QUEENSLAND COURTS

CORONERS COURT OF QUEENSLAND

FINDINGS OF INVESTIGATION

CITATION:	**Non-inquest findings into the death of Patient A**
TITLE OF COURT:	Coroners Court
JURISDICTION:	SOUTHPORT
DATE:	30 April 2026
FILE NO(s):	2025/2217
FINDINGS OF:	Melinda Zerner, Coroner
CATCHWORDS:	CORONERS: Obesity, Atrial flutter, oxygen saturations, oxygen therapy, Hypoxaemia, obstructive sleep apnoea, obesity-hypoventilation syndrome, type two respiratory failure

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Introduction

1. Patient A (deceased) was born died on 15 May 2025, at the John Flynn Private Hospital.

2. A doctor from the John Flynn Private Hospital reported Patient A's death to the Coroner because his death was identified as a potential healthcare related death within the definition of a reportable death in the *Coroners Act 2003*. There was a concern regarding the monitoring of Patient A's oxygen saturations and late detection of severe hypoxaemia (a low oxygen level) which may have caused him to go into cardiac arrest.

3. The role of a Coroner is to investigate reportable deaths to establish, if possible, the cause of death and how the person died. The purpose of a coronial investigation is to establish the facts, not to cast blame or determine criminal or civil liability. An investigation is about attempting to find the root cause of the incident that precipitated the death and in appropriate circumstances to analyse systemic failures that contributed to the death and to design remedial responses.

4. In making my findings, they are based on proof of relevant facts on the balance of probabilities. I am not able to make adverse findings against, or comments about individuals, unless the evidence provides a comfortable level of satisfaction that they caused or contributed to the death.

Circumstances of the Death

5. Patient A was morbidly obese. He had had a laparoscopic band and had lost weight from 180 to 160 kg. His BMI was still high at >59. He was considering further bariatric surgery (sleeve gastrectomy).

6. Patient A had a history of sleep apnoea which was treated with a CPAP machine which he had acquired three months prior. He had variable compliance with nightly use. He normally slept semi-upright in a chair.

7. On 9 May 2025, Patient A presented to the John Flynn Private Hospital with exertional shortness of breath and non-productive cough which had started about four days prior. He had arrived at the Gold Coast from Far North Queensland about seven days prior to his admission. It was noted later in his hospitalisation that he had been short of breath for weeks and had received antibiotics for a chest infection.

8. On arrival to the Emergency Department (ED), Patient A was noted to by hypoxic (low oxygen levels) on room air, with saturations of 82-90% (normal is above 94%). He was also in atrial flutter (abnormal heart rhythm) with a rapid pulse of around 150 to 170 beats per minute. He had an elevated D-dimer (blood test used in helping diagnose possible clots in the lung) which resulted in him undergoing an urgent pulmonary angiogram of the lungs. No clots were identified but he had decreased inflation of the lung and a small right pleural effusion (fluid in the lining of the lung). A screen for respiratory viruses, including COVID was negative. He did have an elevated Troponin which is a marker of heart muscle damage, and elevated liver function tests. His blood gas result indicated he likely was a chronic carbon dioxide retainer with a carbon dioxide level of 67.

9. Patient A was admitted to the Coronary Care Unit and was commenced on cardiac monitoring. His atrial flutter was treated with intravenous medications, but it did not improve. He was prescribed a blood thinner with a plan for electrical cardioversion. An ultrasound was first though required to rule out a blood clot in his heart.

10. Patient A remained in atrial flutter, but his pulse slowed to the low 100s when he was changed to another medication. It was noted when he changed position, particularly with his head falling forward, his oxygen saturations would fall. He was not always compliant with his use of oxygen.

11. On 12 May 2025, Patient A was noted to have very low saturations when he was not using oxygen (77-80%), improving to the low 90s when he had the oxygen mask in place. Nurses tried to assist Patient A by resting the oxygen mask on his chin with an increase in the rate of the oxygen delivery. This resulted in an improvement to his oxygen saturations. He was noted to have frequent apnoeic episodes when he was asleep.

12. On 13 May 2025 at 2am, Patient A was incontinent of urine and at 3am he was observed to be nauseated and sweating heavily. While orientated to time and place, he was having difficulty following instructions from staff. It was documented that he 'needed to be monitored closely'. Another entry in the notes refer to Patient A as being agitated and disoriented. The oxygen saturations were recorded every 2-4 hours.

13. At 5.30am, an emergency code was called due to Patient A developing a low heart rate and then going into an asystole (no heart rhythm) cardiac arrest. Resuscitation was difficult and there was a delay in establishing his airway. A definitive endotracheal tube was not in place until 30 minutes after he went into cardiac arrest. An improvement in his colour and saturations only became recordable after he was intubated. An echocardiogram did not reveal any concerns, but his Troponin level peaked at 383.

14. Patient A was transferred to the Intensive Care Unit (ICU). He was difficult to ventilate due to his size and adequate oxygen saturations could only be achieved when he was receiving 100% oxygen. He had cardiogenic shock which was treated. He required urgent dialysis. An echocardiogram showed severe left ventricular dysfunction and a dilated right ventricle, moderate regurgitation of tricuspid and mitral valves, and aortic valve sclerosis.

15. Patient A had no meaningful neurological improvement. A CT brain showed he had developed severe cerebral oedema with pressure causing bulging of the brain.

16. On 15 May 2025, following discussions with Patient A's family, it was decided to withdraw active medical support, and at 6.30pm Patient A was declared deceased.

17. On 22 May 2025, a nurse wrote a retrospective entry for 11 May 2025 at 9pm. She states,

> *Phoned Dr approx. 2030hrs RE: pt desaturating when head forward or lungs constricted by body position. Dr Stated pt has severe sleep apnoea & to keep him as upright as possible, keep oxygen on & monitor sats overnight. Same attended and handed over. Bariatric bed awaiting arrival. Pt frequently removing oxygen therapy. Encouraged pt to leave oxygen insitu. Same reapplied.*

Preliminary Medical Opinion

18. To guide the investigation an opinion was sought from a Forensic Medicine Physician, Dr Samantha Duncan. She reviewed Patient A's medical records and provided a summary of events. In conclusion she advised,

> *This was a man with a deranged background cardiorespiratory physiology. It would have been important to titrate his oxygen delivery to his oxygen saturations. There were competing demands of not removing hypoxic respiratory drive (the drive to breathe at a normal rate in someone who was persistently raised carbon dioxide levels) against oxygenating a heart which was struggling with a rapid arrythmia. The treating team could reflect on the path chosen. However, a patient such as Patient A's ought to have had continuous saturation monitoring, especially in a CCU environment.*
>
> *When he was becoming agitated and confused, medical review was warranted as well as another blood gas level as it was likely at that time that he had rising carbon dioxide levels and decreasing effective respiratory effort. Although difficult to state with certainty if this would have been outcome changing, earlier recognition and treatment, including positive pressure ventilation, may have prevented his subsequent arrest.*

19. The Cause of Death as stipulated by the clinicians is not disputed by Dr Duncan. That is, the Cause of Death is brain death due to or as a consequence of cardiorespiratory arrest, due to or as consequence of ~~s~~evere hypoxaemia due to or as a consequence of obstructive sleep apnoea.

Statements

20. I obtained statements from the nurse who had primary care of Patient A on the afternoon shift of 11 May 2025, and the night shift of 11/12 May 2025. I also obtained a statement from Patient A's treating cardiologist.

21. The treating cardiologist has advised,

    a. There were no written orders made for oxygen administration, but he says during the ward round he gave verbal instruction to continue supplemental oxygen by either facemask or nasal prongs to maintain adequate oxygen saturation.

    b. His attention had not been drawn the venous blood gases which had been performed on admission. He says it is possible as they were performed in the ED, that they had not been filed in the paper chart in the Coronary Care Unit when he reviewed Patient A.

    c. He considers the most likely cause for Patient A's increased oxygen requirements was a combination of his pre-existing obesity-hypoventilation syndrome and his cardiac dysfunction due to the arrhythmia.

    d. He does not recall being contacted by the CCU staff following his morning round on 11 May 2025 regarding Patient A's increased oxygen requirements prior to the cardiac arrest.

e. He believes the cardiac arrest was most likely caused by the combination of hypoxaemia due to pre-existing obesity-hypoventilation syndrome, and the bradycardic effects of the beta-blockers which were administered to prompt restoration of sinus rhythm.

22. The nurse caring for Patient A on the afternoon shift disputes the cardiologist was not contacted. She says at 8.30pm she notified the cardiologist of Patient A's increased oxygen requirements. She was concerned about Patient A's ongoing noncompliance with the oxygen delivery devices and that Patient A's oxygen saturations were below 90% without oxygen, when he was not sitting upright. She recalled the cardiologist told her that Patient A had severe sleep apnoea and advised her to try and keep him as upright as possible, to keep the oxygen on overnight and to continue to monitor his oxygen saturations. He did not specify the amount of oxygen therapy.

23. As noted above, there was a retrospective entry by the nurse following Patient A's death referring to her discussion with the cardiologist. This note was provided to the cardiologist to seek his response. He advised,

> *That the clinical note was made only in retrospect, I think reflects the degree of concerns that the nursing staff had regarding his clinical status at the time, and I do not believe that the severity of his situation was effectively relayed to me. There was certainly no mention of respiratory distress or cognitive impairment in the clinical notes or written statement, and if I gave advice to ensure that he continued to have supplemental oxygen delivered, with an upright posture to maintain his airway, this would have seemed appropriate to me in the circumstances. If he had continued to desaturate despite the measures advised, then I consider it would have been appropriate for the nursing staff to have initiated a MET (Medical Emergency Team) call, which would have prompted urgent review by the intensive care medical officer on call.*

Respiratory and Sleep Physician Expert Opinion

24. Dr Lau, a respiratory and sleep physician who is the Director of Thoracic Medicine and Director of Sleep Medicine at the Royal Brisbane and Women's Hospital was asked to review the care provided to Patient A, and to provide his expert opinion concerning that care.

25. Dr Lau has opined,

    a. The results from the initial venous blood gas obtained in the ED was consistent with chronic, compensated type 2 (hypercapnic) respiratory failure, rather than an acute respiratory process.

    b. In the absence of prior correspondence from a sleep physician or available diagnostic sleep studies, and in the context of the deceased's morbid obesity, these findings are clinically significant in that they strongly support a diagnosis of obesity hypoventilation syndrome.

    c. Given the absence of significant findings on the CT pulmonary angiogram, these findings suggest Patient A was experiencing obesity-hypoventilation syndrome. The atrial flutter alone would not have caused the degrees of hypoxaemia, nor the presence of chronic hypercapnia, particularly in the absence of pulmonary oedema on cross-sectional imaging.

d. The elevated partial pressure of carbon dioxide was also clinically significant because, in accordance with the hospital's Oxygen Therapy guideline, such a result would generally prompt confirmation with an arterial blood gas and consideration of a controlled oxygen strategy, typically targeting oxygen saturations in the range of 88-92 per cent for patients with hypercapnic respiratory failure.

e. The likely explanation for Patient A's increasing oxygen requirements in the lead up to the cardiac arrest was progressive decompensation of untreated obesity-hypoventilation syndrome, with worsening type 2 respiratory failure.

f. There is limited evidence to suggest that worsening arrhythmia was the primary driver for the need for increased oxygen. The documented heart rate was improving.

g. There appeared to be limited recognition of chronic hypercapnia respiratory failure as the primary pathology, and of its subsequent decompensation as the principal driver of Patient A's clinical deterioration.

h. While the management of obesity-hypoventilation syndrome would not ordinarily fall within the scope of cardiology practice, earlier involvement of respiratory and sleep medicine services may reasonably have assisted in recognising and addressing the underlying cause of the deceased's respiratory failure.

i. Overall, the available records suggest a system-wide under recognition of hypercapnia respiratory failure, its physiological consequences, and the need for targeted management, noting that supplemental oxygen alone does not treat hypercapnia respiratory failure.

j. There also appears to have been limited recognition of more subtle changes in the deceased's mental state. Difficulties maintaining oxygen therapy and reduced ability to sit upright in a recliner may have represented early indicators of worsening hypercapnia and impaired cognition.

k. Considerable efforts were made by nursing staff to address Patient A's low oxygen saturations through adjustments to monitoring equipment and oxygen delivery devices. They though were ineffective and at times led to over-oxygenation. The emerging confusion may have been an early indicator of physiological deterioration. Further escalation and more targeted clinical review may reasonably have altered the clinical trajectory.

26. In conclusion Dr Lau says he agrees with the Cause of Death, and states,

> *In summary, the available medical evidence supports that obesity hypoventilation syndrome was the most likely primary pathology underlying the deceased's presentation, with subsequent decompensation of chronic hypercapnic respiratory failure accounting for the progressive clinical deterioration. In my opinion, the atrial flutter observed during the admission is more likely to have represented a consequence of chronic hypoxaemia, rather than its primary cause, although it may have contributed to further physiological destabilisation. In the absence of alternative explanatory pathology, this case highlights the importance of recognising hypercapnic*

respiratory failure as a distinct clinical entity and considering targeted management strategies beyond supplemental oxygen alone.

Cardiologist's Response to the Expert Opinion

27. The cardiologist was provided the opportunity to provide a response to Dr Lau's opinion. In general, he is in agreement with Dr Lau's conclusions but finds it difficult to accept the arrhythmia was not a least a contributing factor to Patient A's deteriorating clinical status, and states,

    I would, however, agree in hindsight that obesity-hypoventilation syndrome was likely the dominant factor in his clinical deterioration and subsequent cardiac arrest. The arrhythmia does not appear to have been a complication of the chronic ventilatory issues, and I still consider it was the proximate cause of his clinical presentation.

28. The cardiologist maintains his view that he was not aware of the blood gas taken in the ED. He suggests in hindsight a case could have been made for Patient A to have been admitted directly to the Intensive Care Unit on the night of his presentation to the hospital. He did not become aware of Patient A's admission to the following morning at 7am when he was provided a brief handover from the night duty medical officer. The only issues were that Patient A's peripheral oxygen saturations fell when he removed his oxygen mask.

29. The cardiologist states,

    There was discussion during my initial ward round regarding the use of his CPAP machine, with he and his wife informing me that he was not using it at home and had not brought it with him to the Gold Coast when he travelled from Far North Queensland. I was informed that he had advised nursing staff that he would have refused to use it even if one had been supplied, and in that context, I interpreted his refusal to keep his oxygen masked in place as non-compliance with therapy, rather than altered consciousness. At no time were any concerns raised with me about his cognitive status, and at the time of my clinical reviews, which on both occasions occurred during morning ward rounds, he had been alert and cooperative. I note Patient A's family's description, in their submission to the Office of the Health Ombudsman (OHO), of episodes of extreme respiratory distress through the night, which were not communicated to me by the nursing staff, and which, if I had been made aware, would certainly have prompted me to request review by the intensivist on-call with a view to having him transferred to the intensive care unit for assisted ventilation.

30. In conclusion, the cardiologist says he has reflected on Patient A's clinical management and agrees there was a lack of recognition of the significance of obesity-hypoventilation syndrome and hypercapnia respiratory failure. He states,

    Although I am prepared to accept my share of responsibility for the poor outcome, I do not consider that I was effectively informed of his clinical deterioration during the night of 11th May. Again, my deepest condolences and sympathy go out to Patient A's family, and I am hopeful that subsequent measures taken by both myself and the hospital will minimise the future possibility of such outcomes.

31. I note the family have lodged a complaint about the cardiologist to the Office of the Health Ombudsman (OHO). I have been advised the complaint has been referred to Ahpra (Australian Health Practitioner Regulation Agency). A conciliation case remains open with the OHO.

Statement from the ED Physician

32. The Emergency Medicine Consultant who reviewed Patient A and arranged for his transfer to the Coronary Care Unit was asked to provide a statement. She says,

    a. Patient A presented with a history of shortness of breath on exertion for 4-5 days following a recent flight from Far North Queensland. He had a dry cough, no fever and on questioning, advised he had no chest pain. The only history offered was obesity with recent weight loss.

    b. On arrival, Patient A's oxygen saturations were reported at 86% which improved to 90% on three litres of oxygen via nasal prongs. His heart rate was 170 (high) and his blood pressure was 137/100 (high). He was determined to be in atrial flutter from his ECG.

    c. Investigations were organised which included a Troponin level (68); venous blood gas (pH 7.36, pCO2 67, Bicarb 38, and Base Excess 9.8); CT pulmonary angiogram (no pulmonary embolism); a respiratory viral PCR (negative).

    d. Based on her assessment and history, Patient A's acute symptoms of shortness of breath on exertion were due to atrial flutter and that he had likely been in that rhythm for 4-5 days prior to his presentation to the ED. She states,

        Patient A's compensated respiratory acidosis on his venous blood gas in the absence of significant lung disease I presumed was secondary to obesity. The respiratory acidosis indicated by the high PCO2 was compensated by raised bicarbonate which would suggest it was not an acute abnormality. Patient A's gave no history of significant lung disease nor did the CTPA show and underlying lung disease.

        My management plan for Patient A's was aimed at what I felt were his acute presenting symptoms related to atrial flutter. I managed Patient A's in the ED with rate control, administration of intravenous magnesium sulphate and oral Metoprolol and anticoagulation with Enoxaparin administered subcutaneously. Cardioversion would risk stroke in a patient with symptoms for 5 days. I asked the ED Registered Nurse (RN) in charge of the shift in ED to book a bed in the Coronary Care Unit. The RN in-charge contacted the Hospital Coordinator to book the bed in the CCU. I opted not to contact the Cardiologist at 11pm to provide a handover as I did not anticipate a clinical deterioration overnight or that the Cardiologist would suggest different management overnight, based on my observation of Patient A's in the ED and the findings of the investigations conducted in the ED.

    e. The calling criteria was altered to oxygen saturations of 90% on 3 litres of oxygen in view of his raised pCO2 which would mean a clinical review should his oxygen saturations drop below 90% on 3 litres of oxygen. She recalls the

blood gas results prompted her decision to make this alteration to the calling criteria.

f. In her view, admission to ICU was not required as Patient A did not require any additional support such as BiPAP/high flow oxygen or inotropes. She considered oxygen saturations of 90% on low flow oxygen to be adequate for a patient with chronic CO2 retention. The CCU was a higher dependency level of care than on the ward and she felt that was appropriate at the time she reviewed Patient A.

g. The blood results were available in paper and available in conjunction with her notes for the cardiologist to review.

33. In conclusion she states,

> *My assessment of Patient A's was that he had an acute presentation of shortness of breath on exertion for 4-5 days and I considered this to be due to Atrial Flutter. The Venous blood gas showed a compensated respiratory acidosis potentially secondary to hypoventilation of obesity, however, I admitted Patient A's to the Coronary Care under Cardiology for the management of his acute presentation.*

Hospital's Response

34. I wrote to the hospital as I had had a similar case involving issues with oxygen administration and monitoring with another hospital within the same group of hospitals, in which I published findings on 19 July 2024[1]. In those findings the hospital escalated the issue to the governance team, to look at it nationally. I wanted to establish what progress had been made and if it had been adopted by the John Flynn Private Hospital.

35. The change to the oxygen administration policy has been a protracted process, but significant progress has been made. I have been provided with a detailed timeline of actions which have been taken since I raised my concerns regarding the death of Mr K. In summary,

a. The National Clinical Governance Unit (NCGU) determined a national oxygen administration policy was required which, once approved for release, would replace the local policies used by individual hospitals.

b. It was necessary to consult the Australian Commission on Safety and Quality in Health Care (ACSQHC) because the medication charts used by this group of hospitals was developed by the ACSQHC.

c. In March 2025, the NCGU developed an audit tool and undertook an audit to determine whether a sticker or an embedded section on the medication chart was the best method of capturing an oxygen prescription. A sample sticker was subsequently developed and sent to a select group of hospitals for trial.

d. In May 2025, a draft national policy was sent out for consultation to various stakeholders.

[1] https://www.coronerscourt.qld.gov.au/__data/assets/pdf_file/0004/805891/non-inquest-findings-public-interest-matter.pdf

e. The ACSQHC agreed to the sticker being applied to the medication chart.

f. In July 2025, changes were made to the policy following feedback including a targeted assessment utilising the Targeted Oxygen Assessment & Documentation Sticker and the sticker was simplified. A training package and other educational material was also being developed.

g. In September 2025, further changes were made to the draft policy when it was tabled at the relevant sub-committee meeting.

h. In January 2026, the policy was updated to include requirements for the immediate post-operative patient. It was again sent out for consultation. It was hoped the policy and associated documents would be published in April 2026. The policy and associated bundle of documents include:

    i. The national policy, which will replace any local policy.

    ii. A prescription sticker, which is to be used by on PBS Acute and Long Stay Medication Charts.

    iii. A new standalone 'track and trigger' observation chart – CO2 retainer with a Y/N question.

    iv. A nursing self-directed learning package.

    v. Discontinuance of oxygen therapy.

    vi. Adverse outcomes of oxygen therapy i.e. hypercapnic respiratory failure (signs, symptoms and management).

    vii. Other patient risks, for example falls or pressure injuries.

    viii. Documentation of oxygen management, inclusive of CO2 retainers, review time frames and oxygen device management.

    ix. Oxygen therapy and management during transfer of care.

    x. Risks associated with oxygen use.

    xi. Consumer engagement and experience considerations.

    xii. Indwelling Pleural Catheter considerations.

36. The author of the correspondence states,

> *I wish to assure your Honour that the Hospital is committed to implementing the national policies as directed by Ramsay. It is important to note that this represents a significant shift in current practice and will require substantial change management. The requirements involve not only nursing staff but also a large cohort of accredited practitioners, up to 500 individuals who are not employees of the hospital but independent contractors. Embedding these changes across such a broad and diverse group will be complex and will take time to implement safely and effectively. The Hospital fully supports the intent of*

the policies and will work towards full compliance.

37. I have been further advised that considering Patient A's passing, the NCGU has updated the draft oxygen policy to include a section specifically for bariatric patients. In the interim the hospital has carried out a structured education program on oxygen administration which has been delivered to the Coronary Care Unit nursing staff.

Conclusion

38. After considering the material obtained during the coronial investigation, I consider I have sufficient information to make the necessary findings required by s 45(2) *Coroners Act 2003* in relation to Patient A's death.

39. I consider the actions by the ED physician to have been reasonable in the circumstances. Patient A was stable with oxygen prescribed with a target saturation range and that Patient A was for review if his oxygen saturations dropped below 90%. There were no issues overnight.

40. I find there were some clinical errors and oversight in this case as Patient A's oxygen requirements changed. I agree with Dr Lau that there were some system wide issues with an under appreciation of what was occurring for Patient A. Unfortunately, he was not provided with appropriate treatment for his obesity hypoventilation-syndrome and subsequent hypoxaemia. I acknowledge relevant information may not have been conveyed to the cardiologist when contacted by nursing staff in the evening of 12 May 2025, and that he was not again contacted despite continued concerns about Patient A's condition. Patient A should have been medically reviewed during the late evening, night shift when nursing staff were concerned about Patient A's oxygen saturations and when Patient A had started to become confused.

41. I accept Dr Lau's opinion that with further escalation and more targeted clinical review, it may reasonably have altered Patient A's clinical trajectory. That is, I find it is unlikely Patient A would have gone into cardiac arrest and died had appropriate assessment and treatment have been provided in a timely manner. I accept the Cause of Death certificate as recorded by the hospital clinician and which was endorsed by Dr Lau.

42. The cardiologist has been referred to Ahpra by OHO and I am satisfied the hospital is undertaking considerable efforts to change practice at a national level to ensure more safeguards are in place concerning the administration of oxygen, particularly in circumstances where a patient is a carbon dioxide retainer. On this basis I am not satisfied that it is in the public interest to hold an Inquest (formal court hearing). I do though consider it would be helpful to publish these findings to once again highlight the dangers associated with inappropriate oxygen administration. I have therefore sought the consent of Patient A's family to publish a version of these findings which does not identify Patient A.

43. I extend my condolences to Patient A's family and friends for their loss. To lose someone in such unexpected circumstances is very difficult. I recognise there are no words which can adequately express the depth of your sorrow, or the profound impact Patient A's loss has had on you all.

Findings required by s.45

Identity of the deceased – Patient A

How he died – 1(a) Complications of obstructive sleep apnoea and obesity hypoventilation syndrome
2 Morbid obesity

Place of death – John Flynn Private Hospital 42 Inland Drive TUGUN QLD 4224 AUSTRALIA

Date of death– 15/05/2025

I close the investigations.



Melinda Zerner
Coroner
CORONERS COURT OF QUEENSLAND - BRISBANE OFFICE
30 April 2026