OFFICE OF THE STATE CORONER

FINDING OF INQUEST

CITATION:	**Inquest into the death of Terence James Robinson**
TITLE OF COURT:	Coroner's Court
JURISDICTION:	Brisbane
FILE NO(s):	COR 2482/04(5)
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FINDINGS OF:	Mr Michael Barnes, State Coroner
CATCHWORDS:	CORONERS: Inquest, status epilepticus, monitoring of psychiatric patients, emergency sedation, transfer to primary hospitals.

REPRESENTATION:

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Findings of the inquest into the death of Terence James ROBINSON

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Pursuant to s28 (1) of the *Coroners Act 2003* an inquest was held into the death of Terence James Robinson. These are my findings. They will be distributed in accordance with requirements of s45(4) and s46(2) of the Act.

Introduction

On 6 October 2004 Mr Robinson was transported by ambulance from the Belmont Private Hospital (the BPH) where he had been an in-patient for sixteen days to the Princess Alexandra Hospital. On arrival at the Princess Alexandra Hospital (PAH) Mr Robinson was found to be unconscious and unresponsive. Investigations confirmed that he was suffering status epilepticus. He died two days later without regaining consciousness.

These findings seek to explain the circumstances of Mr Robinson's death and make recommendations aimed at reducing the likelihood of deaths occurring in similar circumstances in the future.

The Coroner's jurisdiction

Before turning to the evidence, I will say something about the nature of the coronial jurisdiction.

The basis of the jurisdiction

Because the doctors at the Princess Alexandra Hospital considered the death was *"not reasonably expected to be the outcome of a heath procedure"* within the terms of 8(3)(d) of the Act, they were obliged by s7(3) to report the death to the police or a coroner. Further, as Mr Robinson was the subject of an involuntary treatment order under the *Mental Health Act* 2000 his death was a "*death in care*" within section 9 of the Act. That also required that it be reported to a coroner. Section 11(2) confers jurisdiction on a coroner to investigate such a death and s28 authorises the holding of an inquest into it.

The scope of the Coroner's inquiry and findings

A coroner has jurisdiction to inquire into the cause and the circumstances of a reportable death.

The Act, in s45(2), provides that when investigating a death the coroner must, if possible find:-

- the identity of the deceased,
- how, when and where the death occurred, and
- what caused the death.

After considering all of the evidence presented at the inquest, findings must be given in relation to each of those matters to the extent that they are able to be proved.

An inquest is not a trial between opposing parties but an inquiry into the death. In a leading English case it was described in this way:-

> *It is an inquisitorial process, a process of investigation quite unlike a criminal trial where the prosecutor accuses and the accused defends… The function of an inquest is to seek out and record as many of the facts concerning the death as the public interest requires.* [1]

The focus is on discovering what happened, not on ascribing guilt, attributing blame or apportioning liability. The purpose is to inform the family and the public of how the death occurred with a view to reducing the likelihood of similar deaths. As a result, the Act, authorises a coroner to "*comment on anything connected with a death investigated at an inquest that relates to –*

(a) public health or safety ; or

(b) …

(c) ways to prevent deaths from happening in similar circumstances in the future.[2]

The Act prohibits findings or comments including any statement that a person is guilty of an offence or civilly liable for something.[3]

The admissibility of evidence and the standard of proof

Proceedings in a coroner's court are not as constrained as courts exercising criminal or civil jurisdiction because s37 of the Act provides that "(*T)he Coroners Court is not bound by the rules of evidence, but may inform itself in any way it considers appropriate."*

This flexibility has been explained as a consequence of an inquest being a fact-finding exercise rather than a means of apportioning guilt: an inquiry rather than a trial.[4]

A coroner should apply the civil standard of proof, namely the balance of probabilities, but the approach referred to as the *Briginshaw* sliding scale is applicable.[5] This means that the more significant the issue to be determined, the more serious an allegation or the more inherently unlikely an occurrence, the clearer and more persuasive the evidence needed for the trier of fact to be sufficiently satisfied that it has been proven to the civil standard.[6]

It is also clear that a coroner is obliged to comply with the rules of natural justice and to act judicially.[7]This means that no findings adverse to the interest of any party may be made without that party first being given a right to be heard in opposition to that finding. As *Annetts v McCann*[8] makes clear,

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[1] *R v South London Coroner; ex parte Thompson* (1982) 126 S.J. 625
[2] s46(1)
[3] s45(5) and s46(3)
[4] *R v South London Coroner; ex parte Thompson* per Lord Lane CJ, (1982) 126 S.J. 625
[5] *Anderson v Blashki* [1993] 2 VR 89 at 96 per Gobbo J
[6] Briginshaw v Briginshaw (1938) 60 CLR 336 at 361 per Sir Owen Dixon J
[7] *Harmsworth v State Coroner* [1989] VR 989 at 994 and see a useful discussion of the issue in Freckelton I., "Inquest Law" in *The inquest handbook*, Selby H., Federation Press, 1998 at 13
[8] (1990) 65 ALJR 167 at 168

that includes being given an opportunity to make submissions against findings that might be damaging to the reputation of any individual or organisation.

The investigation

I turn now to a description of the investigation into this death.

Police attended the hospital and arranged for Mr Robinson's body to be transferred to the John Tonge Centre where an autopsy was conducted.

A police officer also attended at Mr Robinson's home and took possession of all of his medication as identified by his wife.

Because the investigation was primarily to focus on the treatment provided to Mr Robinson at the Belmont Private Hospital it is not a matter which most police are adequately trained to investigate. Indeed after making minimal initial inquiries police concluded that the death was not suspicious and that no criminal charges were likely to arise. This led them to conclude that "*this matter will most appropriately be dealt with by the coroner".*

Accordingly the matter has effectively been investigated by the staff of this office, primarily by them requesting the medical practitioners involved in the treatment of Mr Robinson to supply detailed statements which were then provided with Mr Robinson's medical records to independent experts for critique.

I accept that an investigation of a death such as this poses special problems for most police officers who have very limited experience in dealing with such matters. I have commented in an earlier inquest into a medical adverse event, that coroners are hindered by not having resort to assistance from specialist investigators such as those who assist them with inquiries in many other esoteric settings. In the inquest into the death of Katherine Sabadina[9] I recommended that the Chief Health Officer collaborate with me to develop a process whereby appropriately qualified investigators could be nominated to perform this function. Regrettably, such is the continuing rate of change in Queensland Health that the recommendation, while not opposed, has not been advanced.

I am hopeful that discussions with the inaugural Health Quality and Complaints Commissioner may be more fruitful as the Act which creates that office specifically provides for the Commission to undertake such investigations.

The inquest

A pre-inquest conference was convened on 3 March 2006. Ms Jenny Rosengren was appointed counsel assisting. Leave to appear was granted to Mr Robinson's wife, the Belmont Private Hospital and Dr Mark Spelman.

[9] the findings in that matter can be found at http://www.courts.qld.gov.au/Sabadina-findings_final.pdf

When the matter came on for hearing the wife of Mr Robinson indicated that she no longer intended to be separately represented. Those assisting me conferred with her before and during the hearing.

The hearing commenced on 8 May 2006 and proceeded over the succeeding three days. Twelve witnesses gave evidence and 46 exhibits were tendered.

The evidence

I turn now to the evidence. Of course, I can not even summarise all of the information contained in the exhibits and transcript but I consider it appropriate to record in these reasons the evidence I believe is necessary to understand the findings I have made.

Mr Robinson's medical history

CVA in 1999

An explanation of the events that led to Mr Robinson's death must start with a description of an incident that occurred in 1999. Late at night, in October of that year, his wife found Mr Robinson suffering from some type of seizure. She took him to the Princess Alexandra Hospital emergency department. An urgent CT scan of his head revealed no haemorrhage or localized lesion. It was decided to continue investigations as an outpatient the following day and he was discharged later that morning.

The cause of that episode was never conclusively established. However an MRI scan in October 2004 demonstrated a lesion in the anterior limb of the internal capsule of the brain. It seems reasonable to assume this lesion was caused by the October 1999 event.

Following the cerebro vascular event referred to above, Mr Robinson developed an atypical psychiatric disorder, although his diagnosis remained uncertain. Professor Eadie, a neurologist, had told Mr Robinson that the October 1999 episode would cause him to have more difficulty in controlling his emotions and temper.[10] That proved correct in that he suffered from a mood disorder which had a number of atypical features, including very rapid drops in his mood frequently associated with serious suicidal behaviours. Mr Robinson also developed an impulse control disorder which resulted in him getting into repeated verbal and physical altercations with members of the public. These episodes could occur in the absence of alcohol but when alcohol was involved they tended to be more serious.

A causal connection between the October 1999 incident and the onset of Mr Robinson's psychiatric problems has not been clearly established by the evidence. Dr Robert Henderson, neurologist, said that the lesion seen in the

[10] T102

MRI is located in the area of the brain that controls pathways from more cortical structures which impact upon body movements and sensation, as opposed to psychological or personality related factors.[11] Dr Richard Boyle, neurologist, concurred with this view.[12] However, Drs Lawrence and Kubler considered a causal connection could not be excluded.[13]

The changes bought on as a result of his illness were far reaching. Prior to suffering the stroke in 1999 Mr Robinson owned his own business supplying garden products and he had no criminal history. Since the CVA he has had a recurrence of depressive illness characterised by marked irritability, anger and aggression and suicide attempts.

Mr Robinson's mental health carers sought to assist him in a number of ways including cognitive behavioural therapy, alcohol counselling and various medications. None of these approaches resolved Mr Robinson's problems and he continued to suffer from depression and violent outbursts which sometimes led to police intervention.

Uncertain but on going psychiatric illness

Whatever the original cause of Mr Robinson's condition, there is no doubt that by 2001 he was in need of fairly intensive psychotherapy and in 2001 he began receiving regular treatment at the Belmont Private Hospital.

Mr Robinson came under the care of Dr Mark Spelman, a psychiatrist, in October 2001. Throughout 2002, 2003 and 2004, Mr Robinson attended upon Dr Spelman on a semi regular basis; every six to eight weeks. He attended a cognitive behavioural therapy program at the hospital and was prescribed Paroxetine for his depression and Sodium Valproate to counter his mood swings.

Dr Spelman was still unable to provide a definitive diagnosis of Mr Robinson's condition. Nor was he able to assist Mr Robinson maintain sufficient stability for him to continue running the small business he had successfully operated since 1996. This is not meant as criticism of Dr Spelman but rather to highlight the frustrating situation Mr Robinson was in. Nor should it be concluded that he played no part in the maintenance of that state of uncertainty; Mr Robinson's refusal to abstain from alcohol even though he had ample evidence that it exacerbated his condition was an unfortunate complicating factor.

In early 2004, his alcohol fuelled outbursts had twice brought him into conflict with police and had resulted in minor criminal charges being preferred. Mr Robinson pleaded guilty and was fined and placed on probation**.**

[11] T120
[12] T215
[13] T185 & T231

Incident precipitating his final admission

In September 2004, Mr Robinson became involved in an ongoing dispute with some neighbours. This resulted in some shouting and abuse but no physical violence. It had been brewing for some days and his wife considers he had reason to feel badly treated by them. On 16 September he was at home with his two sons and apparently feeling resentful about the way the neighbours had been interacting with him. He was playing music quite loudly and drinking alcohol.

At about 11.30pm police attended at the house in response to a noise complaint. Mr Robinson did not react in a constructive way to their presence and violence between he and the police ensued. This resulted in Mr Robinson being arrested and taken from the premises. He was charged with resisting arrest, assaulting police and wilful damage.

Mr Robinson sustained some not insignificant head injuries and assorted abrasions during this struggle. The arresting officers took Mr Robinson to the Redlands Hospital where he received first aid treatment but was not admitted. He was then taken to the watch house, charged and released.

Mr Robinson continued to stew on what he believed to be the unfairness of his situation and such was his level of anxiety that his wife, who had been away on the night of the incident, persuaded him to seek admission to the Belmont Private Hospital in the hope that his anger and distress would subside with treatment.

Belmont Private Hospital admission on 20 September 2004

The BPH records show Mr Robinson was admitted to the hospital as a voluntary patient at approximately 1.30pm on 20 September 2004. This was in accordance with instructions that Dr Spelman had left as part of Mr Robinson's management plan, to the effect that he should be admitted to the hospital if he presented following a violent outburst.

Initial assessment

On admission, it was recorded that Mr Robinson was alert and co-operative. There were no physical abnormalities apart from a contusion of his left eye and some conjunctival haemorrhage. There were no neurological abnormalities. He was calm and oriented and there was no evidence of excessive anxiety or agitation. It was noted that his medications then consisted of 30mg of Paroxetine and 100mg of Aspirin every morning and 500mg of Sodium Valproate every night. Ten milligrams of Diazepam, a sedative and 5-10mg Zolpidem, a sleeping tablet, were added on an "as required" basis.

21 to 28 September 2004

Mr Robinson's first week in BPH was largely uneventful. He was cooperative and not displaying significant mood or anxiety symptoms. There was no expression of suicidal ideation or evidence of the presence of anger or vengeful retaliation plans. He interacted well with staff and family members

and displayed no difficulties communicating or attending to normal activities of daily living. Pathology investigations were undertaken which revealed no significant abnormalities.

On 22 September 2004, Dr Gills reviewed Mr Robinson and increased his Sodium Valproate to 500mg twice a day as he had been returning sub-therapeutic serum levels. Dr Spelman reviewed Mr Robinson on the following day. Physically there was evidence of significant facial swelling and bruising from the incident prior to his admission. Dr Spelman had no immediate concerns for Mr Robinson's physical state; he suspected he had a closed head injury and ordered a CT scan. It was noted Mr Robinson appeared to have amnesia for recent events.

Dr Spelman reviewed Mr Robinson again on 24 September 2004. Mr Robinson continued to complain of a headache and Dr Spelman organized for Dr Jonathan Farrah, general practitioner, with some 10 years experience, to review Mr Robinson on 27 September 2004.

Mr Robinson was allowed day leave on 26 September 2004. He left the hospital at 9.05am and spent the day with his wife and family. He returned at 4.30pm.

Dr Farrah reviewed Mr Robinson on 27 September 2004, at which time Mr Robinson continued to complain of a headache. Dr Farrah carried out a comprehensive physical examination and found no neurological abnormality. A CT scan of his head was ordered to exclude intracranial pathology. That evening, his wife and children picked him up from the hospital, took him out for dinner and then returned him to the hospital.

The CT scan ordered by Dr Farrah was performed by an external provider on the morning of 28 September 2004. On his return to the hospital, Dr Spelman reviewed Mr Robinson in conjunction with his wife at which time Mr Robinson's issues with impulse control were discussed. Dr Spelman did not have the results of the CT scan at the time of this review.

29 September 2004 – Mr Robinson is regulated

On 29 September Mr Robinson's wife picked him up from the hospital at 8.30am and returned him at approximately 1pm. He was reviewed by Dr Spelman in the afternoon, at which time Dr Spelman was aware the CT scan had detected no neurological abnormalities. Dr Spelman decided he would also order an MRI and an EEG in order to further investigate whether any organic explanation for Mr Robinson's violent behaviour could be identified. He said in evidence this was in part motivated by advice from Mr Robinson's solicitors that it was likely that Mr Robinson would be incarcerated as a result of the charges arising from the incident on 16 September. Mr Robinson was also told of his solicitors' pessimistic prognosis.

Dr Spelman decided to commence Mr Robinson on Naltrexone the following day with a dosage of 50mg to aid in alcohol abstinence which had proven problematic for him on many occasions. Mr Robinson had been prescribed

Naltrexone in January 2004 and had not experienced any adverse reactions to this medication.[14]

At 9.30pm, Mr Robinson was observed by a nurse to be settled and reactive and chatting with staff. There were no concerns. However, between 9.30pm and 11.00pm there was a significant change in his mental state. At 11.00pm he was found, during routine checks, to have barricaded himself in his room by pushing the bed against it. He had cut his wrists with a nail file. He was lying on the bed, ignoring staff requests. When staff gained entry, he was tearful and unresponsive. He stated he wanted to end it all. A suicide note, written to his family and others, was found in his room.

Mr Robinson was transferred to the Special Care Unit ("SCU"). Dr Spelman was telephoned and informed of Mr Robinson's distressed state and he ordered that Mr Robinson be given 10mg of the sedative Diazepam, 20 mg of the sleeping tablet Zolpidem and 100mg of Quietapine an atypical antipsychotic medication. Dr Spelman ordered the Quietapine because although Mr Robinson had no previous history of psychotic episodes, the drug is commonly used to control episodes of acutely disturbed behaviour, including anxiety and agitation which Mr Robinson had been reported as displaying.[15]

At approximately 11.55pm, Dr Spelman received another telephone call from a nurse to inform him that despite the administration of these medications, Mr Robinson had become increasingly anxious and agitated and was expressing a desire to break out of the hospital and to harm himself. In response, Dr Spelman ordered two 10mg wafers of Olanzapine, which is another atypical antipsychotic medication. Dr Spelman says he decided to order this different medication in a relatively high dose because it was apparent that the Quietapine had not had the desired effect and it was considered necessary to control Mr Robinson's behaviours without delay, as Dr Spelman was concerned that Mr Robinson was a danger to himself and the staff.[16]

The literature indicates that Quietapine and Olanzapine should be used with caution when a patient has a history of seizures. Although Mr Robinson had not previously taken either of these anti-psychotic drugs, it was Dr Spelman's understanding that he had never had a seizure and therefore these medications were not contra-indicated in treating him.[17] Dr Spelman considered that given the types and dosages of the drugs administered, it was likely Mr Robinson would remain sedated for a period of 24 to 48 hours.

30 September to 5 October 2004

Dr Spelman attended at the BPH at 12.10 am on 30 September 2004. The medication had taken effect and Mr Robinson was sleeping soundly. Dr Spelman regulated Mr Robinson under the Mental Health Act, that is he formed the opinion that Mr Robinson was suffering from a mental illness as

[14] T108
[15] T78
[16] T82
[17] T75

defined by the Act and that he was a danger to himself if not made the subject of an involuntary treatment order. Dr Spelman informed Mrs Robinson of this decision and the reasons for it. Mr Robinson remained sedated for the rest of the day and Dr Spelman reviewed him twice more, at 7.45am and 5.10pm. During the day he was given the Naltrexone which had been previously ordered.

Dr Steve Prowacki was the psychiatrist on call to cover for Dr Spelman on 1, 2 and 3 October 2004. At review on 1 October 2004, Mr Robinson presented as sedated and was able to give an account of his recent actions. He described continuing self-harming urges.

On 2 October 2004, Dr Prowacki found it difficult to further assess Mr Robinson's underlying mood as he presented as quite withdrawn. However, he said in evidence that he was not overly concerned with Mr Robinson's presentation nor his level of sedation.[18]

Mr Robinson was not reviewed by any medical specialist on 3 October 2004 because no concerns were raised by nursing staff about his presentation and it was not standard practice at the BPH for patients in the SCU to be reviewed on a daily basis. The hospital has a standard of care to the effect that it is desirable for patients in the unit to be reviewed six times a week.[19] Therefore, Dr Prowacki did not review Mr Robinson on this day. However, the nursing notes show that his condition had continued to deteriorate during that day.[20]

Dr Farrah reviewed Mr Robinson on 4 October due to nursing staff concerns about Mr Robinson's deterioration over the previous 48 hours. On examination Mr Robinson was disorientated in time and place and had a right sided up-going plantar and possible right sided weakness. Dr Farrah considered the most likely diagnosis was an ischaemic left sided cerebral event but was not able to exclude a psychiatric cause of these symptoms.[21] Dr Farrah ordered an MRI and considered there was a need to undertake an ECG to ensure that there was not a problem with Mr Robinson's heart which was causing an interruption of the blood supply to his brain resulting in a cerebral event.

Dr Spelman reviewed Mr Robinson later that day and noted that he was only slowly regaining alertness from the sedation regime implemented on the evening of 29 September 2004. Mr Robinson recognised Dr Spelman, but he was amnesic for events in the past week. It was difficult to gain his co-operation to properly assess his neurological state. Dr Spelman noted from the nursing notes that Mr Robinson's mood had been mostly depressed and he made the decision to withhold all medication (including the anti-convulsant, Sodium Valproate), until after the MRI and blood test results were available.

[18] T138
[19] T140
[20] T35
[21] T151

Dr Spelman's differential diagnosis included a neurological event, a prolonged reaction to the medication given to him following the attempted suicide or catatonia. He considered it was more likely there was a psychiatric rather than an organic explanation for Mr Robinson's presentation.[22] Dr Spelman acknowledged that Mr Robinson's condition had deteriorated and there was a great deal of uncertainty as to the cause of the deterioration. However, he believed it was in Mr Robinson's best interests to remain in the BPH rather than being transferred to a medical facility because if there was any improvement or change in his mental state, there was a real risk of him being a danger to himself and others.[23] Dr Spelman arranged for Dr Alston Unwin, the Director of Medical Services at BPH, to provide a second opinion.

Dr Unwin assessed Mr Robinson on 5 October. He thought that while it was possible that Mr Robinson was catatonic, he favoured an organic cause on account of the upgoing right plantar and his fluctuating levels of consciousness. Dr Unwin considered a catatonic stupor, which is part of the schizophrenic process, was an unlikely diagnosis, because although some of his behaviour, in particular his aggression and deliberate self harm were hard to explain, [24]Mr Robinson had showed no evidence of this process in the past. Dr Unwin expressed this view to Dr Spelman.[25]

Later that day, Dr Unwin was informed of the results of the MRI – it was clear, apart from the injury I have assumed was a result of the 1999 CVA. Once this information was at hand, it became clear to Dr Unwin that there was no organic abnormality to explain Mr Robinson's condition. He was satisfied the upgoing right plantar could be explained by the existence of the old lesion in the anterior limb of the left internal capsule of the brain. In these circumstances Dr Unwin considered it was more likely there was a psychiatric reason for his presentation, such as a depressive stupor.[26]

Dr Spelman reviewed Mr Robinson on three occasions on 5 October 2004 and observed that his condition had deteriorated further from when he had assessed him on the previous day.[27] Mr Robinson had been incontinent of urine overnight and his oral intake had been poor over the previous 24 hours. He was more withdrawn and not responding to verbal stimuli. Although the MRI results had reduced Dr Spelman's concern about an organic cause, he requested Dr Farrah to review Mr Robinson again. Dr Farrah performed a lumbar puncture in order to exclude encephalitis. He considered such a diagnosis was unlikely but that "*we had to actually dot the Is and cross the Ts before proceeding to ECT*".[28] The plan was to give Mr Robinson electroconvulsive therapy (ECT) the following day.

[22] T87
[23] T85 to 89
[24] T34
[25] T39
[26] T56 and T 58
[27] T92
[28] T158

Emergency transfer to the PAH on 6 October

There is a nursing entry record at 5.42am on 6 October saying that Mr Robinson had slept throughout the night, he was assisted with repositioning and there was no response to nursing intervention. At approximately 6am, Dr Spelman was telephoned at home by a nurse and told that an ambulance had been called to transfer Mr Robinson to a medical facility as his condition had seriously deteriorated. Dr Spelman arrived at the hospital prior to the ambulance. He noted that Mr Robinson appeared to have had some sort of neurological episode, possibly an epileptic seizure or a CVA. He was having significant difficulties breathing, his oxygen saturations were reduced and he was peripherally shut down. He was unresponsive and unconscious. At approximately 6.50am, Dr Spelman telephoned Mrs Robinson and informed her of the seriousness of her husband's condition and that he was being transferred to the PAH.

Dr Spelman wrote a letter to the PAH summarizing the treatment provided to Mr Robinson at the BPH. This letter seems to have been misplaced at the PAH. The PAH records include copies of the radiological and pathology results requested whilst Mr Robinson was an inpatient at the BPH and the Mental Health Act forms. Dr Farrah travelled in the ambulance with Mr Robinson and recalls taking Dr Spelman's letter with him.

On arrival at the PAH at 7.06 am, Mr Robinson was taken into the resuscitation bay in the A & E Department. Dr Farrah handed Mr Robinson over to a registrar at the PAH and during a 20 minute conversation outlined his clinical history including the recent seizure activity.[29] Mr Robinson was noted to be having continuous grand mal seizures. A CT scan of his brain was undertaken which demonstrated no focal abnormality. Status epilepticus was provisionally diagnosed and was confirmed by an EEG later in the day.

Mr Robinson was admitted to the Intensive Care Unit at the PAH at 9.10 am. He was paralysed and sedated. A lumbar puncture was performed and he was treated with broad spectrum antibiotics until the results were available. Dr Peter Kruger, intensive care specialist saw Mr Robinson on his arrival in ICU. He was given Dr Spelman's letter and was aware Mr Robinson had experienced trouble with his behaviours and a variety of medications, although at that time he did not know precisely what these medications were.[30] Dr Kruger requested his registrar to ring the BPH for a more detailed history regarding the events in the previous few days.

Mr Robinson developed diabetes insipidus during the night of 6 October as a result of damage to his brain. By 7 October it had become evident to Dr Kruger that Mr Robinson's brain was potentially dead on account of the seizure activity. A repeat EEG performed in the afternoon showed a flat trace and a repeat CT scan revealed a grossly swollen brain.

[29] T167
[30] T255

Over the evening of 7 October and early hours of the next day, Mr Robinson became hypotensive requiring support with IV fluids, dopamine and noradrenaline. A four vessel angiogram was performed on the morning of 8 October 2004 which confirmed the clinical impression that Mr Robinson was brain dead. After discussion with his family, ventilatory support was withdrawn and his cardiac output ceased at 4.10 pm.

Autopsy findings

On 9 October 2004, an autopsy was undertaken by an experienced forensic pathologist, Dr Lampe, at the John Tonge Centre. The proximate cause of death was identified as being global cerebral hypoxic injury due to or as a consequence of status epilepticus.[31]

Global cerebral hypoxic injury is essentially a lack of oxygen to the brain which can occur as a result of prolonged seizures. With a seizure the pressure in the brain increases with the associated potential risk that smaller vessels and arteries in the brain are unable to receive sufficient signals. These cells react to a lack of oxygen by releasing water with the consequence that the brain swells.

Status epilepticus describes prolonged or clustered seizures which sometimes develop into non stop seizures. In a normally functioning brain, the nerve cells (neurons), communicate with one another by firing tiny electrical signals that pass from one cell to another. A seizure occurs when the firing pattern on the brain's electrical activity suddenly becomes intense and abnormal.

Expert evidence on the cause of the fatal condition

Whilst the cause of Mr Robinson's death seems relatively clear, the cause of the onset of his status epilepticus is not so. I was greatly assisted in considering this central issue by the experts who provided reports and gave evidence at the inquest. I shall summarise the most relevant aspects of their views.

Common triggers of seizures include:

- withdrawal from alcohol;
- a severe chemical imbalance in the blood;
- a local problem involving the brain such as head trauma, stroke, brain tumours and infections including encephalitis and meningitis;
- drug reactions to prescription medications.

Dr Kubler, a pharmacologist, opined that the potential causes of the status epilepticus in Mr Robinson's case can be narrowed down to the last two of these possibilities, namely:

- the old ischemic injury demonstrated on the MRI undertaken on 4 October 2004 which predisposed Mr Robinson to seizures;

[31] Ex 2.1

- the administration of the Quietapine and Olanzepine on the evening of 29 September 2004.[32]

However, Dr Kubler also referred to a further complicating factor concerning the other drugs given to Mr Robinson. It seems that the Sodium Valproate is capable of masking some seizure activity and that it has been reported, although rarely, that if Paroxetine is abruptly ceased that can contribute to the emergence of the condition. In these circumstances, the withdrawal of these medications at the direction of Dr Spelman on 4 October 2004 may have been one of many factors that precipitated the epilepsy.[33]

Dr Boyle, a neurologist, thought a drug reaction to Mr Robinson's prescribed medications was the most likely cause for Mr Robinson's altered state of consciousness and epilepsy.[34]

This raises the question of whether epilepsy should have been diagnosed prior to 6 October 2004. Dr Henderson, a neurologist, thought there was nothing about Mr Robinson's presentation prior to this time which was clinically consistent with the diagnosis of status epilepticus.[35] He and Dr Kubler[36] opined that it is possible Mr Robinson had non-convulsive status epilepticus whilst he was an inpatient in the BPH. This is an extremely rare condition where a patient is having seizures but not outwardly fitting.[37] Dr Boyle considered it was unlikely that Mr Robinson had non-convulsive status epilepticus prior to the night of 5 October 2004, because patients with this condition are usually more rousable than Mr Robinson was.[38] Dr Farrah had previously encountered patients suffering from non-convulsive status epilepticus and he did not believe that Mr Robinson was suffering from this condition when he examined him on the evening of 5 October 2004.[39]

Dr Henderson considered that Mr Robinson's condition became essentially irreversible some time on the evening of the 5th or during the day on the 6th.[40] Dr Henderson thought it was possible that Mr Robinson was suffering from some psychiatric stupor which spontaneously developed into active epilepsy late on 5 October or subsequently but more likely that Mr Robinson had an ongoing neurological process preceding his transfer to the PAH.[41]

Dr Boyle thought the epilepsy commenced some time during the night of 5 October and the early hours of 6 October 2004, when Mr Robinson was not under constant supervision.[42] Dr Kruger explained that uncontrolled fitting for

[32] T227 & T229
[33] T232
[34] T219
[35] T116
[36] T241 - 242
[37] T128
[38] T217
[39] T166
[40] T123
[41] T129
[42] T217

a period of 30 minutes can result in severe brain damage and whilst he could not say with any degree of certainty the period of time over which Mr Robinson had been fitting, he doubted that it would have been for many hours.[43]

Dr Kubler considered that by the time Mr Robinson's clinical state became overt, the seizure disorder was very entrenched as evidenced by the unsuccessful treatment which he was given at the PAH. For these reasons Dr Kubler considered that an earlier transfer was unlikely to have changed the outcome.[44]

Findings required by s45(2)

I am required to find, as far as is possible, the medical cause of death, who the deceased person was and when, where and how he came by his death. I have already dealt with this last aspect of the matter, the manner and circumstances of the death. As a result of considering all of the material contained in the exhibits and the evidence given by the witnesses, I am able to make the following findings in relation to the other aspects.

Identity of the deceased – The deceased was Terence James Robinson

Place of death – Mr Robinson died at the Princess Alexandra Hospital

Date of death – He died on 8 October 2004

Cause of death – Mr Robinson died from global cerebral hypoxic injury due to or as a consequence of status epilepticus.

Comments and preventive recommendations

Section 46, in so far as it is relevant to this matter, provides that a coroner may comment on anything connected with a death that relates to public health or safety or ways to prevent deaths from happening in similar circumstances in the future.

A number of issues raised by the evidence in this matter warrant consideration from that perspective. They are:-

- the appropriateness of Mr Robinson's drug therapy;
- the need for an emergency sedation policy at BPH;
- the daily review of patients in the SCU; and
- the timeliness of Mr Robinson's transfer to a primary hospital.

Appropriateness of the drug therapy

The rapid decline and death of Mr Robinson and the possibility that the drugs administered to him contributed to his fatal seizures naturally prompts consideration of whether his medication was appropriate, and, in particular,

[43] T256
[44] T236

whether one rather than two benzodiazepines should have been administered and whether it was appropriate to administer anti-psychotic drugs to a patient who was not exhibiting signs of psychosis.

Dr Spelman sought to justify the drug therapy he used on the basis that the different drugs used variously relieved the patient's anxiety, helped him sleep and ameliorated his agitation.

The two benzodiazepines prescribed to Mr Robinson by Dr Spelman were Zolpidem and Diazepam. Mr Robinson had taken this combination of medications in the past without difficulty. There is no policy at BPH mandating that mono-therapy with respect to benzodiazepines.[45]

Dr Kubler, a pharmacologist, considered that whilst it is open to administer two benzodiazepines, optimal practice is only to prescribe one such drug so as to make the medications easier to interpret in the event of an adverse drug reaction.[46] Dr Goodwin agreed with this.

However, none of the experts who gave evidence considered that these drugs contributed to the subsequent decline of Mr Robinson.

Dr Lawrence saw no difficultly with Mr Robinson being given Quetiapine and Olanzapine even though he had no history of psychosis and had already been given the benzodiazepines. She explained that whilst Quetiapine and Olanzapine are anti psychotic medications, they have traditionally and widely been used for the management of acutely disturbed, aggressive or abusive behaviours and are commonly administered in conjunction with benzodiazepines.

Dr Kubler was not critical of the decision to prescribe anti-psychotic drugs given that Mr Robinson was severely distressed and agitated and presenting as a significant risk to himself and the staff caring for him.[47]

Dr Goodwin said that while he would not have ordered anti-psychotic drugs for Mr Robinson, he accepted that such a decision was not outside the realm of appropriate practice and the decision is one for the treating clinician.[48]

Dr Lawrence also considered it was appropriate for Quetiapine to have been ordered by Dr Spelman when he was initially telephoned by a nurse late in the evening on 29 September 2004. This is because Quetiapine has particular value in not causing or aggravating depression and is the preferred anti-psychotic drug in treating a patient with mood disturbances.[49] With respect to Dr Spelman's decision to subsequently order Olanzapine in wafer form, Dr Lawrence considered this was appropriate largely because Quetiapine does

[45] T107
[46] T237
[47] T232, L18
[48] T263
[49] T170

not come in wafer form and a wafer is absorbed more quickly than a tablet.[50] Drs Kubler and Goodwin disagreed with Dr Lawrence's evidence to the effect that wafers are absorbed more quickly than tablets. Dr Kubler thought that whilst Mr Robinson required further medication subsequent to the administration of the Quietapine shortly after 11 pm on 29 September 2004, the dose of this medication could have simply been titrated upwards.[51] Dr Kubler did not think that Dr Spelman's decision to administer both anti-psychotics contributed to the onset of the seizures.[52] Dr Goodwin agreed with this conclusion.[53]

It was suggested during the course of the hearing that the 20mg of Olanzapine ordered by Dr Spelman at 11.55pm on 29 September 2004 was a relatively large dose. Dr Lawrence considered that such a dose was not excessive.[54] Dr Kubler explained that such a dose is towards the upper end of the recommended range. Dr Kubler said he would have given a starting dose of 5 to 10 mg. However, he stated this was a matter of clinical judgement.

As a result of considering the evidence of all of the experts who gave evidence and/or supplied reports, I have come to the conclusion that it can not be shown that the decisions of Dr Spelman to order the drugs he did were wrong. It was clear that there was some divergence of opinion among the various specialists as to what they consider to be the best response to Mr Robinson's competing needs. However, as I understand the evidence, none of them considers that the decisions taken by Dr Spelman were improper and all accept that clinicians must be free to make their individual assessments of their patients' needs. Most importantly, none of them asserted that the drug therapy could be shown to have been responsible for Mr Robinson's death.

An emergency sedation policy

Dr Goodwin suggested that the BPH should consider implementing an emergency sedation policy with an emphasis on mono-therapy and benzodiazepines as the initial agents of choice. The benefit of a focus on such medications is that they can be titrated and reversed as necessary. Dr Goodwin explained that whilst any policy around emergency sedation needed to be flexible, its purpose is to guide clinicians through a series of logical steps.[55]

Dr Lawrence considered there is a fairly broad range of acceptable practice in this regard and that whilst such a policy is important in teaching hospitals it is of less value in a hospital such as the BPH where emergency sedation is a matter of clinical judgement to be exercised by relatively senior clinicians.[56] The potential danger of such a policy in a private hospital setting is that it may

[50] T171
[51] T238
[52] T238, T247
[53] T290
[54] T172
[55] T255
[56] T176, 208 -

inhibit a treating specialist from treating a patient appropriately and provide a disincentive for visiting medical officers to apply for accreditation with the BPH.[57]

Dr Kubler explained that there has been an ongoing debate as to whether a relatively high risk medical intervention should be standardized or left to individual clinical preferences. Dr Kubler preferred the standardized approach even in a private hospital setting because there are elements beyond a treating clinicians control which can impact upon an adverse event occurring.[58] He thought any such policy could include guidelines as to the observations and feedback to be provided to the treating clinician.[59]

Dr Goodwin thought that even if such a policy had been in place at the BPH when Mr Robinson was being cared for there, that it is probable the existence of such a policy would have made no difference to the outcome because the policy would have to have been sufficiently flexible to enable Dr Spelman to have made a judgement call as to the types and doses of drugs to be administered.[60]

The Royal Brisbane and Royal Women's Hospital has a policy on rapid tranquilization that was implemented in September 2005. It provides that "*benzodiazepines should be combined with an antipsychotic agent to maximise the effect"* something that Dr Goodwin was vehemently opposed to. This is not to suggest Dr Goodwin is wrong but highlights the divergence of opinion about the issue among experts.

I certainly do not have sufficient expertise or information to make a prescriptive recommendation in this regard but I do consider a policy should be developed.

Recommendation 1 – the development of a rapid tranquilisation policy at BPH

I recommend that if they have not already done so, the BPH give consideration to developing a rapid tranquilisation policy.

I am aware that Queensland Health is in the process of developing a state wide policy on the topic and I would assume that BPH could be assisted by that process.

Daily review of patients in SCU

Mr Robinson was transferred to the Special Care Unit after a suicide attempt on the night of 29 September. He was then given emergency rapid tranquilisation that led to him having a severely compromised level of consciousness from which he never fully recovered.

[57] T208 - 209
[58] 240
[59] T248
[60] T266

Mr Robinson was reviewed daily for the two days after the sedation but then was not seen by a psychiatrist on the third day. He was then regularly reviewed until he was transferred on 6 October. This was consistent with the BPH policy of only reviewing patients in the SCU on six days a week. This policy was criticised by a number of the experts who gave evidence. I consider that criticism valid. As Mr Robinson's case so sadly and graphically demonstrates, patients in his condition can deteriorate very rapidly and there is a risk that nursing staff may not be sufficiently aware of some danger signs.

Recommendation 2 – Daily review of patients in SCU

I recommend that the BPH review its policy of not having a psychiatrist review patients in the SCU daily.

Should Mr Robinson have been transferred sooner?

Another issue on which considerable expert evidence was received concerned the timing of the transfer of Mr Robinson from the BPH to the PAH.

Drs Spelman and Farrah considered that prior to the morning of 6 October 2004, it was more appropriate for Mr Robinson to be cared for as a regulated patient in the BPH, as opposed to in a medical or psychiatric ward at a hospital such as the PAH. Their view was based on the fact that after the results of the CT scan and MRI investigation were known on 5 October 2004, there was reason to believe that Mr Robinson's presentation may have had a psychiatric basis. Their plan was for a lumbar puncture to be performed and following the outcome of that investigation a decision would be made as to whether to transfer Mr Robinson to a medical hospital or proceed with ECT.

Dr Spelman suggested it was particularly important to maintain continuity of care for Mr Robinson when it seemed his problems were psychiatric rather than organic.

Dr Spelman considered that a public hospital medical ward could not provide the same level of safety and protection as a closed psychiatric ward. Had there been a sudden change in Mr Robinson's mental state in a medical ward, he could have simply wandered off and "*anything could have happened*".[61]

Dr Goodwin, on the other hand, thought this was not a serious concern by 5 October 2004 at which time Mr Robinson was unconscious and not eating or drinking. It was Dr Goodwin's view that by this time Mr Robinson's physical and medical needs should have assumed paramount importance.[62] Dr Goodwin thought that Mr Robinson's condition had deteriorated so significantly by 5 October 2004 that this was not a valid reason and that he *"hoped there would be people that could look after Mr Robinson in the public hospital with whom Dr Spelman could liaise."*[63]

[61] T100
[62] T277
[63] T277

Dr Lawrence supported the decision not to transfer Mr Robinson to a medical facility on 4 October 2004. At that time Mr Robinson's condition was not urgent. It would have required transferring him to another hospital for reasons which were not clear, to be under the care of a doctor who would have had no real appreciation of Mr Robinson's psychiatric status and in circumstances where Mr Robinson had a recent history of sudden changes in his behaviours. Dr Lawrence doubted that any public medical or psychiatric ward would have accepted Mr Robinson as a patient at that time.[64] Further, it was reasonable for Dr Spelman to have thought that the most likely explanation for Mr Robinson's presentation was a psychiatric cause, even though her preferred provisional diagnosis was organically based.[65]

However, Dr Lawrence gave evidence that if Mr Robinson had been her patient she would have attempted to transfer him out of the BPH by the afternoon of 5 October 2004 or sought a second opinion from a neurologist. This is because the nursing notes for 2.15pm on that day record that Mr Robinson had been lying unconscious on his bed all morning and had been unresponsive. He had been unable to take any fluids and his wife had only been able to give him two to three spoonfuls of fluids by holding ice up to his lips. Dr Lawrence thought that notwithstanding the MRI results, an organic condition was still the most likely explanation for Mr Robinson's condition and that even if it was a depressive stupor, Mr Robinson's condition was such that he could not be treated with anti-depressant medications and there were no other psychiatric interventions which could have been implemented to change the situation.[66]

Dr Lawrence considered that Mr Robinson's condition had deteriorated to such an extent by this time that a public hospital would have been more inclined to admit him. Having said this, Dr Lawrence stated that she *"couldn't quibble with any of the clinical decision-making that appeared to have been done. It all seems very reasonable in the circumstances as I understood them to be and I still understand them to be. … I would have sought some other assistance a day or so earlier, but … I can equally understand in the circumstances the clinicians … coming to a slightly different conclusion."*[67]

I accept the view that it would have been preferable to at least attempt to transfer Mr Robinson to a medical ward on 5 October. Having regard to his very withdrawn state and fluctuating levels of consciousness, I am not persuaded that the risk of his suddenly become dangerously active should have been given more weight than his urgent need for specialist medical investigation. Dr Spelman was not assisted in making this determination by the hospital policy in force at the time which provided no guidance as to when transfers should be considered.

In the weeks prior to this inquest commencing, the BPH implemented a policy titled "*Transferring a Patient to an External Medical Facility".* The policy has

[64] T180 - 180
[65] T187
[66] T190 - 191
[67] T199 - 200

the effect of enabling the treating doctor in collaboration with hospital staff to exercise a relatively wide discretion in determining whether a patient should be transferred to a medical facility. It is designed to ensure that "*patients who require medical assessment or treatment not available at BPH are transferred to an appropriate facility as soon as practical*." Neither Dr Lawrence nor Dr Goodwin considered that any changes needed to be made to this policy.[68] In those circumstances no further comment from me is required.

Conclusions

This was a very sad case involving a relatively young man with relatively minor health problems dying suddenly, leaving a grieving wife and two young children. This inquest has been somewhat unsatisfactory in that I have not been able to clearly establish the factors that caused the fatal events, nor determine whether earlier or different medical intervention could have prevented them. It is stating the obvious to note that there are still aspects of neuropharmacology that remain mysterious to clinicians and researchers. While I hope that the clinicians involved in this sad death have learnt something from it and that the procedural recommendations I have made may, if implemented, reduce the chances of future similar deaths, I recognise that nothing will provide solace to Terry's family and friends for his sudden death. I offer them my sincere condolences.

This inquest is now closed.

Michael Barnes
State Coroner
Brisbane
8 September 2006

[68] T198 – 199, 279