



MAGISTRATES COURT of TASMANIA

CORONIAL DIVISION



Record of Investigation into Death (Without Inquest)

*Coroners Act 1995
Coroners Rules 2006
Rule 11*

I, Rod Chandler, Coroner, having investigated the death of Mr M

Find, pursuant to section 28 (1) of the Coroners Act 1995, that:

- a) The identity of the deceased is Mr M;
- b) Mr M was born in Devonport in 1971 and was aged 41 years;
- c) Mr M died on 12 September 2012 at the Royal Hobart Hospital (RHH) in Hobart; and
- d) The cause of Mr M's death was a large volume thrombotic stroke within the distribution of the right posterior cerebral artery and posterior inferior cerebellar artery. Significant contributing factors were atherosclerotic and hypertensive cardiovascular disease and emphysema.

Background

Mr M was married to CM. They had a daughter, T. Mrs M was also pregnant with their second child at the time of her husband's death. Mr M was employed as a plumber. He enjoyed good health. However, on 4 September 2012 he told his wife that he had had a dizzy spell at work that day and the following day he complained of a headache. He was a long-term cigarette smoker.

Circumstances Surrounding the Death

At around 7.45am on 6 September 2012, Mrs M noted that her husband had not returned indoors after taking out the rubbish bins. She went outside and found him lying on the front lawn. He appeared unconscious but was breathing. She went inside, called an ambulance and then returned to her husband. He then began "*thrashing about.*" By the time the ambulance arrived Mr M had "*come to*" but he was unable to walk and did not have any feeling down his left side. He was immediately taken by ambulance to the North West Regional Hospital (NWRH).

An examination was undertaken in the Emergency Department (ED). It was recorded that Mr M was drowsy, sleepy and easily roused. Muscle tone was normal. Muscle power on the right side was normal. Power on the left lower and upper limbs had decreased to 4/5. Reflexes were normal; the plantar response was normal on the right side but 'neutral' on the left. There was a loss of left sided sensation and it was recorded, "*unable to perform co-ordination tests.*" The differential diagnosis was noted as: '*seizure, post ictal, intracranial*

pathology, Todd's paresis." The treatment plan was stated to be "1. Bloods. CT non contrast. 2. If ok outpatient MRI and EEG (forms faxed)."

The CT scan of the brain demonstrated a "Normal non-contrast CT brain. No focal lesion seen as a focus of seizure activity." Particularly, there was no evidence of haemorrhage. There was a lumbar puncture in the mid-afternoon. It was reported as normal.

A medical registrar saw Mr M that afternoon. It was noted that he had a two day history of upper respiratory tract infection and that in the morning he had felt dizzy and had started vomiting after he left the house for work. He had laid on the ground and his wife found him some minutes later. Mr M stated that he could hear his wife speaking but was unable to respond to her. An examination revealed decreased power to the left limbs and possible decreased sensation. Cranial nerve examination revealed no particular deficits. However, it did show absent sensation in the left side of the face/neck and normal sensation on the right side. Reflexes were noted to be normal. It was noted by the registrar that Mr M had possibly had a stroke or a transient ischaemic attack. Alternatively, he had encephalitis. After discussion with consultant, Dr Renwar Reben, Mr M was admitted to the High Dependency Unit under Dr Reben's care.

That night it was noted by nursing staff that Mr M complained of coldness and tingling on his left side. Staff noted a pronounced lack of co-ordination and power on the left side. They also noted Mr M was unable to sit on the edge of the bed. In the morning Mr M was seen by Dr Reben during his ward round. Clinical examination at this time showed that cranial nerves two, three, four and six were intact. Sensation to the lower two thirds of the left side of the face was absent. There was also a facial droop on the left side. Muscle power in all four limbs was normal. Sensation testing showed absent joint positions sense and vibration sense on the left side. Light touch was diminished on the left side. The reflexes were intact in all limbs on both sides. There was persistent clonus in the left ankle. Coordination testing showed an abnormal finger to nose test on the left side and an abnormal heel – shin test, also on the left side. The diagnosis was encephalitis. There was a secondary diagnosis of cerebrovascular accident or stroke. A Doppler ultrasound study of the carotid arteries was ordered along with an MRI scan of the brain. (An MRI had in fact been ordered the previous day, albeit on an outpatient basis). The ultrasound was carried out in the afternoon. It showed ante grade vertebral flow; "No plaque, stenosis, dissection or focal abnormality" was identified in the carotid arteries.

8 September was a Saturday and no medical entries were made in Mr M's notes. However, it was recorded by nursing staff that his inco-ordination and altered sensation on the left persisted. He also complained of being fatigued. An EEG was carried out and was later reported as normal.

At a ward round on 9 September consultant physician, Dr Michael Hogg, found that Mr M continued to have impaired sensation and co-ordination on his left side. He was transferred from the HDU to the medical ward. The following morning he was again seen by Dr Reben when it was noted: "Able to stand but unable to lift his left leg to walk, still numb left hand side.....Plan: TOE and MRI in Launceston tomorrow; Impression: ?atypical bacterial

endocarditis.” Mr M was then transported to the LGH and later returned to the NWRH arriving at 6.00pm.

At 9.30pm on 10 September Mr M complained of feeling dizzy and nauseous. He told nursing staff that he felt similarly before the episode at home 4 days previously. It was noted that he was sweaty and pale. He vomited. At 10.00pm he was noted to be still vomiting. The medical registrar, Dr Natalia Oud, was called. She described bilateral horizontal nystagmus and dilated but reactive pupils. Otherwise she considered the signs to be unchanged and it was her impression that Mr M had nausea related to his medications. Symptomatic treatment was commenced.

In the early hours of the following morning a Code Blue was called when Mr M became unresponsive, was *“further fitting”* and bradycardic. It was noted that the MRI scan result was pending. Mr M was intubated, had arterial and central venous lines inserted and was transferred to the Intensive Care Unit. At 8.20am he was reviewed by locum ICU consultant, Dr Jonathan Albrett. It was noted: *“Chase MRI report, liaise with neurology team in RHH, lighten sedation, assess neurology.”* At 12.00 noon it was recorded that Mr M had been accepted for transfer to the care of stroke physician, Dr Nicole Hancock at the RHH.

The records indicate that the results of the MRI scan became known to NWRH staff by 1.40pm on 11 September. The impression is stated as: *“Acute infarct in the territory of posterior circulation. There is involvement of the temporal lobe, cerebellum and thalamus of the right side that could be a sequela of an embolic event/dissection of the right vertebral artery.....”* This finding led to the diagnosis of an acute posterior cerebral infarct with haemorrhagic transformation being made. The transfer to the RHH was then effected with Mr M arriving at around 4.00pm. He was admitted to the ICU.

At the RHH Mr M was reviewed by its ICU, neurology and neurosurgical teams. In their view Mr M’s extensive brain stem infarct with progression and mass effect as shown by scanning suggested a profoundly poor prognosis. Following discussion with family members, active treatment was withdrawn. Mr M continued to deteriorate and died at 11.00am on 12 September 2012.

Post-Mortem Report

This was carried out by forensic pathologist, Dr Donald Ritchey. He reports: *“.... The autopsy revealed a well-developed, well-nourished adult Caucasian man with a thrombus (blood clot) within the basilar artery extending from the confluence of the vertebral arteries to the bifurcation of the posterior cerebral arteries. There was marked large volume ischaemic infarction of the ventral and posterior right temporal, parietal and occipital brain lobes, the right cerebellar hemisphere and ill-defined infarction within the brain stem.”*

In Dr Ritchey’s opinion the cause of Mr M’s death was a large volume thrombotic stroke within the distribution of the right posterior cerebral artery and posterior inferior cerebellar artery. Significant contributing factors were atherosclerotic and hypertensive cardiovascular disease and emphysema.

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

Investigation

This has been informed by:

1. An affidavit provided by Mrs M.
2. Patient Care Report of Ambulance Tasmania.
3. A report of consultant radiologist Dr Anil Gupta with memo of the Manager/Chief Radiographer at the LGH. Dr Gupta is the radiologist who carried out Mr M's brain MRI at the LGH on 10 September 2012.
4. Report of Dr John Winter, Acting Clinical Leader of Medicine at NWRH.
5. Report of radiologist, Dr Kenneth McLean. Dr McLean reported upon Mr M's CT brain scan ordered on 6 September 2012.
6. Report of Dr Reben.
7. Report of Dr Albrett.
8. Report of Dr Kunnath Chandran, successor to Dr Winter as Clinical Leader of Medicine at NWRH. It includes a report upon an Adverse Event Investigation undertaken with respect to Mr M' hospital care.
9. A review of Mr M's records at the NWRH and the RHH carried out by research nurse, Ms L K Newman.
10. Reports provided by Dr A J Bell as medical adviser to the coroner.
11. A review of events surrounding Mr M's death carried out by Dr Spencer Toombes, Senior Staff Physician and Director of Physical Education at the Toowoomba General Hospital.

One focus of the investigation concerned events associated with the MRI carried out at the LGH on 10 September. It is helpful to set out the evidence specific to this subject. It follows:

- The NWRH did not have the capacity to carry out MRI scans and its patients had to be transported to the LGH for this procedure.
- The initial request for Mr M's MRI was received by fax at the LGH's Department of Medical Imaging (DMI) at 11.12am on 6 September 2012. The referral form which was completed by Dr Brady Tassicker was not marked 'urgent' and it wrongly showed Mr M to be an outpatient.
- DMI did not act upon the initial MRI referral until the following day when staff attempted to contact Mr M by phone to arrange an appointment. This alerted NWRH staff that an appointment was required. An amended referral form was then faxed to DMI and was received at 3.35pm. It showed that Mr M was a patient in ICU. Again it did not request an urgent MRI. DMI proposed to carry out the MRI on Monday 10 September and this was agreed by NWRH.
- Dr Reben contends that following his consultation with Mr M on 7 September that he directed that the MRI scan of the brain be carried out as a matter of urgency. In his statement he says: *"I requested an urgent MRI scan and TOE. But it's usually impossible to get it done on Fridays. The case was discussed with the cardiology*

registrar and radiology department in LGH. They didn't think it was urgent or that it needed to be done on Friday and that they would do it on Monday." It is not apparent from the evidence whether this communication in fact occurred. If it did then it did not lead to DMI attending to the MRI as a matter of urgency.

- The MRI was performed by Dr Gupta in the afternoon of 10 September and was completed by 3.58pm.
- Dr Gupta recognised that the MRI demonstrated serious pathology and required urgent release. His report was completed and at 4.24pm was entered on the Picture Archive Communication System (PACS) which made it immediately available to all clinicians within the Department of Health and Human Services including those at the NWRH.
- Dr Gupta asserts that he was *"assured that the result of the (MRI) study would become available to the concerned referring physician in the ICU at (NWRH) immediately."* It is not apparent whom gave Dr Gupta that assurance nor how the referring physician was to be informed apart from the MRI report being promptly entered on the PACS.
- Notwithstanding the serious pathology revealed by the MRI neither Dr Gupta nor any other person at the LGH made contact with medical staff at the NWRH to verbally report on the MRI with a view to deciding whether Mr M should be returned to the NWRH or alternatively should be immediately conveyed to the Stroke Unit at the RHH. Instead Mr M was returned to the NWRH.
- Medical staff caring for Mr M did not become aware of the results of the MRI until 1.40pm on 11 September, which is approximately 21 hours after they became accessible via PACS and more than 5 days after the referral for the MRI was made.
- Medical staff caring for Mr M were not aware that they could access the MRI results via PACS notwithstanding that this service had been available since May 2012.

In his capacity as medical adviser to the coroner, Dr Bell reviewed Mr M's hospital records along with all other material obtained during the investigation. In his subsequent reports he proffers these opinions:

- In the ED, Mr M presented with left-sided motor and sensory signs along with inco-ordination. This indicated a lesion in the brain stem (posterior fossa). The sudden onset was suggestive of a vascular event; i.e. a stroke, haemorrhage or thrombosis.
- A normal report upon the initial CT scan does not preclude a brain stem injury. However, it is suggestive that such injury is attributable to clotting and not to bleeding.
- A diagnosis of encephalitis was not consistent with Mr M's clinical presentation. When the lumbar puncture was reported as normal in the afternoon of 6 September the diagnosis of encephalitis should have been abandoned.
- It was a serious misjudgement on Dr Reben's part to maintain the diagnosis of encephalitis following his attendance upon Mr M on 7 September, particularly in light of the normal lumbar puncture result. Instead, a diagnosis of brain stem infarction/ischaemia should have been made at that time. In all likelihood it could have been proven by a CT scan with contrast and a CT angiogram.

- Mr M should have been transferred to the RHH on 7 September for management in its Stroke Unit.
- Once the MRI was ordered it should have been obtained as a matter of urgency. It was unacceptable to allow the procedure to be delayed for 4 days.
- Once the result of the MRI was known it was poor patient management to allow Mr M to be returned to the NWRH as it did not have the necessary neurological resources to manage his condition. At this point he required either admission to the LGH or transfer to the RHH.
- Optimal treatment required Mr M to be transferred to the RHH Stroke Unit on 7 September. Had this occurred, appropriate treatment, principally in the form of anti-coagulation and/or neurovascular intervention, had the prospect of halting Mr M's neurological deterioration and facilitating his functional recovery.
- By the time a decision was taken to transfer Mr M to the RHH his condition had deteriorated to the extent that his death was inevitable.

It is also beneficial for me to set out the principal opinions expressed by Dr Toombes following his review of this matter. He says that:

- Mr M's initial management in the ED by waiting to see whether his condition improved spontaneously was understandable and appropriate. Subsequently he did receive appropriate therapy for stroke. However, there were deficiencies in the process of his care and/or its documentation. He points to these matters:
 - a. There was no documented handover of specialist care for Mr M for the weekend and there was no evidence that he was seen by any medical officer between Friday and Sunday morning. For a patient in the HDU this "*seems extraordinary.*"
 - b. There was no communication between the LGH radiology department and the NWRH medical staff upon the significant abnormalities revealed by Mr M's MRI.
 - c. Medical staff at NWRH should not have waited until Monday to have the MRI done although this is "*an easy judgement to pass in hindsight.*"
 - d. The communication relating to the urgency of the MRI was sub-optimal and poorly documented.
 - e. Mr M required immediate neurological imaging when his focal neurological abnormality had lasted more than 24 hours. It is likely that this would have demonstrated the posterior circulation stroke and/or the basilar artery thrombus.
- Although criticisms can be made of the care provided by the NWRH it is most likely that Mr M would have died, even if he had received optimal care.

Findings, Comments and Recommendations

I find, having regard to the relevant evidence that Dr Reben, as the consultant responsible for Mr M's care, should have realised, at the latest, at the time of his ward round in the morning of 7 September that his patient had suffered a stroke and made this diagnosis. This of

course is a serious medical condition requiring an urgent response. I also find, accepting the opinion of Dr Bell, that at that time Mr M should have been transferred to the RHH's Stroke Unit as that facility was best equipped to treat his condition and maximise his prospects of recovery.

Regrettably, Mr M did not receive the immediate treatment that his condition mandated. Instead it was determined that further investigation was required, notably an MRI. I have set out in detail the evidence related to this procedure. It demonstrates an indifference to Mr M's proper care and elements of serious farce. Two features require highlighting. The first is the decision by staff at the LGH to permit Mr M to be returned to the NWRH without first discussing the MRI findings with his clinicians. Such discussion should hopefully have led to Mr M being immediately conveyed to the RHH. The second farcical element concerns the PACS. The evidence shows that although this system was in place by May 2012 the medical and nursing staff involved in Mr M's care were either unaware of its existence or did not know how to access it. In the result those treating Mr M did not become aware of the MRI findings until Dr Albrett became involved in his care and took steps to obtain the report. This was not until the early afternoon of 11 September after which the transfer to the RHH was effected. The evidence satisfies me that by this time the deterioration in Mr M's condition made his transfer futile.

It is not possible for me to find that Mr M's death would have been avoided if he had received optimal medical care. However, I am satisfied, and so find, that if Mr M had been promptly diagnosed and transferred to the Stroke Unit on 7 September 2012 that there was a real likelihood that he could have been successfully treated and made a functional recovery. Unfortunately he was denied that prospect.

It needs to be noted that following Mr M's death, Dr Albrett made a report to the NWRH's electronic incident monitoring system (EIMS). This led to an Adverse Event Investigation being undertaken for the Tasmanian Health Organisation - North West. Its resultant report acknowledges some shortcomings related to Mr M's management. It also sets out some recommendations and I am advised that these have been responded to. Details of the recommendations and the responses follow:

1. Nursing and medical staff be educated regarding access to radiology results online with posters and on-line links for using PACS to be sent to all clinical leaders and nursing unit managers at NWRH.

I am advised that this was attended to on 6 November 2012.

2. That ICU and radiology consultants discuss MRI requests and the results be conveyed via telephone and fax immediately following procedure.

I am advised that the Director of Medical Services at NWRH discussed this recommendation with the LGH radiologists but I have not been informed of the outcome.

3. That LGH radiology assess scans and discuss the results with the treating consultant immediately to determine whether a patient should be transferred to another hospital prior to departing the LGH radiology department.

I am advised that the Director of Medical Services at the LGH has discussed this recommendation with the radiologists at LGH but I have not been informed of the outcome.

4. That there be an improvement in collaboration between the Medical and ICU teams at NWRH to promote patient outcomes.

I am informed that in response to this recommendation the NWRH Medical and ICU teams now meet on a monthly basis to improve their collaboration.

5. That all HDU patients at NWRH should have a documented review each day as the minimum standard of care.

I am advised that this has been put in place.

I accept the above recommendations to be appropriate. It is to be hoped that they are all fully implemented and complied with.

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, and 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 overseen by me.

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

Dated: 23 January 2018 at Hobart in the State of Tasmania.

Rod Chandler
Coroner