



# MAGISTRATES COURT of TASMANIA

## CORONIAL DIVISION



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## Record of Investigation into Death (Without Inquest)

*Coroners Act 1995*  
*Coroners Rules 2006*  
*Rule 11*

I, Rod Chandler, Coroner, having investigated the death of Maurice Cecil Nancarrow

### **Find, that:**

- a) The identity of the deceased is Maurice Cecil Nancarrow;
- b) Mr Nancarrow was born at Winchester in the United Kingdom on 30 August 1938 and was aged 76 years;
- c) Mr Nancarrow died on 15 August 2015 at the Royal Hobart Hospital (RHH) in Hobart;
- d) The cause of Mr Nancarrow's death was multiple organ failure due to a large volume aspiration of gastric contents that occurred during anaesthetic induction for rigid cystoscopy.

### **Background**

Mr Nancarrow was a retired salesman and manager. He resided with his wife Joan at 59 Blair Street in New Norfolk. They were married for 58 years and had five children, now all adults. In February 2013 Mr Nancarrow had a Hartmann's procedure with stoma formation to treat his recurrent diverticulitis. Relevantly, Mr Nancarrow was referred to the RHH's urology service on 19 May 2015, with symptoms of bladder outlet obstruction.

### **Circumstances Surrounding the Death**

On 30 July 2015 Mr Nancarrow was admitted to the RHH for surgery to reverse the Hartmann's procedure. The surgeon was Mr Srinu Yellapu. The surgery was not straightforward because of adhesions. The initial laparoscopic procedure was converted to open surgery. There was an inadvertent injury to the spleen which required repair. There was also an accidental serosal tear that required repair.

Post-surgery Mr Nancarrow was progressing well when reviewed in the morning of 31 July. However, that afternoon he complained of being dry and thirsty. His pulse rate rose to 115 bpm in a regular rhythm. His blood pressure was stable. Urine output was reduced. He was treated with an intravenous fluid bolus and an increased rate of intravenous maintenance fluids. He was reviewed at 10.30pm. The tachycardia had persisted. His temperature was slightly elevated. Urine volume had increased.

In the morning of 1 August Mr Nancarrow was found to have a distended abdomen. There had been minimal draining of intra-abdominal fluid. Blood tests continued to show an increased urea compared to creatinine. The albumin level had fallen to 22 mg/L and the CRP had risen to 341 mg/L. In the early afternoon Mr Nancarrow was reviewed by a surgical registrar who increased the intravenous fluid input. Nursing staff reported Mr Nancarrow to be intermittently drowsy during the day. His oxygen saturations fell to 87% despite an increased concentration of supplementary inspired oxygen. When his adult deterioration detection score (ADDS) was recorded at four and oxygen saturations continued to fall, a review by a surgical intern was requested. Crackles were heard in the lung fields. The abdomen remained distended. A chest x-ray was reported to show no pulmonary oedema. Mr Nancarrow was treated with a diuretic for fluid overload. However, he continued to deteriorate and a medical emergency team (MET) call was made at 7.45pm. Oxygen saturations had fallen to 82%. A further chest x-ray was reported to show bibasal atelectasis. A large gastric bubble was noted. The stomach was drained. At this point Mr Nancarrow was intubated as a precaution against aspiration of gastric contents and he was transferred to the intensive care unit (ICU). Standard ICU support was commenced with mechanical ventilation, inotropes, antibiotics and the insertion of a naso-gastric tube.

In the morning of 2 August a clinical examination identified four issues, namely: sepsis, over-sedation, possible adrenocortical insufficiency and respiratory failure due to bibasal atelectasis. Significant doses of corticosteroids were commenced.

Over the following two days there was some improvement in Mr Nancarrow's condition and he was transferred to the surgical ward. However, problems persisted. A sputum culture grew three different organisms consistent with aspiration pneumonia. Mr Nancarrow complained of swallowing difficulties associated with hiccups which caused abdominal pain. An ECG on 6 August showed acute changes in the septal leads consistent with myocardial ischaemia. Renal function was normal with a creatinine level of 61 micromoles/l. On 7 August Mr Nancarrow was afebrile. Inflammatory markers continued to indicate progressive sepsis. There was renal failure with a rise in the creatinine level to 172 micromoles/l. The fluid balance chart showed multiple small urine volumes.

A CT scan on 10 August was reported as: *"Conclusion: Small amount of free fluid in the left perisplenic space. No evidence of acute diverticulitis. Left sided hydronephrosis and hydroureter with possible 7.8mm left vesicoureteric junction calculus. Assessment is difficult due to significant beam hardening effect in the pelvis."*

On 12 August Mr Nancarrow was reviewed by Mr Yellapu. Left lower abdominal pain was considered to be due to renal calculi. The nursing notes made on 12 August state that Mr Nancarrow was able to mobilise with assistance, he was tolerating diet and fluids and his loose bowel actions had slowed in frequency. It was noted that his blood pressure had dropped to 90 (systolic) after his shower that day but this returned to within normal limits once he returned to bed. His white cell count was high.

During the evening Mr Nancarrow's condition deteriorated. He had become hypoxic and tachypnoeic. He had a medical review at 9.30pm. It was noted that his breathing was laboured and the jugular venous pressure was elevated. The abdomen was distended and hyper-resonant. He was treated with a diuretic. At midnight blood measurements showed

creatinine of 194 micromoles/l with other markers of acute renal failure. The haemoglobin level had fallen, the neutrophil count was high as was the platelet count. A diagnosis of fluid overload was made and a diuretic administered.

The following day a chest x-ray showed further deterioration with extensive consolidation on the right side with sub-segmental collapse and volume loss. Less extensive changes were on the left-hand side. Because of his apparent progressive sepsis (thought to be attributable to the blocked left ureter), and hydronephrosis Mr Nancarrow was booked for the placement of a double J catheter, a tube device which is placed in the ureter and is intended to permit the drainage of urine from the kidney into the bladder. However, upon induction of the anaesthetic Mr Nancarrow had a large volume aspiration that led to the procedure being aborted and Mr Nancarrow's return to ICU. Over the following days his condition slowly deteriorated and he died at 10.40am on 15 August 2015. Post-operative findings recorded by Dr Fadi Nuwayhid, the Director of Urology at the RHH, confirmed the presence of a chronic bladder neck obstruction. It had not been possible during the procedure to determine whether the left ureter was blocked.

### **Post-Mortem Examination**

This was undertaken by forensic pathologist, Dr Donald Ritchey. In his opinion the cause of Mr Nancarrow's death was multiple organ failure due to a large volume aspiration of gastric contents that occurred during anaesthetic induction for rigid cystoscopy.

I accept this opinion upon the cause of death.

### **Investigation**

This has been informed by:

1. An affidavit provided by Mrs Nancarrow.
2. Reports provided by Mr Yellapu, Dr Fadi Nuwayhid, and Clinical Associate Professor Andrew Turner, Director of Critical Care Medicine at the RHH.
3. A report from Dr Helen Harris, Deputy Executive Director of Medical Services at the RHH.
4. A review of Mr Nancarrow's records at the RHH carried out by research nurse, Ms L K Newman.
5. A radiology review undertaken by Dr Pip Taplin of Radiology Tasmania.
6. A report upon Mr Nancarrow's medical care and management compiled by Dr A J Bell as medical adviser to the coroner.
7. Discussion at meetings attended by myself, Dr Bell, Ms Newman, Dr Ritchey and State Forensic Pathologist, Dr Christopher Lawrence.

At this point it is necessary for me to record that Dr Taplin's report upon the CT scan of 10 August 2015 differs from the RHH report in several respects. Firstly, it describes the bladder as *"large in volume."* This contrasts with the RHH report which made no reference to the bladder size. Secondly, it takes issue with the suggestion that calculus was present in the left ureter. Instead Dr Taplin says the *"calcification described on the RHH report is unlikely to lie within the distal left ureter"* but acknowledges the difficulty in being certain *"due to the severe beam hardening artefact in the pelvis."* Finally, it records: *"In my opinion, the degree of prominence of the left renal collecting system and the left ureter is not significantly different from that on the right side."* It is my understanding that this finding is inconsistent with a diagnosis of unilateral hydronephrosis.

In his report Dr Bell expresses these opinions:

- The diagnosis of fluid overload made on 1 August was incorrect. Rather, by this date Mr Nancarrow had developed an ileus; that is a non-working bowel which had become distended with air and fluid.
- Sometime during 1 August Mr Nancarrow aspirated (inhaled foreign matter into the lungs) and then developed bilateral lung collapse which was confirmed by CT scan.
- Mr Nancarrow was appropriately treated in ICU up to his discharge on 4 August. By this time his ileus had resolved. Although not recorded at discharge by the ICU Mr Nancarrow's working diagnosis for his future management at this point should have been aspiration pneumonia.
- From 7 August 2015 Mr Nancarrow developed progressive renal failure as evidenced by an increasing creatinine level (a reading on 10 August showed a 2/3<sup>rd</sup> loss of renal function) and his need to pass multiple small volumes of urine as recorded on the fluid balance chart. However, there was a delay in attempting to determine its cause. In particular there was no consideration of bladder neck obstruction, a common cause of renal failure in elderly males.
- The CT scan of 10 August was misreported in the two respects identified by Dr Taplin. In Dr Bell's opinion the scan showed the bladder to be abnormally large.
- Had the large volume bladder been reported it should have led to consideration of a diagnosis of bladder neck obstruction. This in turn should have led to Mr Nancarrow's bladder being drained via a catheter.
- The report of the CT scan of 10 August caused the clinical teams to wrongly believe that the cause of Mr Nancarrow's renal difficulties was a blockage of the left ureter caused by renal calculus (kidney stone).
- By 13 August Mr Nancarrow was seriously ill. Chest x-ray showed significantly increased consolidation changes, especially in the right lung. This clearly evidenced aspiration pneumonia but this diagnosis was not considered. Instead a

wrong diagnosis of fluid overload was maintained and treatment with diuretics continued.

- By 13 August Mr Nancarrow required re-admission to ICU for urgent treatment of his aspiration pneumonia.
- The diagnosis of bladder neck obstruction should have been made, at least by 10 August 2013, particularly in light of the escalating creatinine levels, large bladder and the equal sized renal collecting systems.
- The surgery undertaken on 13 August would probably not have been required if Mr Nancarrow's bladder neck obstruction had been diagnosed and treated.

### **Findings and Comments**

I accept the opinion of Dr Ritchey upon the cause of death.

I am mindful that Dr Bell has been aided by hindsight in his consideration of this case. Nevertheless, I am satisfied and accept that Dr Bell has identified a number of shortcomings in Mr Nancarrow's management at the RHH which should not have occurred and which represent a substandard level of care. These are:

1. The failure to investigate the cause of Mr Nancarrow's progressive renal failure.
2. The failure to report upon the abnormally large bladder shown on the CT scan of 10 August 2015.
3. Reporting the possible presence of calculus in the left ureter supposedly shown on the CT scan of 10 August, when the calcification more likely lay outside the ureter.
4. Misdiagnosing Mr Nancarrow to have a left ureter blocked by calculus when he in fact suffered from a bladder neck obstruction.
5. Failing to diagnose Mr Nancarrow with aspiration pneumonia, evident by 13 August, and initiating appropriate treatment.

It is my understanding that once diagnosed bladder neck obstruction can be simply and successfully treated. If this had been achieved in this instance it is likely that the surgery embarked upon on 13 August would have been unnecessary and the aspiration that eventually led to Mr Nancarrow's death would have been avoided. It therefore follows that in all likelihood Mr Nancarrow's unfortunate death would have been prevented if his bladder neck obstruction had been promptly diagnosed and treated.

I have decided not to hold a public inquest into this death because my investigation has sufficiently disclosed the identity of the deceased, the date, place, cause of death, relevant circumstances concerning how his death occurred and the particulars needed to register his death under the *Births, Deaths and Marriages Registration Act 1999*. I do not consider that

the holding of a public inquest would elicit any significant information further to that disclosed by the investigation conducted by me.

I convey my sincere condolences to Mr Nancarrow's family and loved ones.

**Dated:** 19 June 2017 at Hobart in the State of Tasmania.

**Rod Chandler**  
**Coroner**