CORONER'S COURT
OF NEW SOUTH WALES

Inquest:	Inquest into the death of Harriet Ostler
Hearing dates:	18 to 20 April 2023 at Port Macquarie
Date of Findings:	9 May 2023
Place of Findings:	Coroner's Court of New South Wales, Lidcombe
Findings of:	Magistrate Derek Lee, Deputy State Coroner
Catchwords:	CORONIAL LAW – cause and manner of death, cardiomyopathy, myocarditis, recognition of clinically deteriorating paediatric patient, vital signs observations, Between the Flags, escalation of care, x-ray interpretation, pulmonary consolidation, blood gas, medical review
File number:	2016/315785
Representation:	Ms D Ward SC, Counsel Assisting, instructed by Ms C Moore (Department of Communities & Justice Legal) Mr B Bradley for Mid North Coast Local Health District, instructed by Crown Solicitor's Office Mr N Dawson for RN M Dalton & RN K Johnston Mr R Sergi for Dr R Dixit, instructed by Wotton + Kearney

Findings:

Harriet Ostler died on 22 October 2016 at Port Macquarie Base Hospital, Port Macquarie NSW 2444.

The cause of Harriet's death was sudden cardiac death on a background of rhinovirus infection.

Harriet died of natural causes whilst an inpatient at Port Macquarie Base Hospital. Although Harriet was treated for suspected viral respiratory illness she had significant underlying cardiac pathology which was not recognised. If this had been recognised it is likely that changes would have been made to Harriet's treatment and her care escalated. However, it is unclear whether such escalation, if it had occurred, would have averted the eventual outcome given the sudden and unexpected nature of Harriet's clinical deterioration leading to cardiac arrest.

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1. Introduction

1.1 Harriet Ostler, a much-loved and full-of-life 19 month old toddler, was admitted to Port Macquarie Base Hospital on 21 October 2016. Harriet had been unwell in the days leading up to this admission and after arriving at hospital it was thought she was suffering from a viral respiratory illness. Harriet was provided with treatment and remained at hospital overnight. She showed signs of improvement but also other signs which were concerning. At around 5:45am on 22 October 2016, Harriet was found to be not breathing. Although resuscitation efforts were commenced immediately, Harriet could not be revived and was tragically later pronounced deceased, only approximately 20 hours after she was admitted to hospital.

2. Why was an inquest held?

2.1 Under the *Coroners Act 2009* (**the Act**) a Coroner has the responsibility to investigate all reportable deaths. This investigation is conducted primarily so that a Coroner can answer questions that are required to be answered pursuant to the Act, namely: the identity of the person who died, when and where they died, and what was the cause and the manner of that person's death.

2.2 The rapid deterioration of Harriet's condition on 22 October 2016 raised immediate questions about the cause of her death. Further, the subsequent findings from the post-mortem examination raised additional questions for consideration regarding this issue. Finally, the manner of Harriet's death, or in other words the circumstances surrounding the events of 21 and 22 October 2016, were not entirely clear. That is, given Harriet's relatively brief admission to hospital, which culminated in her tragic death, a legitimate question arose as to whether the care and treatment provided to her was appropriate having regard to what information was available to the clinicians at the relevant time. For all of these reasons, an inquest was required to be held.

2.3 In this context it should be recognised at the outset that the operation of the Act, and the coronial process in general, represents an intrusion by the State into what is usually one of the most traumatic events in the lives of family members who have lost a loved one. At such times, it is reasonably expected that families will want to grieve and attempt to cope with their enormous loss in private. That grieving and loss does not diminish significantly over time. Therefore, it should be acknowledged that the coronial process and an inquest by their very nature unfortunately compels a family to re-live distressing memories several years after the trauma experienced as a result of a death, and to do so in a public forum. This is an entirely uncommon, and usually foreign, experience for families who have lost a loved one.

2.4 It should also be recognised that for deaths which result in an inquest being held, the coronial process is often a lengthy one. The impact that such a process has on family members who have many unanswered questions regarding the circumstances in which a loved one has died cannot be overstated. Regrettably in Harriet's case, this process has been particularly prolonged due in part to the impact of the COVID-19 pandemic and challenges associated with identifying a suitable and available venue and period of time for the inquest to be held.

3. Harriet's life

3.1 Inquests and the coronial process are as much about life as they are about death. A coronial system exists because we, as a community, recognise the fragility of human life and value enormously the preciousness of it. Understanding the impact that the death of a person has had on those closest to that person only comes from knowing something of that person's life. Therefore, it is important to recognise and acknowledge the life of that person in a brief, but hopefully meaningful, way.

3.2 Harriet was born on 29 March 2015 to Jessica (Jess) Zycki and Robert (Bob) Ostler. At the time she was the youngest of three children, and had two older brothers, Milton and Leeroy, who adored her.

3.3 Harriet was a well-contented baby, only crying when it was time for a feed. She began crawling at 8 months and 2 months later she started walking, trying to keep up with her brothers. Jess and Bob have many fond and lasting memories of their three children at this young age: Harriet trying to help Jess with everything; Harriet helpfully getting her brothers' pyjamas and slippers for them at bath time, only to mischievously then run away while they chased after her.

3.4 Harriet had a love for the outdoors and enjoyed going for walks with Jess and her brothers. On the weekend, the whole family would go bush walking together or exploring a creek near their property. She spent many wonderful hours playing with her brothers on the trampoline, on the swings or in their cubby house.

3.5 Harriet's brothers have many precious memories of their little sister: being pushed around outside in their pink car by their parents, the boys pushing Harriet around in her doll's pram, Harriet getting her hair tangled in one of Leeroy's motorised toy trains and then hilariously walking around with it on her head.

3.6 Harriet loved to help feed the chickens on the family property and collect the eggs from their pen. One day, Harriet made her own way to the pen, collected all the eggs by herself and returned to the home with a big smile on her face. Harriet also had a great fondness for the family dog (Digger) and cat (Barney) and would often lie down or sit on them.

3.7 Harriet was known to have her own little daily routine. She wasn't happy until she had her morning cup of tea with mummy and daddy. Then, in the afternoon Harriet would wait on the patio for Bob to come home from work. She would patiently wait for Bob to take off his boots and then bring his work esky inside so that she could unpack it, eagerly anticipating any treats that might be waiting inside for her.

3.8 Apart from her family and pets, Harriet also had a special place in her heart for her favourite teddy bear, "Ted Ted". Harriet took him everywhere with her, always having him tucked under one arm. The two were inseparable, so much so that when it was time for Ted Ted to have a wash, Harriet would sit under the washing line and cry for him.

3.9 Since 2016, Jess and Bob have had three more children: Dulcie, Clancy and Winston. It is heartbreaking to know that they never got to meet their older sister.

3.10 There is no doubt that Harriet's parents, siblings, maternal grandparents, Jules and Debbie, and other extended family members all miss her enormously. The absence of such a special and wonderful little girl simply cannot be filled. However, Harriet remains very much in their hearts and memories today and always.

4. Background to the events of 21 & 22 October 2016

4.1 Harriet was born by normal delivery at 39 weeks gestation. She was noted to be small for her gestational age with a lower birthweight than expected. After developing hypothermia, Harriet was treated with antibiotics for presumed sepsis in the special care nursery, but was otherwise well. After leaving hospital, Harriet developed normally and did not have any significant medical conditions.

4.2 On 18 October 2016, Harriet developed a runny nose but no other symptoms. She developed a cough the next day. By 20 October 2016, Harriet had an elevated body temperature and was noted to be crying and upset, which prompted Jess to return home early from Wauchope. Harriet was given some nurofen and put to bed.

4.3 The evening of 20 October 2016 was uneventful but the following morning Harriet was noted to be lethargic and to have a dry nappy, which was unusual. Jess took Harriet to the emergency department at Wauchope Urgent Care Centre (**WUCC**). On examination, Harriet was noted to be pale, lethargic, floppy, very unsettled, not taking her feeds and feeling hotter than her body temperature reading (which was normal). Clinicians formulated a working diagnosis of probable viral gastroenteritis but it was considered that there was a need to rule out possible meningitis and urinary tract infection. Harriet was given intravenous fluids and a broad spectrum antibiotic. Arrangements were later made to transfer Harriet to Port Macquarie Base Hospital (**PMBH**) for further investigations.

4.4 Harriet was admitted to the PMBH emergency department at around 10:25am. On arrival she was noted to be peripherally cool, distressed and not her normal self. On examination, Harriet was noted to be afebrile and tachypnoeic, with a respiratory rate (**RR**) of 40 breaths per minute, heart rate (**HR**) of 130 beats per minute and to have oxygen saturation of 97% on room air. Harriet's chest was found to be clear with occasional granting but no wheezes or crepitations. A clinical impression of asthma was noted, and Harriet was given salbutamol and scheduled for review by the paediatric team.

4.5 Harriet was given further puffs of salbutamol throughout the morning and urine specimens were collected and sent to the laboratory for analysis.

4.6 Sometime prior to 1:00pm Harriet was reviewed by Dr Rashmi Dixit, the locum paediatrician. On examination, Harriet was noted to be quiet, tired and crying but alert with good tone. Her vital signs were recorded which revealed that she was afebrile, tachypnoeic, with oxygen saturation of 97% on room air, a RR of 40 and a HR of 130. Harriet was also found to have intercostal recession, sub-costal recession, reasonable air entry, vesicular breath sounds and diffuse expiratory wheeze. Dr Dixit considered that her findings were consistent with a viral bronchiolitis or asthma. As Harriet had a history of reduced urinary output as a result of a reduction in her usual oral intake, Dr Dixit also started Harriet on intravenous fluids.

4.7 By around 1:40pm, Harriet was noted to have intercostal recession, increased work of breathing and use of accessory muscles. Her RR was noted to be 45 with 100% oxygen saturations. Notwithstanding, Harriet appeared lethargic and she was given further puffs of salbutamol.

4.8 At around 3:20pm, Registered Nurse (**RN**) Kim Wight performed a top-to-toe assessment of Harriet. She was found to be warm, pink, well-perfused and neurologically appropriate. Harriet was transferred to the children's ward and was later observed to appear to be tired and attempting to go to sleep. Her vital sign observations were found to be stable but it was noted that Harriet had mild work of breathing and slight audible wheeze.

4.9 Dr Dixit reviewed Harriet again at around 4:00pm. On examination, Harriet was found to have oxygen saturations of 98% on room air, RR of 60 and pulse rate (**PR**) of 140. Dr Dixit formed an impression of asthma/bronchiolitis but it was noted that Harriet's cardiovascular and respiratory status was good. Salbutamol was charted for Harriet.

4.10 During this time, Dr Dixit spoke with Jess who requested that a chest x-ray (**CXR)** be performed. However, Dr Dixit explained that with a working diagnosis of viral respiratory tract infection, and in the absence of any deterioration in Harriet's presentation, a CXR was not indicated. Dr Dixit also explained that a CXR did not have much diagnostic value for a child with asthma and/or bronchiolitis. Ultimately, Dr Dixit formed the view that the potential benefit to Harriet was outweighed by the potential risk (exposure to radiation).

4.11 Between 5:00pm and 7:00pm, RN Wright repeated observations for Harriet, who was found to be eating and drinking, interacting with her family, making good eye contact and using words/speech that was age-appropriate. At Jess's request, RN Wright gave Harriet some Panadol and she was rocked to sleep by her mother.

4.12 At around 8:20pm, RN Wright checked on Harriet and found that she was asleep, with a sudden increase in RR and work of breathing, with decreased oxygen saturations. RN Wright discussed Harriet's clinical change with Jess, and told her that she was going to give Harriet salbutamol, apply oxygen and call Dr Dixit (who by this time had left the hospital but remained on call) to review Harriet.

4.13 At around 9:17pm, Dr Dixit reviewed Harriet. She was found to have decreased oxygen saturations (89-90%), marked increase in work of breathing with grunt and chest recessions. Dr Dixit noted that the grunting and chest recessions appeared to improve significantly when Harriet was left alone to settle. Dr Dixit formed the impression that Harriet was suffering from a viral illness with bronchiolitis, and that she had no apparent response to administration of salbutamol. Accordingly, the orders for salbutamol and hydrocortisone as regular medications were ceased. Dr Dixit instead charted an order for salbutamol as required.

4.14 According to RN Wright, she had a discussion with Dr Dixit and recommended a CXR, queried performing a blood gas and making a call to the Newborn and Paediatric Emergency Transport Service (**NETS**) in the event that Harriet required transfer to a hospital able to provide a higher level of care. Dr Dixit considered that a CXR would make Harriet more distressed and RN Wright advised

that a mobile CXR could be performed in the ward. Shortly after RN Wright handed over Harriet's care to RN Kylie Johnston and RN Mary Dalton who were both working the night shift.

4.15 A CXR was ordered at 9:58pm and subsequently performed. At around 10:37pm, Dr Dixit reviewed the CXR report. She noted diffuse mild inflammatory infiltrate and no consolidation.

4.16 Harriet's vital signs were taken throughout the night and into the early hours of the next morning. Her RR was noted to be 76 at 11:45pm and remained at over 60 until 1:45am on 22 October 2016. Despite these measurements being outside normal limits and appearing to meet the criteria for an emergency response, no medical review or advice was sought, and no emergency response was initiated. From about 1:45am to 6:00am, Harriet's RR and HR gradually lowered and she was treated with salbutamol every hour for bilateral wheeze heard on auscultation.

4.17 At 5:45am, Harriet was given salbutamol by RN Dalton. It was noted that the flange on the spacer being used to administer salbutamol did not trigger after the last puff. As RN Dalton put the spacer down she noted that Harriet was not breathing.

4.18 RN Dalton immediately gave Harriet a sternal rub with no response and used in the emergency buzzer to call for the Medical Emergency Response Team (**MERT**). Resuscitation efforts were initiated and Harriet was ventilated with a bag and valve mask. Despite these efforts, Harriet could not be revived and was later tragically pronounced life extinct.

5. Postmortem examination

5.1 Harriet was subsequently taken to the Department of Forensic Medicine at Newcastle where a post-mortem examination was performed by Dr Jane Vuletic, forensic pathologist, on 24 October 2016. The following relevant findings were documented from this examination:

(a) a severely enlarged heart (with the weight being approximately twice that which would be expected for a child of Harriet's weight) which was structurally normal with no evidence of congenital heart disease;

(b) excess accumulation of fluid in the pleural cavities and abdominal cavity, likely to be an indication of terminal heart failure;

(c) abnormal findings on microscopic examination, including nuclear abnormalities, focal myocyte necrosis, fibroelastosis and myocyte hypertrophy;

(d) chronic inflammation in the lung; and

(e) a nasal swab showing evidence of viral infection, with microbiological testing detecting picornavirus.

5.2 In the autopsy report dated 5 May 2017, Dr Vuletic opined that it is likely that enterovirus infection in the lungs precipitated the development of heart failure. Ultimately, Dr Vuletic opined that the

cause of Harriet's death was cardiomyopathy. Dr Vuletic explained that this is a non-specific term which means a disease of the heart muscle.

5.3 Whilst cardiomyopathies may be classified in a number of ways and have a number of possible causes, Dr Vuletic noted that the appearances in Harriet's case do not fit the diagnostic criteria of three commonly recognised conditions seen in children and young persons: hypertrophic cardiomyopathy, arrhythmogenic cardiomyopathy and dilated cardiomyopathy.

6. What issues did the inquest examine?

6.1 Prior to the commencement of the inquest a list of issues was circulated amongst the sufficiently interested parties, identifying the scope of the inquest and the issues to be considered. That list identified the following issues for consideration:

(1) What was the cause of Harriet's death? Did she experience an acute myocarditis and/or a chronic cardiomyopathy and/or some other cause?

(2) Was the care provided by Port Macquarie Base Hospital reasonable in the circumstances known to Hospital staff at the time, with a particular focus upon:

(a) Dr Dixit's interpretation of Harriet's chest x-ray;

(b) The desirability of obtaining a blood gas sample;

(c) Whether there were persistent abnormalities of vital signs suggesting a deterioration in Harriet's condition at any time across her admission;

(d) If so, whether this should have prompted further medical review and/or a change in treatment?

(3) What changes have since occurred at Port Macquarie Base Hospital to better support staff in their care of paediatric patients, particularly around recognising and responding to clinical deterioration in infants?

6.2 In order to assist with consideration of some of the above issues, opinions were sought from the following independent experts:

(a) Professor Simon Craig, a paediatric emergency medicine physician at Monash Medical Centre; and

(b) Professor James Wilkinson, a senior paediatric cardiologist at the Royal Children's Hospital, Melbourne.

6.3 Both experts provided reports which were included in the brief of evidence tendered at inquest, and Professor Craig also gave evidence during the inquest

7. What was the cause of Harriet's death?

7.1 The microscopy results from the post-mortem examination were referred to Professor Mary Sheppard, a consultant in cardiovascular pathology. Professor Sheppard found:

(a) no fibrosis or information to indicate arrhythmogenic right ventricular cardiomyopathy;

(b) no evidence of myocyte disarray in sections of the left ventricle to indicate hypertrophic cardiomyopathy; and

(c) No atrophic myocytes or widespread fibrosis to indicate the updated cardiomyopathy.

7.2 Ultimately, Professor Sheppard concluded that if the toxicology results from the post-mortem examination proved to be negative (which they ultimately were), then Harriet's death represented a sudden cardiac death in a young child with an enlarged heart with normal morphology.

7.3 Professor Wilkinson noted that Harriet's deterioration during the evening of 21 October 2016 culminating in cardiac arrest is compatible with viral myocarditis. He explained that the enlarged heart with fluid collections in pleural cavities, ascites and pericardial fluid indicating acute heart failure, together with the non-specific microscopic findings, is "*fairly typical of this condition*". Professor Wilkinson also noted that myocarditis following a viral infection "*may result from a direct effect of the virus on the heart muscle or from an immunological response initiated by the viral infection*". In the first scenario, "*the histological findings are variable but there are many cases where the illness progresses very rapidly, and death may happen within a matter of hours with relatively little to find on microscopic assessment (histology) of the heart muscle*".

7.4 Ultimately, Professor Wilkinson considered the diagnosis of cardiomyopathy from the autopsy findings to be accurate, as it implied that there was a pathological process affecting the heart muscle. However, as Dr Vuletic acknowledged, a description of cardiomyopathy as the cause of death is non-specific of the cause of this pathology. Professor Wilkinson opined that the presence of pericardial, pleural and peritoneal fluid collections "*are all indicative of rapid onset of cardiac failure*" and the presence of "*a picornavirus (probably a rhinovirus) is presumably the cause of acute viral myocarditis along with viral pneumonia*".

7.5 **Conclusions:** The autopsy identified an enlarged heart due to an unidentified pathological process affecting the heart muscle. However, there was no evidence of any of the most common types of cardiomyopathies in Harriet's case. Further, there was evidence of excess accumulation of fluid in the pleural and abdominal cavities, most likely representing terminal heart failure. Dr Vuletic considered it likely that this was precipitated by enterovirus infection in the lungs. Professor Wilkinson considered that the presence of a likely rhinovirus was the cause of acute viral myocarditis together with viral pneumonia.

7.6 It is evident from the above that the autopsy findings demonstrated evidence of disease of the heart muscle without being able to establish whether this was due to a particular, chronic cardiomyopathy or whether an acute viral myocarditis precipitated the development of heart failure. As the possibility of a less common chronic cardiomyopathy cannot be entirely excluded on the available evidence, the cause of Harriet's death is best described as sudden cardiac death on a background of rhinovirus infection.

8. Dr Dixit's interpretation of the chest x-ray

8.1 Dr Dixit gave evidence that the NSW Health guidelines stipulate that a CXR is not indicated in cases where the patient presents with respiratory illness because the findings can overlap with pneumonia. She said that once Harriet started grunting she considered that a CXR was needed to make sure that Harriet had not developed a pneumothorax or had fluids that require tapping.

8.2 Once the CXR was ordered, Dr Dixit reviewed it and recorded her impressions in Harriet's progress notes at around 10:37pm. She recognised that the CXR was not ideal as it was rotated (most likely due to movement by Harriet) so that the left lung was anterior. Dr Dixit recorded the following impressions:

> Diffuse mild inflammatory infiltrate
> No consolidation

8.3 Consolidation is a term used to describe the x-ray appearance when the alveoli (tiny air sacs within the lungs) are filled with something other than air. In relation to her review of the CXR, Dr Dixit gave evidence that she:

(a) could not recall where she looked at it;

(b) acknowledged the possibility that she viewed the CXR on a terminal at the nurses' station or on a mobile x-ray machine (if that machine had such a facility);

(c) had no memory of viewing the CXR and could not recall how long she spent examining it;

(d) could not recall whether she only looked at the CXR briefly; and

(e) agreed that it was possible that she told Jess that the CXR showed asthma bronchitis and that Harriet's lungs were clear.

8.4 The CXR was later reported on by Dr Stuart Allan. The PMBH medical records indicate that the timestamp of this report was at 10:39pm, only two minutes after Dr Dixit recorded the above impressions. Dr Allan relevantly recorded the following:

> Findings: Extensive pulmonary shadowing consistent with pulmonary consolidation is seen in the right lung field particularly in the right upper lobe but also in the right lower lobe.
> Some pleural shadowing noted in the right lower zone is suggestive of a small effusion.
> Left hemidiaphragm is not defined suggesting pulmonary consolidation of the left lower lobe.
> Appearances suggest extensive pulmonary consolidation throughout both lung fields.

8.5 Notwithstanding the timestamp on Dr Allan's reporting of the CXR, Dr Dixit gave evidence that she did not see Dr Allan's report at any time before Harriet's death (although she had an expectation that it would be available for review during the morning ward round on 22 October 2016). Notwithstanding, Dr Dixit gave evidence that if she had been made aware of Dr Allan's report on 21 October 2016, the only change she would have made regarding Harriet's management plan was to continue antibiotic therapy the following day. Dr Dixit also gave evidence that even if the CXR showed a small pleural effusion, this would not have changed Harriet's acute management as she did not require a procedure to relieve it. Dr Dixit acknowledged the possibility that she was "*anchored*" to her original impression that Harriet was experiencing a viral induced wheeze and overlooked the possibility that the CXR could suggest pulmonary consolidation.

8.6 Professor Craig considered Harriet's CXR to be abnormal and showing alveolar opacity in both lung fields. He formed the view that Harriet's heart appears enlarged and that there is some visible fluid outside the lung but within the chest (pleural effusions). Professor Craig considered that these findings together suggest the possibility of heart failure, but are much more commonly due to infection. Professor Craig initially opined that there was extensive pulmonary consolidation throughout both lung fields, with additional signs possibly consistent with heart failure. However, after seeking the opinion of two radiologist colleagues, Professor Craig later indicated that whilst he remained of the view that pulmonary consolidation is present on the CXR, he was not confident that there are any signs of heart failure.

8.7 One of the consultant paediatric radiologists that Professor Craig spoke to was Dr Siew Swan Yeong, who reviewed the CXR and found diffuse bilateral air space opacities with partial consolidation of the right upper lobe and right lower lung zone medially, a moderate amount of right sided pleural effusion, and a heart size on the upper limit of normal. When asked whether there were any signs of heart failure on the CXR, Dr Yeong indicated:

> If I was rang about the case, I would say with history of fever, I would still favour infection first based purely on imaging but I cannot rule out a mixed [sic] of infection with heart failure and pericardial effusion so correlation with echocardiogram to rule out pericardial effusion and consult with cardiology would be important.

8.8 Professor Wilkinson opined that Dr Dixit's interpretation of the CXR was incorrect because there was consolidation and the changes were more marked than suggested by the word "*mild*". Professor Wilkinson explained that a correct interpretation of the CXR would most likely have resulted in the following:

(a) arrangements for antibiotics and supportive treatment including some physiotherapy, but noting that the rapid progression and Harriet's sudden cardiac arrest a few hours later "*were not predictable and it is doubtful whether they could have been prevented*"; and

(b) arrangement for an echocardiogram, which probably would have shown a pericardial effusion with poor ventricular function, whilst noting that an echocardiogram might have been difficult or impossible to organise until the following morning.

8.9 In the course of preparing his report, Professor Wilkinson spoke to Dr Timothy Cain, the former Director of Medical Imaging at The Royal Children's Hospital in Melbourne, and showed him the CXR. In a statement prepared for the inquest, Dr Cain described his interpretation of the CXR in the following way:

(a) the lungs showed increased density bilaterally indicating that the normal alveolar airspaces were replaced by fluid or inflammatory material, with the most common cause of this appearance being respiratory infection;

(b) any soft tissue density material that replaces the air in the lungs can give the appearance of consolidation; and

(c) except for the denser opacity in the right upper lobe, Dr Dixit's "*description of 'diffuse mild inflammatory infiltrate' was not totally inappropriate*", and most of the focal density in the right upper lobe is due to pleural fluid.

8.10 Overall, Dr Cain expressed the view that the most common cause of an abnormal CXR that fits with the clinical presentation of a patient is usually offered as the most likely diagnosis, and that this is what occurred in Harriet's case. Dr Cain noted that whilst the presence of pleural fluid with a prominent heart could raise the possibility of a cardiac abnormality, fluid overload and other illnesses could also result in a CXR giving a similar appearance. Ultimately, Dr Cain considered that it was reasonable for Dr Dixit "*to look at the [CXR] and interpret the findings to support ongoing treatment for a respiratory infection that was probably viral*".

8.11 Dr Cain explained that exploration of non-respiratory conditions (such as cardiac, cerebral or systemic illnesses) could have been considered "*if the initiated treatment failed to elicit an appropriate clinical improvement*". Further, Dr Cain considered that it would have been unreasonable to initiate further investigations such as echocardiography or laboratory tests overnight given that Harriet appeared settled at times. He also noted that Harriet's rapid decline did not in any event allow for further investigations to be organised or performed.

8.12 Dr Dixit gave evidence that she no longer holds the view that the CXR shows no consolidation. When asked how she might express her view of the CXR today with the benefit of hindsight, Dr Dixit said that she would defer to the expertise of a radiologist and be informed by their expertise as to how they arrived at their conclusion.

8.13 Dr Dixit also gave evidence that having had an opportunity to reflect on the matter, it is now part of her usual practice to seek radiologist review of a x-ray to confirm that it has been reviewed by an appropriate expert.

8.14 **Conclusions:** At the time of review on 21 October 2016, Harriet's CXR was abnormal and demonstrated signs of likely consolidation. In other words, the normal alveolar air spaces had been replaced by fluid or inflammatory material. However, it was unclear at the time whether this clinical picture was more consistent with a cardiac abnormality suggesting the possibility of heart failure, or with a respiratory infection that was probably viral. Dr Dixit's interpretation of the CXR was consistent with the latter, given Harriet's presentation and the working diagnosis of a viral respiratory tract infection.

8.15 Whilst it was reasonable for the CXR to have been initially interpreted in this way, and for Harriet to be treated for a viral respiratory infection, further steps would have been warranted if Harriet did not respond to this treatment. These steps would have involved further investigation such as echocardiography and seeking a cardiology consult to rule out the possibility of any cardiac abnormality. However, given Harriet's later apparent improvement, followed by a sudden and rapid deterioration in her condition, there was regrettably limited opportunity for such steps to taken.

9. The desirability of obtaining a blood gas sample

9.1 Professor Craig explained that a blood gas provides information regarding the amount of oxygen and carbon dioxide in the blood, and the amount of acid in the blood. A blood gas may be taken when there are concerns that a patient with respiratory illness is deteriorating. A normal blood gas would encourage the clinical team to continue current treatment, while an abnormal blood gas might indicate the need for further treatment to improve lung function or other treatment aimed at improving circulation.

9.2 Dr Dixit gave evidence that performance of a blood gas would not, in her mind, be informed by the results of the CXR. She explained that even if the CXR had been "*pristine*" with "*zero findings*", this would not have given her any reassurance. This is because the decision regarding whether to perform a blood gas or not was based on Harriet's respiratory status.

9.3 Notwithstanding, Dr Dixit gave evidence that she had been "*seriously considering*" performing a blood gas. She said that after reviewing Harriet, she spoke to two nurses outside her room and discussed performing a blood gas. According to Dr Dixit, the nurses felt that as Harriet was improving, minimal handling was appropriate. Dr Dixit gave evidence that in response she said, "*OK you're right, she's improved and I agree doing a blood gas is not needed*".

9.4 Later in her evidence, when asked questions by her own counsel, Dr Dixit gave evidence that prior to 2016 she had experience of performing blood gas on paediatric patients as young as Harriet. Dr Dixit explained that the patients typically became distressed, as the drawing of blood needed to be done carefully and slowly with the patient being held still. Dr Dixit gave evidence that she was concerned that performing a blood gas would aggravate Harriet's respiratory status and that with her lungs already inflamed her oxygen requirements would increase with distress, resulting in a decrease in her oxygen saturations.

9.5 Both RN Johnston and RN Wright gave evidence that they were not involved in the apparent conversation described by Dixit. Indeed, RN Wright gave evidence that at the time of her handover

to the night shift, Dr Dixit was already deliberating about having a blood gas performed (as well as about interpretation of the CXR and making a call to NETS). RN Wright gave evidence that she queried whether a blood gas should be performed given Harriet was increasingly tachypnoeic, her oxygen levels were low and she had clinically deteriorated.

9.6 Dr Dixit gave evidence that if she had been made aware of Dr Allan's report this would not have prompted her to consider having a blood gas performed. Dr Dixit said that she had never come across any clinical guideline that suggested that performance of a blood gas should be influenced in any way by the findings from an x-ray. Dr Dixit explained that a blood gas is performed to assess the adequacy of ventilation, and that a CXR may be misleading and overly reassuring in this respect. In other words, it may not reflect a child's ability to ventilate effectively, emphasising the importance of looking at the patient's clinical presentation.

9.7 Professor Craig opined that if Harriet's CXR had been interpreted as being consistent with pneumonia, it would have been reasonable to perform a blood gas in the context of her worsening respiratory rate. If the result of the blood gas was abnormal it may have been enough to prompt further treatment and/or transfer to a larger hospital, particularly if there was evidence of acidosis (build up of acid in the blood). Professor Craig considered that a blood gas could have been performed either time of the CXR or during Harriet's later deterioration between about 11:09pm and 12:47am.

9.8 Professor Craig gave evidence that an abnormal blood gas result would usually make a clinician "*stop and think whether there is something going on here that they are missing*". He explained that an abnormal blood gas result is a "*trigger*" that would prompt retrieval of an unwell child, without necessarily have a definitive diagnosis, to provide a higher level of care with possibly the need for intensive care. He explained that a blood gas is a useful test which does not take long to perform in most cases, and that whilst sometimes it can be challenging (as a child patient may become distressed with the taking of a blood sample) it can be done.

9.9 **Conclusions:** Whilst there were opportunities for a blood gas to have been performed on 21 or 22 October 2016, it cannot be said that these opportunities were clearly indicated and missed. This is because these opportunities were dependent upon interpretation of the CXR, interpretation of the overall trend of Harriet's vital signs observations (discussed further below), and the distress that might be caused to Harriet and its resultant effect on her vital signs.

9.10 If a blood gas had been performed and its results demonstrated an abnormality it is most likely that this would have prompted consideration of whether the working diagnosis for Harriet was indeed correct. At the least, an abnormal blood gas result would likely also have prompted consideration for Harriet's level of care to be escalated so that further investigations could be performed. However, given Harriet's rapid deterioration on the morning of 22 October 2016 it is unclear whether such changes in her management would have materially changed Harriet's clinical course.

10. Were there persistent abnormalities of vital signs suggesting a deterioration in Harriet's condition?

10.1 Professor Craig explained that since 2010, NSW has used the Between the Flags (**BTF**) system for identifying and responding to abnormal vital signs in a patient. For children, vital signs and clinical findings are plotted on a colour-coded chart, divided into zones, appropriate to the age of a child patient. Each coloured zone assists in identifying potentially concerning vital signs. For example, vital signs within the White Zone present normal findings, but vital signs within the Blue Zone represent mildly abnormal findings which require more frequent observation. Vital signs within the Yellow and Red Zones, should prompt a clinical review and emergency response, respectively. Although NSW Health introduced an electronic version of BTF in 2013, the available evidence is unclear whether it was in operation at PMBH in 2016.

10.2 There was differing evidence at the inquest as to the familiarly of the clinicians with the BTF system:

(a) Dr Dixit gave evidence that whilst she is currently familiar with the colour-coded BTF chart, it is possible that she was not familiar with it in 2016;

(b) RN Johnston gave evidence that whilst she is also currently familiar with the BTF system, an electronic version of the BTF chart was not available on PMBH Electronic Medical Record (**eMR**) system in 2016. Instead, a patient's vital signs would be entered on the eMR but no alert or alarm would be provided to indicate to clinical staff that a vital sign was outside normal limits;

(c) RN Wright gave evidence that prior to 2016, she was also familiar with the BTF chart. However she had never used the eMR prior to 21 October 2016 to record vital signs for a patient. She gave evidence that vital would be recorded within the eMR in a section headed "*PT Care – Vital Signs*" and described this as the iView Portal (**iView**).

10.3 The printout of Harriet's vital sign measurements from the eMR indicates that some of the vital signs are displayed in bold and/or with superscript next to the numerical value. RN Wright gave evidence that she could not recall whether:

(a) the information that she was entering in iView in 2016 appeared as it does on the printouts;

(b) iView displayed warnings or alerts that vital signs were outside certain clinical parameters; or

(c) it was possible to access a colour-coded BTF chart from within iView.

10.4 Professor Craig gave evidence that the rollout of an eMR system is complex and poses clinical risks which can be mitigated to a certain extent, but not entirely. He explained that the initial weeks of such a rollout makes it "*much harder to do the same job*" and "*very hard to maintain the same standard of care*" when dealing with "*a large complex beast*". Whilst acknowledging the benefits of electronic documentation, Professor Craig described the initial learning curve as not being straightforward and one with many challenges.

10.5 In order to assist his interpretation of Harriet's vital sign observations, Professor Craig extracted them from the PMBH medical record and plotted them onto a BTF chart. Professor Craig explained that this showed the following:

(a) Harriet was briefly within the Red Zone for RR at around 11:00am having mild respiratory distress, but then settled;

(b) At 8:59pm, Harriet was noted to have "*widespread wheeze on auscultation, moderate recession and tracheal tug*". Professor Craig explained that such findings are signs of difficulty breathing. Harriet was given salbutamol and oxygen and Dr Dixit was called to the ward. Dr Dixit remained on the ward whilst further salbutamol was given (at 9:10pm and 9:35pm) and intravenous hydrocortisone was given at 10:00pm;

(c) By 11:09pm, Harriet's RR had dropped from 76 to 64, although her HR had increased slightly from 150 to 160. Professor Craig considered that it was reasonable for Dr Dixit to leave the ward at this time as Harriet's RR appeared to have settled.

(d) However, at around 12:13am, Harriet was noted to have a fever and a very high RR (76);

(e) At 12:47am, Harriet's RR was again recorded as being very (74). Although this measurement was in the Red Zone, suggesting that an emergency response was required, there is no evidence of any further medical review or any consultation with Dr Dixit.

10.6 Professor Craig therefore considered that Harriet had a further clinical deterioration between 11:09pm and 12:47am, given her increased RR (above 70) and increased HR (172). Despite this, there was no meaningful change in Harriet's management and this did not trigger a medical review or emergency response.

10.7 In relation to Harriet's vital signs, Dr Dixit gave evidence that she:

(a) observed a deterioration in Harriet's condition after 4:00pm;

(b) considered this to be due to a mucus plug or bronchospasm;

(c) instructed the nursing staff before leaving the ward to call her if Harriet's oxygen saturations decreased, RR increased or if Harriet demonstrated any worsening work of breathing;

(d) explicitly told the nurses that if Harriet "*grunts one more time, call me*"; and

(e) could not recall giving precise instructions as to the thresholds for RR or oxygen saturation levels which would prompt the nursing staff to call her.

10.8 Dr Dixit agreed with Senior Counsel Assisting that whilst she gave the nurses "*broad fields*" with which to interpret Harriet's vital sign measurements, she did not nominate specific triggers which would prompt further steps to be taken regarding Harriet's management. When asked questions

about Harriet's vital sign measurements which were taken after she left the ward, Dr Dixit gave evidence that:

(a) a RR of 64 at 11:09pm represented moderate respiratory distress and that this was the sort of change in vital signs that she had in mind when she instructed the nurses to call her;

(b) Harriet experienced another deterioration at 11:45pm which, even in the context of fever, she would have liked to have been notified about;

(c) in her past experience she had found it helpful to know the direction that a child patient was trending;

(d) she would have wanted to be notified of the observations taken at 12:47am;

(e) whilst Harriet's RR and PR were within normal limits at 3:56am, her temperature had increased, although in the context of a fever it was normal for a patient's temperature to fluctuate

10.9 Ultimately, Dr Dixit gave evidence that she was not in a position to speculate what she would have thought at the time if Harriet's vital sign measurements had been communicated to her. Dr Dixit explained that Harriet's clinical observations were complicated by development of fever which could contribute to an increase in RR and HR. However, Dr Dixit expressed the belief that whilst she was unsure what steps (if any) she would have taken, if she was taking a precautionary approach she would have initiated some investigations.

10.10 RN Johnston gave evidence that prior to leaving the hospital, Dr Dixit did not formulate a concrete management plan in the event that Harriet deteriorated. RN Johnston said that her impression was that minimal handling was to be performed and that she was to contact Dr Dixit if she felt that Harriet had deteriorated. However, RN Johnston considered this did not occur overnight on 21/22 October 2016.

10.11 RN Johnston gave evidence that:

(a) a minimal handling technique is a typical approach for children experiencing a respiratory condition;

(b) allowing Jess to lay down in bed next to Harriet had a "*huge impact*", resulting in a decrease in both her RR and HR;

(c) she did not consider Harriet's vital signs to be high because, in her experience, children with a diagnosis of respiratory illness have increased RR and HR;

(d) she did not consider contacting Dr Dixit because with the interventions that she put in place, she saw a decrease in Harriet's vital signs and felt that she was settling;

(e) the changes in Harriet's temperature could be attributed to the working diagnosis of a respiratory viral illness (and nursing staff were not working under any other diagnosis) and/or a result of the paracetamol wearing off;

(f) she did not consider this represented a new symptom about which Dr Dixit should have been informed;

(g) she agreed with Dr Dixit that a patient's HR and RR can increase with a fever; and

(h) she felt that Harriet's observations between 11:09pm and 12:47am were consistent with the diagnosis of viral respiratory illness that she was working with, and that of a "*fractious toddler*".

10.12 Professor Craig acknowledged in his evidence that because Harriet had a fever this would mean that she would have an elevated HR and RR. However, he gave evidence that even though Harriet was settled in bed her RR reached 76 which would not be considered to be mildly abnormal; rather, it was close to the uppermost limit of the Red Zone at 80.

10.13 Professor Craig also acknowledged that child patients with a respiratory illness tend to have a faster RR than child patients with other illnesses such as gastroenteritis. In addition, Professor Craig acknowledged that observation charts do not account for differences between patients. That said, Professor Craig considered that Harriet's increased RR (jumping from the 60s to 76) represented a "*child on the margins*". Professor Craig gave evidence that this made him concerned that this represented a deterioration in Harriet's condition and that if she deteriorated further, performing a blood gas and consulting with NETS would have been appropriate steps in her management.

10.14 However, Professor Craig acknowledged that Harriet's three subsequent vital sign measurements demonstrated apparent improvement. In this regard, Professor Craig explained that it is difficult to distinguish between normalisation of vital signs where a child patient is improving, as opposed to pseudo-normalisation which represents a child in terminal decline. He gave evidence that based simply on the documentation it is not possible to tell the difference, and that without having the patient in front of him, it would be difficult to determine whether the patient was improving or deteriorating.

10.15 Overall, Professor Craig adhered to the opinion which he expressed in his report, that Harriet had a further clinical deterioration between 11:09pm at 12:47am which warranted review by a medical officer and an emergency response, neither of which occurred. However, Professor Craig described the situation confronting the clinicians at the time as being "*not easy*" and that the differences between actual normalisation and pseudo-normalisation of vital signs were subtle. By way of example, Professor Craig noted that Harriet was showing signs of mottling on her trunk at Dr Dixit's assessment at 10:28pm. Professor Craig explained that mottling usually indicates that skin is not receiving enough blood flow which is a "*soft sign*" for serious illness. However, at the same time Harriet's capillary refill was 2 to 3 seconds which is considered to be normal.

10.16 Overall, Professor Craig considered that some of Harriet's vital signs should have been regarded as abnormal, or at least represented a trigger to reconsider her management, but this was not clear-

cut. Professor Craig gave evidence that this was not a case where Harriet experienced an obvious deterioration which was missed, with no mitigating circumstances.

10.17 **Conclusions:** Prior to leaving the ward on 21 October 2016, Dr Dixit did not give explicit instructions to the nursing staff regarding whether they were to call her if Harriet's vital sign measurements reached certain thresholds. In the period between 11:09pm on 21 October 2016 and 12:47am on 22 October 2016, Harriet's vital sign measurements indicated that she had a further clinical deterioration. This should have resulted in a call to Dr Dixit to review Harriet and an emergency response.

10.18 Interpretation of the measurements taken of Harriet's HR and RR were complicated by the diagnosis that nursing staff were working with. In other words, elevated measurements for both vital signs are not uncommon in a child patient with an apparent viral respiratory illness and fever, and are different from other child patients in the rest of the hospitalised population. In addition, Harriet's apparent improvement after 12:47am likely provided a false sense of reassurance. However, there are subtle differences between genuine improvement and terminal decline in a patient on Harriet's clinical course, which are difficult to recognise.

10.19 One additional complicating factor was the inability of nursing staff to record or access Harriet's vital signs on a BTF chart. This would have provided a clear visual representation of the trend in Harriet's clinical progress and when her vital signs reached certain thresholds which warranted an escalation in her management. However, as at 21 October 2016, PMBH was undergoing a transition to an eMR system. Such a transition is complex and presents challenges with maintaining the same standard of clinical care for patients. Since 2016, these challenges have been overcome to a significant extent by changes within the eMR (see further below) and the familiarity of clinical staff with the eMR.

Hydration

10.20 Two additional matters relevant to Harriet's deterioration on 21 and 22 October 2016 arose for consideration in the inquest: management of Harriet's hydration and possible signs of bloating.

10.21 Dr Dixit gave evidence that she was reassured from looking at the fluid chart that Harriet was producing urine. She explained that a child who has a viral illness and is in respiratory distress is losing fluid through their lungs, skin and by breathing. This means that there is no exact correlation between and input and output of fluids.

10.22 RN Johnston gave evidence that she did not consider whether there should be any concern that Harriet not passing urine. RN Wright gave evidence that she was not concerned about Harriet's not passing urine. She said that she was aware that Harriet had been given a couple of intravenous fluid boluses and had maintenance fluids. Further, RN Wright said that she performed a head-to-toe assessment of Harriet and that she did not seem to be dehydrated.

10.23 Professor Craig noted that a fluid balance chart was commenced for Harriet at around 11:00am on 21 October 2016. This recorded intravenous fluid administration as well as oral intake of water and

formula. Professor Craig explained that whilst no urine output was recorded after midnight, this is not unusual "*as children are not usually woken to change and happy overnight*".

10.24 Overall, Professor Craig opined that Harriet's fluid intake was factored into her treatment. She was initially assessed as not having drank much during the day on 21 October 2016 and so was commenced on intravenous fluids. When Harriet was observed to drink more fluid after midnight (approximately 300 mL) the rate of intravenous fluids was reduced. Overall, Professor Craig expressed the view that Harriet's fluid intake was appropriately monitored. Professor Wilkinson also was of the view that Harriet's fluid management was appropriate.

Bloating

10.25 RN Johnston gave evidence that she could not recall any conversation with Jess in which she asked where all the fluid that had been given to Harriet was going. She also could not recall saying anything to suggest that all the fluid that Harriet had been given was in her tummy. She also could not recall any observation at around 2:00am on 22 October 2016 that Jess had noted that Harriet's stomach was bloated. RN Johnston said that she did not think it was possible for a child to be given intravenous fluids for dehydration to become bloated.

10.26 Professor Craig gave evidence that it is likely that Harriet's abdomen would have been bloated with fluid. He explained that when the heart pumps abnormally it becomes less efficient and fluid is not moved around the body as normal. As a result, pressure can build causing fluid leak to out into the lungs but also into the abdomen. Ascites refers to fluid build-up in the abdominal cavity and is commonly seen in cases of heart failure. That said, Professor Craig acknowledged that whilst heart failure in adults is easier to detect (because, for example, leg swelling is more evident) it is more difficult to detect in children where evidence of bloating or swelling can be more subtle and with other respiratory symptoms (such as a runny nose, wheezing and cough) which would be attributed to a viral illness in most instances.

10.27 Professor Craig gave evidence that he did not consider that there was "*one single red flag*" with respect to Harriet's hydration apart from Jess being worried about it. Professor Craig acknowledged that this is often seen in cases where there is an adverse patient outcome. In other words, parents often know that something is wrong with their child whilst clinicians (who obviously do not know the child as well) have difficulty recognising this.

10.28 In Harriet's case, Professor Craig explained that Harriet's elevated RR, lower urinary output than expected and bloated stomach were all concerning, and that over time that they might together highlight that "*something bad was going on*". However Professor Craig explained that without a review by a medical officer "*who can put it all together*" it would be difficult for nursing staff to recognise the significance of these factors "*with all of the complexities going on*". Professor Craig gave evidence that he did not doubt that Harriet was getting worse but expressed uncertainty regarding how obvious this was to the treating team. Professor Craig acknowledged that with hindsight there were "*lots of clues*" that there was "*something going on*" but that it was not clear-cut or straightforward situation where something was "*grievously*" missed.

10.29 **Conclusions:** Whilst Harriet's fluid management was generally appropriate, it is most likely that her underlying cardiac pathology affected its efficacy and resulted in lower urinary output and bloating. These were additional signs (along with Harriet's abnormal vital sign measurements) that suggested a picture of clinical deterioration rather than improvement. However, whilst such signs are more easily recognised with the benefit of hindsight, they were less apparent in October 2016 in the context of a challenging clinical case, particularly in the absence of medical review.

11. What changes have since occurred at Port Macquarie Base Hospital?

11.1 At the conclusion of the oral evidence in the inquest, PMBH provided information that as at 19 April 2023, a patient's vital sign observations are recorded within iView, with the entries been colour-coded. This colour coding differentiates between whether vital signs are within the normal range, the upper and lower limits of the abnormal range, or in the critical range. When a vital sign is recorded in the critical range, iView provides an automatic warning prompt indicating that the vital sign is outside defined critical limits. This requires the clinician entering the information to acknowledge the warning before continuing.

11.2 In addition, if observations are entered within the abnormal or critical ranges, an alert is displayed which indicates that the vital sign is abnormal and directs the clinician to access the BTF observation chart. This BTF chart is displayed electronically with colour coded zones in a similar way as it appeared in hardcopy form prior to the introduction of the eMR.

11.3 Catharine Death, the General Manager of PMBH, provided information regarding a number of improvements made following Harriet's death and after the MNCLHD had examined the circumstances surrounding the death.

11.4 *First*, a Paediatric Critical Care Pathway (**the Pathway**) has been developed and implemented. PMBH recognised that there was no clinical escalation of Harriet's care in accordance with the Clinical Emergency Response System (**CERS**) procedure which existed at the time. The Pathway is designed for paediatric patients (up to the age of 16) who are acutely unwell and/or clinically deteriorating and requiring closer observation with more intensive care. Relevantly, it provides

(a) that if a paediatric patient is admitted to the paediatric ward and placed on the Pathway, the On-Call Paediatric Medical Officer will physically review the patient within 30 minutes and document any changes to the management plan;

(b) that the On-Call Paediatric Medical Officer will contact other specialties for review and input as is clinically indicated; and

(c) for the clinical handover process for paediatric patients with clinical observations outside normal limits.

11.5 In addition, an on-call paediatric registrar role has been created to allow for provision of clinical advice in accordance with the Pathway. Further, nursing staff have a number of responsibilities including competence and confidence in caring for sick paediatric patients in accordance with the relevant NSW Health policy and guidelines for children in acute care settings.

11.6 *Second*, **DETECT** (Detecting Deterioration, Evaluation, Treatment, Escalation and Communicating in Teams) Junior training is part of the State-wide BTF education program, specifically designed for frontline clinical staff who care for infants and children to assist them to improve recognition and management of infants and children who are clinically deteriorating.

11.7 *Third*, there is now a sepsis champion on the paediatric ward whose training is provided within the DETECT Junior training which includes a sepsis component. In addition a clinical nurse educator role was established at PMBH in 2020. This role provides educational support to the paediatric ward for all clinical needs as well as sepsis concerns and conducts the compulsory DETECT Junior training.

11.8 *Fourth*, in March 2019, all junior medical officers (**JMO**) were instructed to attend a paediatric ward round at least once per shift in order to undertake medical reviews of patients as required. Prior to Harriet's death, there was no requirement for a JMO to review a paediatric patient after hours. This has now become a requirement of the JMO position description. An audit has determined that there has been 100% compliance with patients on the paediatric ward undergoing JMO review as required on each shift.

11.9 In addition, Associate Professor David McDonald, Head of Paediatrics at PMBH, gave evidence that staff coverage has now been approved so that there are now two staff specialists, two consultants and 24/7 paediatric registrar cover. Associate Professor McDonald gave evidence that the aim is to have complete on-site cover but this is subject to recruitment considerations and budget limitations. In addition, Associate Professor McDonald referred an increase in the number of registrars meaning that there is less reliance on locum medical officers, which improves quality of care for patients. Associate Professor McDonald explained that the preferred way to provide care is to rely on permanent staff members who work cohesively as a team with entrenchment of collaboration.

11.10 Finally, Associate Professor McDonald referred to the introduction of the Clinical Excellence Commission REACH (Recognise, Engage, Ask, Call, Help) Program which is designed to help patients, carers and families to escalate any concerns with clinical staff about worrying changes in a patient's condition. If they continue to be worried their concerns may be escalated by requesting a clinical review which is to occur within 30 minutes. Finally, further concerns can result in an independent review or rapid emergency response.

11.11 **Conclusions:** The changes made at PMBH since Harriet's death represent improvements in the quality of care provided to paediatric patients. They will hopefully assist in mitigating against the risks posed in complex cases like Harriet's and ensure that there is appropriate recognition of a clinically deteriorating patient and escalation of care. Having regard to the matters set out above, it is neither necessary nor desirable to make any recommendation pursuant to section 82 of the Act.

12. Findings pursuant to section 81(1) of the Act

12.1 Before turning to the findings that I am required to make, I would like to acknowledge, and express my gratitude to Ms Donna Ward SC, Senior Counsel Assisting, and her instructing solicitor, Ms Catherine Moore from the Department of Communities and Justice. I am also grateful to Ms Lara

Vaccaro and Ms Monique Azzopardi, the previous solicitors with carriage of this matter. The Assisting Team has provided enormous assistance during the conduct of the coronial investigation and throughout the course of the inquest. I am extremely grateful for their meticulousness, and for the sensitivity and empathy that they have shown during all stages of the coronial process.

12.2 I also acknowledge the assistance of Detective Senior Constable Louise Currey in compiling the initial brief of evidence.

12.3 The findings I make under section 81(1) of the Act are:

Identity

The person who died was Harriet Ostler.

Date of death

Harriet died on 22 October 2016.

Place of death

Harriet died at Port Macquarie Base Hospital, Port Macquarie NSW 2444.

Cause of death

The cause of Harriet's death was sudden cardiac death on a background of rhinovirus infection.

Manner of death

Harriet died of natural causes whilst an inpatient at Port Macquarie Base Hospital. Although Harriet was treated for suspected viral respiratory illness she had significant underlying cardiac pathology which was not recognised. If this had been recognised it is likely that changes would have been made to Harriet's treatment and her care escalated. However, it is unclear whether such escalation, if it had occurred, would have averted the eventual outcome given the sudden and unexpected nature of Harriet's clinical deterioration leading to cardiac arrest.

13. Epilogue

13.1 There is no doubt that Harriet's death was tragic and untimely, and that her loss is still very deeply felt by all of her family and loved ones.

13.2 On behalf of the Coroners Court of New South Wales, I offer my sincere and respectful condolences, to all of Harriet's family, in particular, Jess, Bob, Milton, Leeroy, Dulcie, Clancy and Winston for their devastating loss. It is clear that Harriet will always be with them all, in their memories and in their hearts.

13.3 I close this inquest.

Magistrate Derek Lee
Deputy State Coroner
9 May 2023
Coroners Court of New South Wale