QUEENSLAND COURTS

CORONERS COURT OF QUEENSLAND

FINDINGS OF INVESTIGATION

CITATION:	**Non-inquest findings into the death of R, a 10 month old boy**
TITLE OF COURT:	Coroners Court
JURISDICTION:	BRISBANE
DATE:	8/07/2022
FILE NO(s):	2020/3216
FINDINGS OF:	Ainslie Kirkegaard, Acting Brisbane Coroner
CATCHWORDS:	CORONERS: health care related death; paediatric cleft palate repair surgery; post-operative respiratory distress; management of endotracheal tube; indicators of appropriate endotracheal tube position; EtCO2 monitoring of intubated and ventilated patients; use of continuous capnography

Background

R was a 10 month old boy who died at a tertiary hospital in Brisbane on 31 July 2020.

His death was reported to the coroner because it was an unexpected outcome of elective cleft palate repair surgery performed on 27 July 2020.

R had been diagnosed antenatally with a unilateral complete cleft lip and right anterior cleft palate. Following birth, he was referred to a local speech pathology service, lactation service, paediatrician and the tertiary hospital's Cleft Palate Clinic. He was also referred for newborn hearing screening. He required three hourly feeds and monitoring of his weight given his feeding difficulties.

R's parents describe him as a happy and bubbly baby who was never sick. He was their first and only biological child who meant a great deal to them.

R underwent successful cleft lip repair surgery at the tertiary hospital on 16 January 2020 at three months of age. There were no acute surgical or post-operative complications. He developed a wheeze after the surgery but was assessed as well for discharge home on 18 January 2020. He presented to the tertiary hospital emergency department the following day, 19 January, with coryzal symptoms, stridor, a barky cough, and reduced oral input and urine output. He was admitted to the Short Stay Unit for monitoring and observed to remain stable. He was discharged home the following morning, 20 January. His parents thought this was just a result of R having had an anaesthetic and irritation to his throat. His father had previously experienced a similar response himself. Subsequently, R returned to hospital as a day case for an uncomplicated and successful suture removal under general anaesthesia following the cleft lip repair.

R's admission to hospital for cleft palate repair surgery

R was scheduled to return for cleft palate repair surgery on 27 July 2020. He developed a cough in the week leading up to the surgery date. His mother phoned the hospital in advance to let them know he had a cough.

R was admitted to the tertiary hospital on 27 July as planned for elective cleft palate repair, myringotomy and bilateral insertion of Shepard grommets (for otis media with effusion and associated hearing loss) that day. He was admitted at 8:06am. Pre-anaesthetic assessment noted his two previous general anaesthesias had been uncomplicated. His parents reported he had a one-week history of cough. The anaesthetist noted slight clear rhinorrhea from the right nostril during R's COVID-19 screening, indicating R had an upper respiratory tract infection. His parents had not noticed he had a runny nose before this. His mother told the anaesthetist she thought R was teething and the rhinorrhea was only one sided because that was the side of his cleft. R's observations were noted to be within normal range, dual heart sounds with no murmur and his lungs were clear on auscultation. He was observed to be happy, appeared well and there were no clinical concerns.

R's parents were expecting a two-day hospital stay and were most concerned about feeding R after his surgery because it had been challenging after his recent cleft lip repair.

Generally cleft palate surgery patients are transferred to an inpatient surgical ward for their post-operative care and have an average length of stay of two days. It is a very rare event that patients require post-operative care in the paediatric intensive care unit (PICU) after their cleft palate surgery.

The general anaesthesia & surgery

Anaesthesia commenced at 9:06am. R was observed to have mild sternal recession on induction. He was intubated with ease. While the mild sternal recession was recognised as unusual in the

context of R's mild presenting respiratory symptoms, it was not severe enough to require a change to the anaesthetic plan. R's three-hour anaesthetic was otherwise uneventful.

The surgery itself was uneventful. Shephard grommets were placed bilaterally, followed by repair of the right unilateral cleft palate. The surgery was completed within the expected timeframe, ending at 11:39am.

At the end of the surgery, R was administered neuromuscular blocker reversal medications. Anaesthesia ceased at 11:54am. R was observed to breathe spontaneously, his oropharynx was suctioned to remove secretions, and he was extubated onto a face mask with a T-piece anaesthetic bag and CPAP was provided. This took place in the operating theatre. On extubation, R developed severe sternal recession and a "see-saw" pattern to his breathing and rapidly desaturated. The anaesthetist diagnosed severe laryngospasm and administered 10mg intravenous propofol. The laryngospasm resolved but R's oxygen saturations were slow to recover.

It was queried whether R had undiagnosed tracheobronchomalacia, a condition in which the airways collapse due to abnormal flaccidity of the airways.

R was transferred to recovery.

R's management in recovery

R was monitored for 120 minutes in recovery, requiring continuous positive airway pressure (CPAP) and oxygen (10L via T-piece anaesthetic bag) during this time.

He continued to have significant respiratory effort and sternal recession which only improved with increased CPAP via the face mask and T-piece anaesthetic bag. Despite this his oxygen saturations remained in the low to mid 90s (normal above 95%). He was observed to rapidly desaturate to the mid-70s if the face mask and CPAP was removed. Harsh crepitations could be heard throughout both lung fields on auscultation.

R regained consciousness at around 12:30pm. He was observed to cry and roll over in the prone position with some blood pooling in his mouth. He was noted to have severe sternal recession and his breathing appeared very uncoordinated. His oxygen saturations dropped to the low 70s. He was given 5mcg fentanyl to settle him and to maintain control of his airway and respiration.

R was reviewed by the paediatric plastic surgeon who was not concerned with the level of bleeding; it was considered to be within the normal and expected range for the immediate post-operative period.

A mobile chest x-ray performed at 1:37pm showed some mild, patchy haziness in the right lower lobe but no other obvious pathology to explain the level of R's oxygen requirement. In particular there was no significant pulmonary oedema, lung collapse or consolidation.

Given R's ongoing requirement for ventilatory support and oxygen, the PICU Registrar and consultant on-call were consulted about transferring R to the PICU for high flow oxygen therapy. R was assessed by both the PICU Registrar and consultant who, in discussion with the anaesthetist, decided R would likely require intubation given the degree of respiratory distress and level of ongoing ventilation and oxygenation support required.

R's stomach was distended due to being on prolonged CPAP support, so an oral gastric tube was placed by the plastic surgery Registrar. It was decided to trial him on high flow oxygen (3L/kg/min and FiO2 0.7) for 30 minutes to see if his oxygen saturations improved as he became less sedated. This commenced at 3:00pm.

The anaesthetist spoke to R's mother about R's condition at this time. His parents had received a phone call at around 11:55am advising his cleft palate had come together and everything had gone

well. They then went to wait in the post-operative waiting area. After an hour R's father went to reception and was told R had only just arrived in recovery. They left the waiting area to set up his room on the surgical ward. They unpacked but were then told they would need to move rooms to be closer to the nurses station as R would require closer monitoring. Shortly after moving rooms, R's father received a call asking to speak with R's mother. She was asked to go to recovery where she spoke with the anaesthetist.

The trial of high flow oxygen stopped at 3:30pm. R had been observed to desaturate to the high 80s despite maximum high flow settings so the anaesthetist decided to intubate R in recovery. He was sedated and intubated at 3:45pm. The anaesthetist observed R to have a grade 2, blood-stained larynx. R was then transferred to PICU for further management.

R's management in PICU

On arrival in PICU R was noted to be intubated and ventilated with good air entry and crackles heard bilaterally on auscultation. Blood-stained secretions were observed periodically in his endotracheal tube which would coincide with an episode of dyssynchrony with the ventilator, followed by a period of desaturation requiring suctioning of secretions to stabilise ventilation and oxygenation.

The nurse-controlled analgesia started in recovery was ceased and replaced with dexmedetomidine and morphine infusions, with a plan for fentanyl boluses for sedation and intermittent paralysis with vecuronium (neuromuscular blockade) to occur as required.

R was not for nasal endotracheal tube, nasal suction or insertion of a nasogastric tube given the potential for damage to the surgical site. A sign stating "do not suction nares" was placed at his bedside alerting the clinical team to this order.

A sample of R's endotracheal tube secretions was ordered to test for cultures and viruses. He was positive for Rhinovirus, a common cold virus.

A further chest x-ray was performed at 6:04pm. An indwelling catheter and an intra-arterial line were inserted.

R was difficult to ventilate during the evening and overnight, requiring high peak pressures on the ventilator. He was noted to have periods of waking and distress and remained on moderate significant levels of sedation with intermittent vecuronium to support his ventilation and oxygenation.

R was observed to wake up and "fight" and "hold his breath" against the ventilator and then desaturate with increased work of breathing. He required suctioning on five occasions during the night shift when blood stained/frank blood secretions were observed in his endotracheal tube.

R's endotracheal tube was re-taped at around 9:00pm after the treating team reviewed the chest x-ray. It was measured at 12cm at the lips and was pulled out by 0.5cm and taped at 11.5cm at the lips and 9.5cm externally. Good air entry was confirmed by auscultation and end tidal carbon dioxide (EtCO2) was observed to be within normal limits. Staff observed good rise and fall of the chest. A repeat chest x-ray was to be ordered and performed the following morning. Cuff pressure was documented as checked throughout the night measuring 20cm H2O (within the normal and expected range).

The Staff Assist buzzer was activated at 11:10pm when the PICU bedside nurse noticed R wake up and grab at his endotracheal tube. He was observed to be a blue/grey colour. R required suctioning and bagging via a T-piece anaesthetic bag which improved his oxygen saturations. Frank blood was suctioned from the endotracheal tube. He was administered sedation and vecuronium and a midazolam infusion was commenced to ensure he remained sedated. This event

resolved quickly following suctioning of R's endotracheal tube and he was placed back on the ventilator.

R's condition during 28 July

R was hypotensive in the early hours of 28 July. The PICU Registrars were called to review him as the treating team were aiming to ensure his mean arterial pressure remained above 50mmHg. This was felt to be due to sedation. His gas exchange was improving and he was likely to be extubated later that day so it was decided to start weaning sedation to help increase his blood pressure. His ventilation support had been weaned across the night shift with a change in rate from 35 to 30 breaths per minute, tidal volume 8 to 7ml/kg, FiO2 0.75 to 0.40.

The PICU medical team overnight considered the possibility of further bleeding because R had ongoing bloody aspirates. His haemoglobin decreased from 101 to 82. It was decided there was no need to escalate the continued oozing/bleeding to the plastic surgery team overnight.

R's mean arterial pressure continued to fluctuate so he was given metaraminol at around 7:00am with a transient increase in his mean arterial pressures.

A chest x-ray performed at 7:07am and reported to the treating team at 7:36am reported the endotracheal tube was adequately positioned in the mid-thoracic trachea and the tip of the oral gastric tube was in the stomach.

Given the challenges overnight and the cause of the post-operative respiratory distress remained unclear, the treating team decided R should remain intubated and ventilated. He was commenced on treatment for fluid overload. His endotracheal tube remained secured, measuring 11.5cm at the lips and 9.5cm externally. Cuff pressure was documented as checked twice through the morning, measuring at between 15-20cm H2O (within normal and expected limits). EtCO2 documented manually by nursing staff on the hour was within normal limits. Equal air entry was observed bilaterally with some added sounds noted. An arterial blood gas taken at 10:40am confirmed adequate oxygenation and ventilation.

There was a plan to re-tape R's endotracheal tube that morning. It was discussed at the PICU ward round. His parents noticed R had become more awake during the morning and moved his arm and shook his head in a familiar way as if saying no. He often shook his head this way.

Events leading up to R's bradycardic arrest at 11:09am

Between 7:00am and 11:09am, R was noted to have four dyssynchronous, "splinting" like episodes on the ventilator. He would become more awake and agitated, moving his arms vigorously and his oxygen saturations would decrease. The first two episodes resolved with suctioning of his endotracheal tube. R was administered vecuronium after the second episode, followed by a morphine bolus delivered from the infusion pump, and a dose of fentanyl. Splinting was observed to continue and within 60-90 seconds, R was noted to be very difficult and 'stiff' to bag, despite having been given the muscle relaxant. The third episode triggered a request for a second dose of vecuronium and fentanyl which may or may not have been given.

R very quickly progressed to a fourth episode precipitating a period of profound desaturation with oxygen saturations of 29% and bradycardia (heart rate 29bpm). CPR commenced and an emergency call was activated at 11:09am.

R's parents were aware the PICU nurses had wanted to suction and re-tape his endotracheal tube that morning. The bedside nursing team tried to instigate the agreed plan and obtain assistance from other PICU nurses at around 10:30am. Clinical demand within PICU at that time meant the additional nursing support required was not available at that time, so it had to be reprioritised.

R's parents perceived there to be disagreement between one nurse and a PICU Registrar regarding the sedation required to perform suctioning and re-taping. While R was being suctioned, they heard him make a sound they identified as an audible cry. R's parents recall the suctioning continued despite what appeared to them to be his distress and this was followed by the events triggering the emergency call activation. One of the clinicians later reported hearing R make a small noise, "*not a loud baby cry but you could definitely tell it was him*" but in one of the earlier episodes of dyssynchrony.

Issues arising during the emergency resuscitation

At 11:14am, Code EMCO was activated after two rounds of CPR had not achieved return of spontaneous circulation. R received 42 minutes of CPR. He was successfully placed on ECMO (extra-corporeal membrane oxygenation) with surgery commenced at 11:31am for extra-corporeal life support via cervical cannulation and flow established by 11:50am. He remained intubated with rest settings on the ventilator.

Some of the clinicians involved in the emergency resuscitation noted abdominal distension evident prior to CPR commencing. A number recall a second oral gastric tube was sited during the resuscitation to assist in repeated aspiration of the air accumulating in R's stomach. Some say they voiced concerns about the abdominal distension without these concerns influencing a change in approach. Others later confirmed that while they were concerned, they were focused on their assigned role in the resuscitation and did not voice their concern at the time.

There were concerns about the integrity of the tapes and securement of R's endotracheal tube during CPR. Multiple efforts were made during the resuscitation to try and secure it. The tapes were variously described as being 'wet' and 'soaked' by blood and ooze. One clinician described re-securing the tapes prior to draping but the continued oral ooze remained a concern. Clinicians reported it was difficult to maintain line of sight of the endotracheal tube during draping and preparation for ECMO cannulation. The allocated airway clinicians were bent down under the surgical drapes in order to hold the endotracheal tube and continued to bag R while he was being prepped by the paediatric cardiothoracic surgical team for cannulation. The paediatric cardiothoracic surgeon documented "*as an added precaution, one of the nursing staff manually held the tube in position for the duration of the procedure*."

R had to be moved and rotated 180 degrees on the bed in order to facilitate extra-corporal cannulation and allow the cardiothoracic surgical team access to him while CPR continued. This meant his head was eventually at the foot of the bed when the ECMO procedure commenced. The oxygen tubing attached to the pendant mounted flowmeter at the head of the bed could not reach R when he was rotated. Portable oxygen cylinders needed to be sourced in order to ensure he received continuous 100% oxygen as the cycles of CPR continued.

Repeat chest x-ray taken at 11:54pm reported the tip of the endotracheal tube to be at T2 and the tip of the oral gastric tube to be in the grossly distended stomach. The ECMO catheters were in a satisfactory position, one within the aorta and one in the right atrium with surgical clips projected over the upper neck.

R was moved to a different location within PICU. On handover it was documented he needed "a new ETT" due to the amount of bloody ooze observed during CPR. Information was also handed over that R's endotracheal tube was reported to have a leak in the morning prior to his arrest.

R's endotracheal tube was advanced by 1cm by the medical team at around 4:00pm. After advancement, air entry was noted to be very quiet by auscultation and increased amounts of air were aspirated from his stomach. A repeat chest x-ray performed at 4:39pm demonstrated the endotracheal tube had advanced and the distal oesophagus was observed to be dilated with gas. The reporting radiologist escalated concern to PICU about the possibility of oesophageal intubation at this time.

Direct visualisation noted the endotracheal tube had migrated into R's oesophagus. He was re-intubated at around 5:30pm. Despite a grade 1 view, neither a size 4.0 nor a size 3.5 endotracheal tube could be passed. A size 3.0 endotracheal tube was passed and secured at 14cm at the lips and 6cm externally.

Throughout the course of the afternoon R was seen by the respiratory team with a view to assessing his airway with a bronchoscopy the following day. He was also seen by the surgical team given his significant gastric distension. This resolved following re-intubation.

A chest x-ray taken at 9:18pm demonstrated the tip of the endotracheal tube sited at the carina, possibly extending minimally into the right main bronchus, with slight retraction recommended.

The plan that evening was to stabilise R on ECMO. He remained deeply sedated with rest settings on the ventilator. It was difficult to assess his neurological status. Consideration was given to a CT brain scan overnight but it was decided to defer this until the following morning given the risks associated with transporting a patient on ECMO during the night shift.

Although R's heart and lung function improved on EMCO, CT imaging revealed severe hypoxic brain injury. This was confirmed on MRI brain. Following ongoing discussions with R's parents about his poor neurological prognosis, they made the very difficult decision to redirect his care to comfort measures. R was extubated on the PICU balcony at 5:26pm on 31 July 2020 and declared deceased at 5:50pm.

Post-mortem findings

A full autopsy was performed on 5 August 2020. The brain was retained for specialist neuropathology examination. The final autopsy report issued on 13 October 2021.

Internal examination noted the recent sutured repair of the palate. The wound was clean and intact with no significant swelling, acute haemorrhage or infection. The airways showed no obvious occlusion or definite malformation. The lungs were heavy, firm and had a solid appearance. Microscopic examination showed some changes consistent with mild upper respiratory tract infection and changes in the lungs suggestive of evolving diffuse alveolar damage. There were no cardiorespiratory congenital abnormalities. There were changes of active CMV infection in the submandibular glands. Microbiology testing detected CMV DNA in both lungs and *Pseudomonas aeruginosa* was also cultured from lung tissue. Specialist neuropathology examination of the brain noted changes of global hypoxic ischaemic encephalopathy. Molecular biology and metabolic screening were unremarkable.

Having regard to these findings and the clinical history, the pathologist was not able to identify the underlying cause of R's initial respiratory distress. Possibilities included a reaction to anaesthetic (though this was not suspected clinically), obstruction due to bleeding or foreign material or a possible respiratory tract disorder such as tracheomalacia or bronchomalacia. While there was no definite evidence of respiratory tract disorder at autopsy, the pathologist advised it could not be entirely excluded as this diagnosis is best made under direct observation with bronchoscopy during life. The presence of acute respiratory tract infection (Rhinovirus and CMV) may have exacerbated any symptoms due to associated airway inflammation and swelling. While there was no autopsy evidence of any obstructive complication from the surgical site, the degree of haemorrhage into the airways was difficult to assess particularly following a period of survival. The cause of R's subsequent cardiorespiratory arrest was also unclear and may or may not have been related to the position of the endotracheal tube given the clinical symptoms of falling oxygenation, difficult ventilation and refractory to treatment – this could not be determined at autopsy. I note the pathologist's advice that the presence of evolving diffuse alveolar damage most likely represents a complication of the cardiorespiratory arrest rather than a precipitating factor in the initial post-operative respiratory issues.

The pathologist determined the cause of death to be hypoxic ischaemic encephalopathy as a consequence of postoperative respiratory distress requiring intubation and ventilation complicated by cardiorespiratory arrest of unknown cause as a consequence of cleft palate repair surgery.

Tertiary hospital clinical review outcomes

The tertiary commissioned a Root Cause Analysis (RCA) of the care R received during his admission. This is a systemic analysis of what happened and why and is designed to make recommendations to prevent adverse health outcomes from happening again, rather than to apportion blame or determine liability or investigate an individual clinician's professional competence. It is conducted by a review team or panel who had no involvement in the patient's care.

The RCA panel compromised clinicians with expertise in paediatric plastic surgery, anaesthesia, intensive care (medical and nursing) medicine and patient safety and quality, with members internal and external to the hospital and interstate experts from these fields. The team's analysis was also informed by expert opinion from a respiratory physician, an independent radiologist and an external interstate paediatric intensivist.

I note R's parents were invited to meet with the panel and share their experience, perspective and recall of events. They offered their own questions for the panel's consideration.

I have had the opportunity to consider the final RCA report and recommendations.

The RCA Panel concluded:

- **The anaesthetist's decision to proceed with the anaesthetic on 27 July 2020 was reasonable**

The panel recognised the persistent challenge for paediatric anaesthetists in balancing the risk of mild respiratory symptoms (common in this patient age group) and the need for the child's surgery to proceed. The panel did not find evidence of any clinical indication for R's surgery to have been cancelled.

- **It could not identify a definitive cause for R's prolonged and sustained post-operative respiratory distress**

The panel noted there was consideration of differential diagnoses including aspiration of blood stained oral secretions, negative pressure pulmonary oedema, the possibility of lower respiratory tract infection and tracheobronchomalacia.

I note the panel's consideration of this aspect of R's care was informed by independent paediatric respiratory physician opinion which identified the most likely cause of the initial deterioration as starting with an episode of laryngospasm when R was extubated. The possibility of bronchomalacia causing the event was unlikely because R had no history of chronic stridor or wheeze, and his previous cleft lip surgery was uneventful. The episode of severe upper airway obstruction was thought likely to have resulted in post-obstructive pulmonary oedema (POPE, also known as negative pressure pulmonary oedema) which develops in patients who have spontaneous respiratory effort against an obstructed upper airway. This generates severe negative intrathoracic pressures leading to severe hypoxaemia and pulmonary oedema. The most common cause in adults is post-extubation laryngospasm following surgery; laryngospasm is described to have occurred in 3.1% of children having cleft palate repair. Patients often develop rapid onset respiratory distress after the obstructive event and in severe cases require intubation and ventilation.

- **The management plan in recovery was appropriate – earlier intubation and transfer to PICU would not have affected the outcome for R**

- **R's management in the PICU overnight on 27 July 2020 was appropriate**

- **While the presence of blood/ooze was a challenge for the treating team to manage, it was not a significant abnormal finding post cleft palate repair surgery.**

The decrease in R's haemoglobin overnight on 27 July was considered likely dilutional as R had received two 10mls/kg boluses of 0.9% normal saline between the two haemoglobin results. His hypotension was thought to be related to the level of sedation required rather than indicating another cause.

The panel recognised and agreed R appeared to be on a trajectory of improvement coming into the morning of 28 July.

- **There was a missed opportunity to assess and directly visualise the position of R's endotracheal tube and potentially resecure it in the optimal position for ventilation**

The panel considered the sound R was heard to make and whether it might have been caused by dislodgement of the endotracheal tube or leak around the tube.

The panel was unable to reconcile the differing recollections of when and how R made a sound prior to his bradycardic arrest on 28 July. It noted that if a patient is intubated, they cannot cry or phonate unless the vocal cords are able to come together. The presence of a correctly placed endotracheal tube between the cords would prevent this. However, if there is a leak around the endotracheal tube, sound of the air escaping around the tube may be heard as air can travel up from the lungs around the tube and into the supraglottic areas. This would not be a distinguishable sound like a cry but more likely to sound like gas escaping or a bubbly noise. If the sound was indeed a cry, this could indicate the endotracheal tube was not in an optimal position between and below R's vocal cords and should have triggered urgent review of the tube's position.

Clinicians recalled seeing an EtCO2 trace that provided reassurance R's endotracheal tube was in position. However, the panel identified it is possible to have an EtCO2 trace if the endotracheal tube is sitting above the larynx. It noted that full ventilation assessment requires review of the other parameters of ventilation – tidal volume, the actual EtCO2 value, pattern of EtCO2 trace, arterial blood gases.

- **The most likely cause of R's bradycardic arrest was hypoxia but it was not possible to identify the exact time period in which this occurred or its absolute cause**

- **Capnography was not used during the emergency resuscitation**

EtCO2 monitoring is recognised internationally as recommended practice in assessing CO2 clearance and as an indicator of endotracheal tube position and adequacy of ventilation. This practice is applicable to intensive care units and is a critical clinical indicator, particularly during CPR, for a patient who is intubated. The panel noted it is normal practice to continuously display and monitor EtCO2 in PICU from the ventilator screen. This data was not captured in Metavision but documented by a single value entered manually every hour by nursing staff when undertaking and recording other patient observations.

The EtCO2 trace was disconnected when R was removed from the ventilator, so EtCO2 was not used to monitor CO2 clearance further throughout the resuscitation meaning there is no absolute evidence confirming the tube's position and adequacy of ventilation.

The practice of bagging a patient without capnography was current practice within the PICU. A number of the clinicians involved in the resuscitation felt that having access to R's EtCO2 via the

vital signs monitor during the resuscitation could have improved their ability to assess his clearance of CO2 during CPR and to assess his ventilation.

While there was an option to connect and monitor CO2 on the vital signs monitor, this was not instigated. This would have required a member of the team to collect necessary equipment from the storeroom then connect, set up and calibrate on the monitor prior to use. The panel recognised this was not practical during CPR.

There was also the option to have reconnected and monitored CO2 on the ventilator screen but this was not usual practice in the PICU as this monitoring is difficult to visualise on the ventilator screen.

The panel noted no consideration was given to using the single use EtCO2 monitors on the resuscitation trolleys.

It was noted there are challenges in monitoring EtCO2 for patients whose position has to be rotated to facilitate surgical access for ECMO cannulation, affecting the length of the lead required to reach across the bed. That said, the disposable CO2 monitoring available on the resuscitation trolleys could have been used to mitigate this.

The panel noted work already underway at the time of the RCA to change the process of monitoring EtCO2 in the PICU to ensure that all essential monitoring including EtCO2 will be displayed and monitored from the vital signs monitor.

The panel recommended that continuous capnography should be used for all patients with tracheal tubes who are intubated and ventilator dependent unless there is a clinical exception, and EtCO2 should also be used when a patient requires manual ventilation, disconnection from the ventilator for suctioning and for transport.

I note that as at September 2021, EtCO2 monitoring units providing continuous waveform capnography had been installed to all monitors in use for patients with tracheal tubes who are intubated and ventilator dependent in the hospital. If waveform capnography is not immediately available, a colorimetric CO2 detector can be used until an EtCO2 sensor is available. EtCO2 monitoring is to remain attached to the bag during disconnection from the ventilator, manual ventilation, and suctioning.

- **The persistent gastric dilatation and apparent need for a second oral gastric tube during the emergency resuscitation on 28 July were missed opportunities to trigger assessment and review the position of R's endotracheal tube**

The panel noted that while some abdominal distension is possible during CPR using bag-mask ventilation, in R's case, ventilation occurred using a T-piece anaesthetic bag attached directly to his endotracheal tube.

While the chest x-ray from 7:07am that morning reported the endotracheal tube to be adequately positioned in the mid thoracic trachea, it was noted R appeared to have been positioned with his chin projected slightly down at the time this image was taken. If his head was moved or tilted this could have increased the risk of his endotracheal tube becoming more mobile and move from an adequate position.

The panel identified the abdominal distension could have been an indication the endotracheal tube was either not in an optimal position or was in the oesophagus. There should not be any gastric distension if an endotracheal tube is correctly positioned within the trachea, and it is highly unusual to have to insert a second oral gastric tube during CPR to assist with aspirating air from the patient's stomach. The panel considered this action alone should have prompted further investigation as the cause of the continued gastric dilatation.

- **There was a lost opportunity to review, reposition and/or replace R's endotracheal tube**

The panel noted there were attempts made to reinforce the endotracheal tube tapes with tracheostomy tape prior to ECMO cannulation. While this is recommended practice to secure endotracheal tubes in adult patients, the use of tracheostomy tape for this purpose is unusual practice in paediatrics, particularly for infants in R's age group. A further attempt was made using adhesive tape.

The panel noted with particular concern the apparent need for a nurse to manually hold the endotracheal tube in position for the duration of the EMCO procedure.

A number of clinicians told the panel they were reassured of the position of R's endotracheal tube by the initial CO2 reading from the arterial blood gas taken in the early stages of CPR, hearing air entry upon auscultation on at least three occasions and seeing rise and fall of his chest. The continuous oxygen saturation trace was also reassuring. It was reported as fluctuating anywhere from the high 70s to mid-90s throughout the resuscitation. Minutely saturation observations were captured in Metavision from 11:03am and for the first 18 minutes of CPR, ranging from 25% to 92% with an average value of 64%.

The panel noted clinical literature suggesting auscultation is not a reliable indicator of endotracheal tube placement in adult patients.

The panel acknowledged the clinicians reasonably inferred the CO2 of 46mmHg obtained at 11:12am indicated adequate alveolar ventilation and was therefore an indication of appropriate endotracheal tube position. This suggests some ventilation was occurring at that point in time. However, the panel felt there could have been further concurrent consideration of concerns about the securing R's endotracheal tube and that potentially adequate positioning of the tube had been compromised. If so, CO2 is expected to rise at rate of 12mmHg per minute in the absence of ventilation. The panel noted a significant metabolic (lactic) acidosis with a lactate of 5.8 and a Base Excess of -14.2, possibly indicating R was struggling for an undefined period prior to this.

The panel noted the arterial blood gas was obtained three minutes after CPR commenced. No further blood gas was able to be obtained from that line during the resuscitation. The next blood gas was obtained after R was successfully cannulated and established on ECMO. The panel discussed that a femoral sample could have been attempted to obtain a further blood gas during CPR.

Senior clinicians reflected that they could have considered both monitoring EtCO2 and directly visualising the position of R's endotracheal tube during the resuscitation. There was no overt discussion regarding directly visualising the position of the endotracheal tube or consideration of removing it and reintubating R.

The panel considered that while there was evidence leading the clinicians to infer the endotracheal tube was in a satisfactory position, there were also signs potentially indicating the tube was not in the trachea and these should have triggered further assessment to gain assurance of R's airway. It was not possible to identify exactly when the endotracheal tube migrated to the oesophagus. Independent interstate paediatric intensivist review considered it more likely than not the endotracheal tube was dislodged sometime between 11:54am (when the endotracheal tube was reported as being in the trachea) and 4:39pm, possibly during the transfer once R was established on ECMO. The independent paediatric intensivist considered another scenario whereby the endotracheal tube may have become dislodged at some point before or during the arrest, then become spontaneously reinserted into the trachea for the post-ECMO chest x-ray, only to become displaced again following transfer was less likely. Independent radiologist review of the chest x-ray images confirmed the primary reports regarding the endotracheal tube's placement.

- **The need for extra-corporeal life support for an elective admission for surgery not normally requiring post-operative PICU care was highly unusual and should require the resuscitation team to re-evaluate the cause of the patient's acute deterioration**

- **There were challenges in identifying and establishing roles during the resuscitation, specifically establishing a consistent airway doctor and nurse**

Up to seven PICU senior medical officers either held a role in the resuscitation at some point or were observed to be in attendance, both inside and outside R's room during the resuscitation. The panel identified there was not a consistent airway doctor and nurse until approximately 8-10 minutes into the resuscitation and prior to these roles being established, the clinicians responsible for managing the airway may have changed up to four times. The duty intensivist and a senior PICU nurse were established as the accountable airway clinicians from this point in time and until R was established on ECMO.

The panel identified there appeared to be limited handover between the bedside team and the resuscitation team regarding the course of events leading to R's deterioration and notwithstanding the cognitive load on the resuscitation team, there was suboptimal handover between clinicians swapping in and out of key roles in the resuscitation team.

- **The noise and activity levels during the resuscitation appeared to significantly compromise effective team communication – there was no evidence of graded assertiveness or issues of hierarchy between team members.**

I am advised that senior hospital staff met with R's parents on 8 April 2021 to discuss the issues examined by the RCA process and the recommendations and learning opportunities flowing from it. This included discussion around the actions being taken to implement CO2 monitoring of intubated and ventilated patients and updating hospital guidelines. I note R's parents were given a copy of the RCA Report following this meeting.

In addition to changes made to improve the use of continuous capnography for patients with tracheal tubes who are intubated and ventilator dependent, I note the hospital has also implemented nationally recognised clinical recommendations for airway management in both the PICU and the emergency department, supported with airway training and regular airway practice. The hospital has updated and implemented a single guideline for the management of intubation and ventilation by both medical officers and nursing staff in the PICU. The updated guideline incorporates information about interpreting capnography including during CPR and actions to be taken to identify and manage obstruction and displacement of the endotracheal tube. The guideline was published in March 2022.

Findings required by s.45

Identity of the deceased – [deidentified]

How he died – R died from complications of unexpected significant respiratory distress requiring intubation and ventilation following elective cleft palate repair surgery performed at a tertiary hospital in Brisbane on 27 July 2020. Despite autopsy and a comprehensive multidisciplinary Root Cause Analysis undertaken by the tertiary hospital, the definitive cause of R's initial post-operative respiratory distress is unknown. I note the opinion of an independent paediatric respiratory physician that it may have started with the episode of laryngospasm when R was extubated, causing post-obstructive pulmonary oedema leading to rapid onset respiratory distress. I am satisfied the anaesthetist's decision to proceed with the anaesthetic on 27 July 2020 was clinically appropriate, and the management of R's respiratory distress in recovery and then in the paediatric intensive care

unit overnight was reasonable and appropriate. It is possible hypoxia caused R's bradycardic arrest on 28 July 2020, but the definitive cause has not been identified. Nonetheless there were a number of missed opportunities over the course of 28 July 2020 to assess, review and ensure R's endotracheal tube was correctly positioned to optimise his ventilation, both prior to his sudden arrest and during the subsequent emergency resuscitation efforts, particularly when R's end tidal carbon monoxide (EtCO2) monitoring was disconnected when he was taken off the ventilator during the resuscitation; managing his abdominal distension with a second orogastric tube; efforts to secure his endotracheal tube with tape and the need for a nurse to manually hold the tube in position throughout the ECMO procedure. I am satisfied the tertiary hospital has carried out a very comprehensive analysis of the events leading up to R's death and identified and implemented ways to improve the airway management of patients with tracheal tubes who are intubated and ventilator dependent, including by the use of continuous EtCO2 monitoring and improved guidelines for medical and nursing staff responsible for managing intubated and ventilated patients.

Place of death – Tertiary hospital in Brisbane

Date of death – 31/07/2020

Cause of death - 1(a) Hypoxic ischaemic encephalopathy
1(b) Post-operative respiratory distress requiring intubation and ventilation, complicated by cardiorespiratory arrest of unknown cause
1(c) Cleft palate (surgically repaired)
2 Cleft lip (previous surgical repair); Rhinovirus and Cytomegalovirus(CMV) infection

I close the investigation.

Ainslie Kirkegaard
Acting Brisbane Coroner
CORONERS COURT OF QUEENSLAND
7 June 2022