of observations) then standard of care would be to review the patient in person at the time."

83. Following the record of Mr A's vital signs at 1.30am, the clinical notes do not record further review of Mr A's vital signs until 9.50am. Nor do the notes record that anyone contacted or tried to contact Dr B again about Mr A's situation.

84. Dr B told HDC that he had "a good working relationship with the nursing staff" and "would have given instructions by phone to be contacted again in the event Mr A did not improve". Dr B stated:

> "[If] the nursing staff [had] contacted me again (or even if they had asked me to come in at 0130) I absolutely would have. It was common for me to do so — usually once or twice each night. I was staying [close by] at the time."

85. Having considered the evidence, I am of the opinion that it was not necessary for Dr B to review Mr A personally in the early morning of 7 Month4.

Opinion: Dr C — adverse comment

86. I am concerned about two aspects of the care provided by Dr C:

- The decision to treat Mr A with trimethoprim on 8 Month4.
- The decision not to increase Mr A's steroid dosage after identifying that he had sepsis.

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

Decision to provide trimethoprim

87. Dr Clarke advised:

> "While targeted therapy based on the culture and sensitivity could have included trimethoprim or ciprofloxacin, [Dr C] had been advised by the ID physician not to cover PCP due to the renal function, implying that the ID physician felt that trimethoprim was relatively contraindicated in the context of [Mr A's] renal status. To prescribe trimethoprim in the context of renal failure and ID advice to not give trimethoprim/sulfamethoxazole without consideration to safer alternative or renal dosing would be considered a moderate departure from accepted care and would be seen as illogical by a group of my peers."

88. Dr C explained that Mr A's UTI was susceptible only to trimethoprim, ciprofloxacin, and gentamycin, and she believed that gentamycin was not appropriate because of Mr A's poor renal function. She stated:

> "[T]he duration and dose of trimethoprim needed for UTI infection was less than for PCP. I did not feel that my decision to treat UTI with trimethoprim would pose the same risk of renal function deterioration. I did not feel that I went against the advice of the ID consultant. The Medsafe datasheet for trimethoprim states that where the eGFR is 10 or above, a standard dose may be used but plasma levels should be monitored after approximately 3 days of treatment. [Mr A's] eGFR was 10."

89. Dr Clarke advised:

> "I recognise that the treatment dose of trimethoprim for Pneumocystis pneumonia is higher than the dose prescribed by [Dr C] for the treatment of UTI. While I recognise that the Medsafe datasheet indicates that there need be no dose reduction for a patient with an eGFR of 10 or greater, [Mr A] was exactly on the borderline for this recommendation, clinically deteriorating (meaning his eGFR might drop further), and had a previous history of acute kidney injury while on trimethoprim for UTI. In the context of such a complex patient, with a history of previous acute kidney injury while on trimethoprim, I believe that the decision to prescribe trimethoprim in this context (and particularly in view of a potentially safer effective option being available, namely ciprofloxacin) would be one that most of my colleagues would have shared with either a Renal Physician or an Infectious Diseases Specialist."

90. I accept Dr Clarke's advice that — notwithstanding the relatively small dose of trimethoprim provided and Mr A's eGFR of 10 — Dr C's decision to commence trimethoprim was not appropriate. Furthermore, I find that Dr C's choice of trimethoprim over ciprofloxacin was sub-optimal, as ciprofloxacin was not contraindicated by Mr A's renal issues. Overall, I am critical of Dr C for putting Mr A's health at unnecessary risk by providing him trimethoprim rather than ciprofloxacin, and without consulting further with an infectious diseases specialist.

Decision not to increase steroid dosage

91. Dr Clarke advised HDC:

> "[Mr A] had been on long term steroid therapy with prednisone and had received a dose reduction shortly before his deterioration to 5mg daily and prior to this he had been on 10mg daily for at least the duration of the admission. It is noted by a nurse that the prednisone was withheld after the deterioration as [Mr A] was not able to swallow this. People who take long term steroids are at risk of adrenal insufficiency in the event of developing sepsis and as such their steroid dose needs to be increased to mitigate the risk in the setting of sepsis. I note that there was not an increase in steroid dose for 'stress cover' once sepsis was recognised by [Dr C]. Standard of care would be to increase the steroid cover in long-term steroid exposed patients in the context of sepsis, particularly in the setting of relative hypotension (Dellinger, et al., 2013). Failure to do so would be considered a moderate departure from the standard of care and would be considered unreasonable by a group of my peers."

92. Dr C explained that she considered increasing Mr A's stress dose in the afternoon of 8 Month4, but "felt it would be futile, as Mr A had deteriorated significantly during the day and was unlikely to survive much longer".

93. Dr Clarke advised:

> "I accept that if a palliative pathway had been initiated that it would be reasonable to withhold the steroid stress dose, but in the context that intravenous antibiotics were still being continued it seems that a decision to withdraw active care had not been made at the time of [Mr A's] death, although a ceiling of care excluding transfer to [the public hospital] or resuscitation had been agreed upon."

94. I accept Dr Clarke's advice that the standard of care in the event of sepsis for long-term steroid exposed patients is to increase their steroid dosage. I also accept her advice that as no "decision to withdraw active care" had been made in respect of Mr A, Dr C's decision not to increase Mr A's steroid dosage could not be justified on palliative grounds.

95. I recognise that Mr A's health was very fragile at the time concerned. However, decisions to withhold treatment on palliative grounds can be made only following a concrete decision to pursue a palliative pathway. No such decision had been made in respect of Mr A, so it was not open to Dr C to withhold treatment from him on palliative grounds. Therefore, I am critical of Dr C for deciding not to increase Mr A's steroid dosage in response to sepsis.

Opinion: Health centre— breach

96. As a healthcare provider, the health centre is responsible for providing services in accordance with the Code. In this case, I am satisfied that the issues with the care that Mr A received arose because of individual errors. However, in addition to direct liability for a breach of the Code, under Section 72(2) of the Health and Disability Commissioner Act 1994 (the Act), an employing authority is vicariously liable for any acts or omissions of its employees. A defence is available to the employing authority under Section 72(5) if it can prove that it took such steps as were reasonably practicable to prevent the acts or omissions.

97. In Month4, Dr B was an employee of the health centre. As set out above, I have found that Dr B breached Right 4(1) of the Code by providing care to Mr A that fell below the appropriate standard in a number of respects.

98. At the time of these events, the health centre did not have a nurse-initiated sepsis pathway or an Early Warning Score system. Dr Clarke advised HDC that a nurse-initiated sepsis pathway would help to prevent the recurrence of several of the errors that occurred in Mr A's care, including:

- Failure to identify sepsis.
- Failure to draw blood for culture in response to sepsis.
- Failure to provide the most appropriate antibiotic in response to sepsis.
- Failure to increase steroid dosage in response to sepsis.

99. Dr B told HDC that he believes his failure to identify sepsis was partially caused by his focus on a diagnosis of congestive heart failure. Although Dr B remains individually responsible for this oversight, I am concerned that neither he nor the nursing team he worked with were better supported by the health centre's policies and practices to respond appropriately to the signs that Mr A had sepsis.

100. Dr Clarke advised that "the use of a sepsis pathway may have reduced the risk of this erroneous decision [Dr B's decision not to provide antibiotics] being made, and as such that the institution of such pathway is likely to prevent this event happening again in future."

101. I acknowledge the health centre's submission that it "was not entirely convinced" that the sepsis pathway it later implemented would have "prevented the incorrect treatment trajectory provided to [Mr A]" if it had been in place at the time. The health centre's submission was put to Dr Clarke, who advised HDC that it did not cause her to change any of her previous advice. I accept Dr Clarke's advice and remain concerned that the health centre did not have adequate systems in place at the time to help its staff recognise and respond to cases of sepsis appropriately.

102. Accordingly, I find that the health centre did not take such steps as were reasonably practicable to prevent Dr B's omissions, and that the health centre is vicariously liable for Dr B's breach of the Code.

103. I acknowledge that since these events the health centre has developed and instituted a nurse-initiated sepsis triage flow chart "for the assessment and initial clinical management by nursing staffs of patients clinically symptomatic of sepsis". It has also "incorporated an 'Early Warning Score System' into [the] patient observation chart." I welcome these initiatives.

Recommendations

104. I recommend that Dr B:

a) Provide a written apology to Mr A's family. The apology is to be sent to HDC within three weeks of the date of this report, for forwarding to Mr A's family.

b) Reflect on his failings in this case and provide a written report to HDC on his reflections and the changes to his practice he has instigated as a result of this case, within six months of the date of this report.

c) Provide evidence to HDC, within six months of the date of this report, of the further education he has undertaken since the time of these events, including:

i. His involvement in the GPEP programme;

ii. His research into neutropenic fever syndrome, hypotension and shock in adults, acute decompensated heart failure, and sepsis.

105. I recommend that Dr C:

a) Provide a written apology to Mr A's family. The apology is to be sent to HDC within three weeks of the date of this report, for forwarding to Mr A's family.

b) Reflect on her failings in this case and provide a written report to HDC on her reflections and the changes to her practice she has instigated as a result of this case, within six months of the date of this report.

106. I recommend that the health centre:

a) Provide a written apology to Mr A's family. The apology is to be sent to HDC within three weeks of the date of this report, for forwarding to Mr A's family.

b) Review the effectiveness of its:

i. "Nurse Initiated Sepsis Pathway/Sepsis Triage Flow Chart"; and

ii. "Early Warning Score System" in the patient observation chart;

and report to HDC on the outcome of the review, within six months of the date of this report.

c) Report to HDC on the outcome of its audit of the time taken from assessment/ provisional diagnosis of sepsis to antibiotics being administered, within six months of the date of this report.

Follow-up actions

107. A copy of this report with details identifying the parties removed, except the expert who advised on this case, will be sent to the Medical Council of New Zealand and the Royal New Zealand College of General Practitioners, and they will be advised of Dr B's name.

108. A copy of this report with details identifying the parties removed, except the expert who advised on this case, will be sent to the Health Quality & Safety Commission and the district health board, and they will be advised of the health centre's name.

109. A copy of this report with details identifying the parties removed, except the expert who advised on this case, will be 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 Sarah Clarke, a rural medicine specialist:

"My name is Dr Sarah Leanne Clarke and I have been asked to provide an opinion to the Commissioner on case number C17HDC01683. I have read and agree to follow the HDC's guidelines for independent advisors. I am a registered medical practitioner with vocational registration in the scopes of Urgent Care and Rural Hospital Medicine. I have worked in Rural Hospital Medicine for approximately 9 years including over four years of experience in rural hospital clinical leadership.

I have been instructed by the Commissioner to provide my advice as to whether the care provided to [Mr A] by [Dr B] on and around 07 [Month4] was reasonable in the circumstance, and why.

In particular, I have been asked to comment on:

1. [Dr B's] omission to request a blood culture when [Mr A] developed a fever on 7 [Month4].
2. The reasonableness of [Dr B's] decision not to commence antibiotics on 7 [Month4].
3. Any other matters in this case that I consider warrant comment.

For each question, I have been asked to 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 a departure do I consider this to be?

c. How would it be viewed by my peers?

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

[Mr A] was transferred to [the health centre] on 27 [Month3] from [the public hospital] for rehabilitation following a lengthy admission with two major abdominal operations. His medical history included type 2 diabetes, vasculitis, hypertension, hyperlipidaemia, hypothyroidism, chronic renal failure and with myositis and rapidly progressive glomerulonephritis lupus for which he was taking immunosuppressive medications.

On the evening of 6 [Month4] observations were recorded as 'BP 172/90, RR 18, Temp 36.3, O2 sats 99% on air, BGL 7.1 mol/l'. On 7 [Month4] at 0130 hrs [Mr A] is reported to have vomited and his observations were recorded as 'BP 170/100, HR 120 irreg, Temp 37.7, RR 28, BGL 12.5mmol/l, O2 sats 86%'. The notes state 'MO informed' who ordered 'Codeine Phos 15mg, Cyclizine 25mg'. Further notes state that the medications were given with 'some effect' and no further observations were recorded in the nursing note. A urine sample was collected which is recorded as having 'Leukocytes present' and was sent for microscopy, culture and sensitivity.

At 0950 when [Dr B] reviewed [Mr A] he had shortness of breath on exertion and had vomited earlier in the day, though he was able to speak and not febrile. His observations were noted as 'T 37.3, sat 86% RA, RR 28' and there were mild crepitations on the right lower lobe with normal air entry. [Dr B] formulated a differential diagnosis of 'aspiration pneumonia, congestive heart failure and UTI' and diagnosed mild lung congestion after a chest x-ray revealed mild congestion and no obvious consolidation. He gave instructions to refrain from giving further subcutaneous fluid, raise the bed and provide oxygen, and requested blood tests. He decided not to give intravenous frusemide due to abnormal kidney function. Later that day a nurse informed [Dr B] of [Mr A's] blood test results including CRP 76 and creatinine 456 and that he had a temperature of 38 degrees. [Dr B] decided not to start antibiotics immediately as [Mr A] was not neutropenic (with total white blood count 2.0 and neutrophil 1.6).

When [Dr B] reviewed [Mr A] on the morning of 8 [Month4] [Mr A] was alert with no fever and saturation 92% on 1L/min oxygen. During a verbal handover [Dr B] asked [Dr C] to repeat blood tests that day and advised that she may need to start antibiotics if there was neutropenia or a positive urine culture (the result of which was pending).

[Dr C] examined [Mr A] and found he had rapid breathing and crackles in the left base. Her impression was a chest infection rapidly evolving in an immunocompromised patient with multiple comorbidities. [Dr C] began antibiotic treatment with piperacillin and tazobactam following a telephone discussion with the infectious disease consultant. She also prescribed trimethoprim for [Mr A's] urinary tract infection. [Mr A's] urine culture results showed an infection with Enterobacter and [Dr C] replaced the trimethoprim with ciprofloxacin as [Mr A] was unable to swallow the trimethoprim. Unfortunately, [Mr A] continued to deteriorate entering a comatose state and dying later that day. A blood culture performed on 8 [Month4] and available post mortem showed a growth of Klebsiella pneumoniae.

[Dr B's] omission to request a blood culture when [Mr A] developed a fever on 7 [Month4].

Indications for blood culture include:

1. Clinical features of sepsis including tachycardia, tachypnoea, increased or subnormal temperature and change in sensorium, hypotension or prostration
2. Suspicion of infective endocarditis
3. Pyrexia of unknown origin
4. Unexplained leukocytosis or leukopenia
5. Systemic and localised infections including suspected meningitis, osteomyelitis, septic arthritis, acute untreated bacterial pneumonia or other possible bacterial infection (Ntusi, Aubin, Oliver, Whitelaw, & Mendelson, 2010)

At the time that [Mr A] developed a temperature of 38 his other observations were recorded as blood pressure 129/75, heart rate 103, respiratory rate 28 and oxygen saturations of 85% on 1 litre/minute of supplementary oxygen. As such he had many

of the above criteria for clinical features of sepsis including at least tachycardia, tachypnoea and increased temperature, and he was known to have leukopenia with likely 'possible bacterial infection' based on a known leukocytosis in the urine and an abnormal chest x-ray with hypoxia, tachypnoea and unilateral clinical chest findings that could be consistent with pneumonia. In this situation the accepted practice would be to collect blood for culture. Failure to do so was a moderate departure from the standard and this would be viewed by my peers as unreasonable. The departure is considered moderate because the collection of blood for culture per se does not immediately impact upon the treatment of the patient's condition as positive results are often not available for many hours to days, though may direct antimicrobial therapy once available.

I recommend that a nurse-initiated Sepsis Pathway be instituted at [the health centre] for use at presentation and on the ward when there is a change in clinical status, and that it include guidance as to when blood should be drawn for culture, to help to prevent a similar occurrence in future.

The reasonableness of [Dr B's] decision not to commence antibiotics on 7 [Month4].

Sepsis is a clinical syndrome characterised by systemic inflammation due to infection with mortality estimated to be upward of 10 percent. (Schmidt & Mandell, 2018). In early 2012 the standard diagnostic criteria for sepsis, as defined by the Surviving Sepsis Campaign Guidelines Subcommittee, was documented or suspected infection with some of a list of variables as seen in Table 1. A 2016 taskforce was set to better define sepsis suggesting that the 2012 guidelines were in effect at the time of the event in question.

TABLE 1. Diagnostic Criteria for Sepsis

Infection, documented or suspected, and some of the following:
General variables
Fever (> 38.3°C)
Hypothermia (core temperature < 36°C)
Heart rate > 90/min^{-1} or more than two SD above the normal value for age
Tachypnea
Altered mental status
Significant edema or positive fluid balance (> 20 mL/kg over 24 hr)
Hyperglycemia (plasma glucose > 140 mg/dL or 7.7 mmol/L) in the absence of diabetes
Inflammatory variables
Leukocytosis (WBC count > 12,000 μL^{-1})
Leukopenia (WBC count < 4000 μL^{-1})
Normal WBC count with greater than 10% immature forms
Plasma C-reactive protein more than two SD above the normal value
Plasma procalcitonin more than two SD above the normal value
Hemodynamic variables
Arterial hypotension (SBP < 90 mm Hg, MAP < 70 mm Hg, or an SBP decrease > 40 mm Hg in adults or less than two SD below normal for age)
Organ dysfunction variables
Arterial hypoxemia (Pao_2/Fio_2 < 300)
Acute oliguria (urine output < 0.5 mL/kg/hr for at least 2 hrs despite adequate fluid resuscitation)
Creatinine increase > 0.5 mg/dL or 44.2 μmol/L
Coagulation abnormalities (INR > 1.5 or aPTT > 60 s)
Ileus (absent bowel sounds)
Thrombocytopenia (platelet count < 100,000 μL^{-1})
Hyperbilirubinemia (plasma total bilirubin > 4 mg/dL or 70 μmol/L)
Tissue perfusion variables
Hyperlactatemia (> 1 mmol/L)
Decreased capillary refill or mottling

WBC = white blood cell; SBP = systolic blood pressure; MAP = mean arterial pressure; INR = international normalized ratio; aPTT = activated partial thromboplastin time.

Diagnostic criteria for sepsis in the pediatric population are signs and symptoms of inflammation plus infection with hyper- or hypothermia (rectal temperature > 38.5° or < 35°C), tachycardia (may be absent in hypothermic patients), and at least one of the following indications of altered organ function: altered mental status, hypoxemia, increased serum lactate level, or bounding pulses.

Adapted from Levy MM, Fink MP, Marshall JC, et al: 2001 SCCM/ESICM/ACCP/ATS/SIS International Sepsis Definitions Conference. *Crit Care Med* 2003; 31: 1250–1256.

Reproduced from *Surviving Sepsis Campaign: International Guidelines for Management of Severe Sepsis and Septic Shock: 2012* (Dellinger, et al., 2013)

At 0950 when [Dr B] reviewed [Mr A] his observations were 'T 37.3, sat 86% RA, RR 28' and [Mr A] was noted as having mild crepitations on the right lower lobe with normal air entry. A urine sample collected overnight was recorded as having a preliminary result of 'Leukocytes present' which is suggestive of possible infection. [Dr B] gave a differential diagnosis of 'aspiration pneumonia, congestive heart failure and UTI' and later concluded there was 'mild congestion' based on his interpretation of the chest x-ray. [Mr A] demonstrated tachypnoea and it is also likely that, if measured, he would have had arterial hypoxaemia (based on saturations of 86% on room air). His heart rate is not reported in the ward round note. If the heart rate was above 90 at that time then he would have met the criteria of sepsis being 'infection, documented or suspected, and some' of the variables described above and as such antibiotics should have been prescribed. If the heart rate was below 90, and accepting that arterial oxygenation was not measured, then the criteria for sepsis was not definitely met at

that time and as such it may be considered reasonable not to prescribe antibiotics at this time while awaiting further results.

During the evening shift sometime between 1900 and 2400hrs [Mr A's] observations were recorded as 'T 38, BP 129/75, HR 103, RR 28' and 'O2 sats 85% on 1 litre O2 via NP'. [Dr B] was contacted with these observations and was advised of the blood results from earlier that day. At this time [Mr A] certainly met the above criteria for sepsis namely he had suspected infection (urine, possibly chest), tachypnoea, tachycardia, likely hypoxaemia, leukopenia, elevated C-reactive protein and a decrease of >40mmHg systolic blood pressure compared to his pre-deterioration BP (172 to 129), the latter of which would further characterise [Mr A] as having severe sepsis (Dellinger, et al., 2013). Accepted practice in the setting of sepsis is to prescribe antibiotics (Dellinger, et al., 2013). Failure to identify sepsis and/or to prescribe antibiotics at this time represents a severe departure from the standard of care and would be seen by a group of my peers as unacceptable.

[Dr B] states that 'It was suggested by the renal team that the AKI could be due to antibiotics'. The reference in the discharge summary from [the public hospital] is to trimethoprim, an antibiotic which is known to cause and/or potentiate kidney injury (Fraser, Avellaneda, Graviss, & Musher, 2012), and would not preclude the use of other non-nephrotoxic antibiotic therapies and as such need not have been a consideration in the decision of whether or not to commence antibiotic therapy.

I would recommend that a nurse-initiated Sepsis Pathway be instituted at [the health centre] for use at presentation and on the ward when there is a change in clinical status to help to prevent a similar occurrence in future. I would also recommend that an empiric antibiotic protocol be included in this pathway with an indication of where to get specific advice should the antimicrobial agents recommended in the protocol be either contra-indicated or unavailable in the rural setting.

Any other matters in this case that I consider warrant comment.

Decision to not review [Mr A] in person at the time of initial deterioration.

[Dr B] was made aware of [Mr A's] deteriorating status including tachypnoea, tachycardia and significant hypoxia, at 0130 on 07 [Month4] and managed this deterioration by telephone with oral medications. Further observations are not recorded in the nursing note for that shift. Had [Mr A] improved with the therapy given by phone and his heart rate, respiratory rate and hypoxia returned to normal levels then it may be reasonable that [Dr B] did not review [Mr A] in person at that time. If, however, [Mr A] remained tachycardic, tachypnoeic and hypoxic despite the therapies instituted by telephone order (which seems likely given his next reported set of observations) then standard of care would be to review the patient in person at that time. Failure to review a patient with such a significant deterioration from baseline and lack of response to initial therapy would be considered a severe departure from the standard of care and would be considered significantly below expectations by a group of my peers.

I recommend that, in addition to a nurse-initiated Sepsis Pathway, an early warning score escalation policy be customised and introduced to [the health centre] to help prevent a similar event happening in future.

[Dr B's] interpretation of the chest x-ray.

[Dr B's] interpretation of the chest x-ray was of 'mild congestion' where-as the later formal report stated 'There is a little patchy change at the left lung base' and the post mortem examination revealed resolving and acute aspiration pneumonia, both suggesting that [Dr B's] initial interpretation of the chest x-ray was incorrect. Interpretation of investigations must be performed with consideration as to the clinical context. At the time that chest x-ray was performed [Mr A] had suffered an acute deterioration from the night before of his respiratory rate (18 to 28) and his oxygen saturation (99% on room air to 86% on room air) which are both suggestive of an acute respiratory compromise. At that time, given the context that [Mr A] had not developed a fever, this misreading of the chest x-ray represents a mild departure from standard of care, and would be considered a reasonable error of judgement by my peers. At the time that [Mr A] developed a fever and the blood test results were available showing a leukopenia and elevation of C-reactive protein [Dr B] should have had a high index of suspicion that the changes he'd seen on the chest x-ray and described as 'congestion' may have represented infection, and as such his interpretation that 'there was no significant consolidation on the chest x-ray to represent pneumonia' would represent a moderate departure from standard of care and the decision to 'rule out' respiratory infection based on the x-ray interpretation would be viewed as unreasonable by a group of my peers. I recommend that a Sepsis Pathway be established at [the health centre] and that this include a checklist as to possible sources of sepsis to act as a reminder to clinicians to consider all findings in the context of possible sepsis, and to help prevent such an occurrence from happening again in future.

The choice of Trimethoprim to treat the UTI.

Pneumocystis Pneumonia (PCP) is ordinarily treated with Trimethoprim-Sulfamethoxazole and requires a renal dose adjustment in patients with renal insufficiency due to the potentially nephrotoxic trimethoprim component (Thomas & Limper, 2018). [Dr C] chose to treat with antimicrobials after discussion with the infectious disease specialist (ID). She also noted [Mr A] to have an Enterobacter urinary tract infection, although it is not clear whether this was also discussed with ID. Enterobacter species can be multi-drug resistant due to many mechanisms (Chambers, Eliopoulos, Gilbert, & Saag, 29). While targeted therapy based on the culture and sensitivity could have included trimethoprim or ciprofloxacin, [Dr C] had been advised by the ID physician not to cover PCP due to the renal function, implying that the ID physician felt that trimethoprim was relatively contraindicated in the context of [Mr A's] renal status. To prescribe trimethoprim in the context of renal failure and ID advice to not give trimethoprim/sulfamethoxazole without consideration to safer alternatives or renal dosing would be considered a moderate departure from accepted care and would be seen as illogical by a group of my peers. I would recommend that a Sepsis Pathway with reference to empiric antibiotic therapy, including relevant contraindications and precautions, be instituted at [the health centre] and that

deviation from this pathway in the context of sepsis should require specific discussion with ID to help prevent a further such occurrence happening in future.

Not increasing the steroid dose after the identification of sepsis.

[Mr A] had been on long term steroid therapy with prednisone and had received a dose reduction shortly before his deterioration to 5mg daily and prior to this he had been on 10mg daily for at least the duration of the admission. It is noted by a nurse that the prednisone was withheld after the deterioration as [Mr A] was not able to swallow this. People who take long term steroids are at risk of adrenal insufficiency in the event of developing sepsis and as such their steroid dose needs to be increased to mitigate this risk in the setting of sepsis. I note that there was not an increase in steroid dose for 'stress cover' once sepsis was recognised by [Dr C]. Standard of care would be to increase the steroid cover in long-term steroid exposed patients in the context of sepsis, particularly in the setting of relative hypotension (Dellinger, et al., 2013). Failure to do so would be considered a moderate departure from standard of care and would be considered unreasonable by a group of my peers. I would recommend that a nurse-initiated Sepsis Pathway be instituted at [the health centre] for use at presentation and on the ward when there is a change in clinical status, and that this pathway include a reminder as to 'stress cover' steroid dosing to help to prevent a similar occurrence in future.

Signed Dr Sarah Leanne Clarke"

The following further expert advice was obtained from Dr Clarke:

"My name is Dr Sarah Leanne Clarke and I have been asked to provide an opinion to the Commissioner on case number C17HDC01683. I have read and agree to follow the HDC's guidelines for independent advisors. I am a registered medical practitioner with vocational registration in the scopes of Urgent Care and Rural Hospital Medicine. I have worked in Rural Hospital Medicine for approximately 9 years including over four years of experience in rural hospital clinical leadership.

I have been instructed at this time by the Commissioner to provide a response upon review of [the health centre's], [Dr C's], and [Dr B's] responses to my previous advice (including their annexures) as to whether the information or submissions in these responses changed my original advice in any way, or raised any new issues.

Response to [the health centre's] response

I am pleased to see that [the health centre] has acted upon my recommendations and has developed and instituted the use of both a nurse-initiated sepsis pathway and an early warning system. The provision of an antimicrobial guideline and the clarification of when to access Infectious Disease advice will also be of value. I have no doubt that these initiatives will be of benefit to future patients and will significantly reduce the likelihood of an event such as [Mr A's] occurring again. The education that has been provided to staff will ensure that these initiatives are used appropriately and the audit will ensure that these improvements are maintained. I am satisfied that this response

from [the health centre] will mitigate the risk that unrecognised sepsis poses to patients. I also note that [the health centre] has an education programme that includes both medical and nursing staff. [Dr B's] comment of being 'focussed on' the diagnosis of heart failure has highlighted the constant risk of conformational bias in clinical decision making, being a risk that is present in all levels of medical care and in all facets of medicine. It is likely that in [Mr A's] case that all staff decisions may have been subject to an element of conformational bias and, in light of this new insight from [Dr B], I additionally recommend that [the health centre] introduce the topic of 'conformational bias' into its staff education curriculum moving forward, at least for medical and nursing staff and noting that this may also be beneficial to a wider staff audience. This recommendation comes in response to [Dr B's] comment which highlights an area of risk in all of medicine that has not yet been mitigated in many areas of healthcare. I believe that it is fortunate that it has been highlighted in this case and I hope that this be viewed as a positive opportunity moving forward.

Response to [Dr C's] response

1. I recognise that the treatment dose of trimethoprim for Pneumocystis pneumonia is higher than the dose prescribed by [Dr C] for the treatment of UTI. While I recognise that the Medsafe datasheet indicates that there need be no dose reduction for a patient with an eGFR of 10 or greater, [Mr A] was exactly on the borderline for this recommendation, clinically deteriorating (meaning his eGFR might drop further), and had a previous history of acute kidney injury while on trimethoprim for UTI. In the context of such a complex patient, with a history of previous acute kidney injury while on trimethoprim, I believe that the decision to prescribe trimethoprim in this context (and particularly in view of a potentially safer effective option being available, namely ciprofloxacin) would be one that most of my colleagues would have shared with either a Renal Physician or an Infectious Disease Specialist. As to the suggestion of performing serum trimethoprim levels after three days — this is not a test that I have ever seen performed in clinical practice, including during my many years of urban and provincial hospital practice, and I doubt whether it is a test that could be performed at [the health centre] in a manner that a result would be available within a clinically relevant timeframe. It would therefore be outside my scope to comment as to how this test result may have impacted upon the relative safety of the prescription of trimethoprim in this context.

2. Wass and Arlt (2012) state that 'adrenal crisis can occur in any patient treated with 5 mg or more of prednisolone (equivalent to 20 mg of hydrocortisone orally) for more than four weeks'. As [Dr C] stated, she was aware that [Mr A] was on a dose of 5mg prednisone per day, which was long term therapy and given the diagnosis of systemic lupus erythematosus (SLE) with rapidly progressing glomerulonephritis (RPGN). Given this knowledge, [Mr A] should have received an increased dose of steroid when [Dr C] made the diagnosis of sepsis. [Mr A's] missed dose the prior day was not relevant to the decision not to provide a stress dose of steroid in the context of sepsis. [Mr A's] inability to swallow a tablet was also of no relevance to this decision as hydrocortisone is an acceptable alternative to prednisone in this context and can be delivered intravenously. I accept that if a palliative pathway had been initiated that it would be reasonable to withhold the steroid stress dose, but in the context that

intravenous antibiotics were still being continued it seems that a decision to withdraw active care had not been made at the time of [Mr A's] death, although a ceiling of care excluding transfer to [the public hospital] or resuscitation had been agreed upon. As such, I continue to maintain that the failure to provide stress dose steroid to [Mr A] to be a moderate departure from standard care.

Response to [Dr B's] response

A. [Dr B] accepts his error.

B. I agree with both [Dr B] and his specialist colleague that starting empiric antibiotics when [Mr A] developed fever would have been a better choice.

During the evening of 07 [Month4] [Mr A] had his observations recorded as 'T38, BP 129/75, HR103, RR 28' and 'O2 sats 85% on 1 litre O2 via NP' which constituted a significant clinical deterioration, and [Dr B] was contacted with this information along with the blood results from earlier in the day which showed an elevated C-reactive protein overall leukopenia, on a background of immunosuppression. While any one of these abnormalities (each individual observation, or each individual blood test result) can be explained by alternative reasoning, the constellation of clinical signs and biochemical markers together points to a picture of sepsis, a diagnosis that should have been in the forefront of [Dr B's] mind in this scenario and one that should have been emergently acted upon. Any current New Zealand sepsis pathway would support my view, as does the evidence referenced in my previous opinion.

As stated in my original opinion, I do not accept the concern regarding nephrotoxicity of antibiotics as a valid reason to withhold antibiotics in a patient with renal failure who meets sepsis criteria as options that are safe for this situation do exist. I also do not accept a concern regarding bowel obstruction to be relevant to withholding antibiotics in this context.

While I accept that heart failure and sepsis can co-exist in a patient, the relationship between heart failure and other morbidities is complex and the overarching issue in this case is that [Dr B] was 'focussed on' a diagnosis of heart failure and failed to identify sepsis as a critical part of [Mr A's] deterioration. [Dr B] did not make the diagnosis of sepsis, and neither did he act upon his own suspicion of infection as described in his differential diagnosis that included UTI and aspiration pneumonia. I maintain that failure to prescribe antibiotics in this case was a severe departure from standard care. I acknowledge that the use of a sepsis pathway may have reduced the risk of this erroneous decision being made, and as such that the institution of such pathway is likely to prevent this event happening again in future.

C. Many of New Zealand's rural hospitals operate an on call medical roster where the doctor available for the medical care of the patients overnight is offsite and asleep unless required. As such, accepted standards of care vary from one hospital to another, and are influenced by many factors including (but not limited to): nursing experience; medical experience; the relationships and familiarity between the nursing and medical staff; rostering patterns. Ultimately, the important thing is that the patient receives the right care at the right time. It is common for a rural hospital

doctor to provide initial treatment by phone, caveated with an expectation that the doctor will be contacted again should that therapy not be effective within an agreed timeframe. [Mr A's] deterioration on the evening of 07 [Month4] was severe and thus should have been considered serious and urgent. As stated in my original advice 'Had [Mr A] improved with the therapy given by phone and his heart rate, respiratory rate and hypoxia returned to normal levels then it may be reasonable that [Dr B] did not review [Mr A] in person at that time.' It is not clear from the information provided exactly how [Mr A] responded to the therapy prescribed over the telephone as there does not appear to have been timely repeat observations recorded other than 'Pt reported he was comfortable. O2 sats improved. O2 left on at 1L/min O2'. I note that this requirement for oxygen was a significant deterioration to the saturations recorded as 99% on room air on 06 [Month4]. As stated in my original advice, 'If, however, [Mr A] remained tachycardic, tachypnoeic and hypoxic despite the therapies instituted by telephone order … then standard of care would be to review the patient in person at that time. Failure to review a patient with such a significant deterioration from baseline *and lack of response to initial therapy* would be considered a severe departure from the standard of care and would be considered significantly below expectations by a group of my peers'. I accept that either scenario may have been the case, and that the clinical notes do not include enough information for me to conclude whether [Mr A] had, or had not, responded adequately to the therapy prescribed over the telephone. As such, I stand by my original opinion.

D. Tests are performed to add information to a diagnostic picture and should not be interpreted in isolation. [Dr B's] interpretation of the chest xray as showing 'mild congestion' indicates that he recognised that the xray was not normal, and in considering this within the clinical context of tachypnoea and hypoxia it is reasonable to consider this abnormality to be related to heart failure, while infection should still have remained on the differential. It is also true to say that a normal chest xray cannot rule out infection (or other pathology) in the chest. Once [Mr A] had developed fever, elevated C-reactive protein, and leukopenia, along with the tachycardia, tachypnoea and hypoxia, then this abnormality noted on the chest xray should have been reconsidered within this context and I maintain that failure to reconsider this remains a departure from the standard of care.

I am pleased that [Dr B] has taken this opportunity to refresh his knowledge in the areas of sepsis, neutropenic fever, hypotension, shock and heart failure, and I am sure that this additional learning will benefit his future patients.

Signed Dr Sarah Leanne Clarke"

The following further expert advice was obtained from Dr Clarke:

"I have reviewed the attached documents provided, and [the health centre's] response, and this does not cause me to change any of my advice.

Sarah"