

SOUTH



AUSTRALIA

## FINDING OF INQUEST

*An Inquest taken on behalf of our Sovereign Lady the Queen at Adelaide in the State of South Australia, on the 19<sup>th</sup>, 20<sup>th</sup> and 30<sup>th</sup> days of July 2004, before Wayne Cromwell Chivell, a Coroner for the said State, concerning the death of Colin Eric Betts.*

*I, the said Coroner, find that Colin Eric Betts aged 59 years, late of the Glenside Campus of the Royal Adelaide Hospital, Eastwood, South Australia died at Eastwood, South Australia on the 30<sup>th</sup> day of July 2001 as a result of pulmonary thromboembolism due to left deep calf vein thrombosis.*

### **1. Reason for inquest**

- 1.1. On 9 March 2001 Dr D Short, Medical Practitioner, made an order pursuant to Section 12(1) of the Mental Health Act 1993 ('the Act') immediately detaining Colin Eric Betts to the Glenside Campus of the Royal Adelaide Hospital, an approved treatment centre as defined in the Act. On 10 March 2001, Dr A S Czechowicz, Psychiatrist, confirmed Dr Short's order pursuant to Section 12(4) of the Act. On 12 March 2001 Dr B Kelly, Psychiatrist, made a further order for detention pursuant to Section 12(5) of the Act. An order pursuant to Section 12(5) has effect for 21 days.
- 1.2. Following an examination by Drs Page and Heint, Psychiatrists, on 30 March 2001, a second order for detention for a further 21 days was made pursuant to Section 12(6) of the Act.
- 1.3. On 11 April 2001 the Guardianship Board of South Australia made an order for continuing detention pursuant to Section 13(1) of the Act, detaining Mr Betts for a period of six months up to and including 11 October 2001.

- 1.4. Accordingly, at the time of his death on 30 July 2001, Mr Betts was ‘detained in custody pursuant to an Act or law of the State’ within the meaning of Section 12(1)(da) of the Coroners Act 1975, and an Inquest into his death was therefore mandatory by virtue of Section 14(1a) of the said Act.

## **2. Introduction**

- 2.1. On 30 July 2001, Mr Betts was a patient at Glenside Hospital in Jacaranda Ward.
- 2.2. At about 2pm that day, Mr Betts was seen wandering around the ward in a confused state. Clinical Nurse Peter Brzycki took him to the toilet, about 7 metres away.
- 2.3. In the toilet, Mr Betts began to shake and then he collapsed. Clinical Nurse Brzycki lowered him to the floor and sought assistance.
- 2.4. A medical emergency was called, oxygen was administered and an ambulance was called.
- 2.5. SA Ambulance Service paramedics arrived at 2:17pm and administered saline and maintained oxygen supply.
- 2.6. While he was being prepared for transportation to the Royal Adelaide Hospital, Mr Betts suffered a cardiac arrest. The paramedics commenced cardio-pulmonary resuscitation with the assistance of Glenside staff. Mr Betts was intubated and adrenalin was administered, but unfortunately he could not be revived and his life was pronounced extinct at 2:52pm by paramedics in the presence of Dr Short (see Exhibit C2a, p2).

## **3. Cause of death**

- 3.1. A post-mortem examination of the body of the deceased was performed by Dr J D Gilbert, Forensic Pathologist, on 1 August 2001. Dr Gilbert concluded that the cause of Mr Betts’ death was ‘pulmonary thromboembolism due to left deep calf vein thrombosis’ (Exhibit C3a, p1).
- 3.2. Dr Gilbert commented:
  - '1. Death was due to pulmonary thromboembolism due to left deep calf vein thrombosis. There was also evidence of paradoxical thromboembolism involving one of the kidneys attributable to passage of thromboembolic material across the patent foramen ovale. The thromboembolic event that caused death had been

preceded by earlier episodes as indicated by the finding of older organised thromboemboli in the lungs and established infarction in the embolised kidney.

Sedation resulting from medication with antipsychotic agents (olanzapine and haloperidol) and sedative/tranquillisers (temazepam, clonazepam, valproate) may have limited the deceased's mobility and thus predisposed to deep calf vein thrombosis. I note that the psychiatric nurse who witnessed the deceased's collapse remarked in his statement that the deceased had been '...feeling a bit drowsy of late and we have been monitoring his medication'. No other risk factors were identified.

2. analysis of a specimen of blood obtained at autopsy reportedly showed a blood alcohol concentration of nil. The blood contained a therapeutic level of olanzapine and subtherapeutic levels of valproic acid, temazepam and 7-aminoclonazepam (metabolite of clonazepam). No haloperidol or prazosin was identified in the blood although the laboratory detection limits for both were high and detection of therapeutic levels of both may not have been possible.
3. There were no injuries or other markings on the body to indicate the involvement of another person in the death.'

(Exhibit C3a, p5)

- 3.3. I accept Dr Gilbert's conclusions and find that the cause of Mr Betts' death was pulmonary thromboembolism due to left deep calf vein thrombosis.

#### **4. Background**

- 4.1. Some background information concerning Mr Betts is contained in the statement of Dr Rajani Srinivasan (Exhibit C6a). Dr Srinivasan states:

'Mr Colin Betts came to Jacaranda Ward in March 2001. He had been initially referred to the ACIS team by his local General Practitioner on 25<sup>th</sup> January 2001. The ACIS team assessed him as living in very squalid conditions with deterioration in self-care. He was also known to have abused alcohol for a long period of his life. He had been divorced from his wife for several years. He was diagnosed at Flinders Medical Centre with dementia secondary to alcohol abuse. He was subsequently sent to Patterson East Ward at Glenside Hospital.

Colin was difficult to manage due to agitation, confusion and memory loss and was therefore moved to Behavioural Support Services and Brentwood South. He was eventually placed at City Gardens but absconded and was returned to Brentwood South. He was eventually approved for high-level care in a secure dementia ward. On 12<sup>th</sup> March 2001 we assessed his suitability for placement in Jacaranda Ward because he was at risk from the younger, more disturbed patients at Brentwood Ward.

I had concerns about whether Jacaranda Ward was safe enough for Colin to prevent him from absconding. I agreed to accept him in the ward as a trial on the basis that if he was too difficult to manage he would be returned to Brentwood. A further condition was that Brentwood staff would continue to deal with the Guardianship Board regarding his detention. I made a note of my conditions and concerns in his casenotes on 12<sup>th</sup> March

2001. When he came to Jacaranda Ward he was awaiting a Guardianship Board hearing which later confirmed his detention order. I had no involvement in Colin's detention.

Colin was a big challenge to manage. Jacaranda staff were constantly alert and aware that he wanted to abscond, and he was therefore monitored very closely. Because of his short-term memory problems he was constantly asking staff about leaving the ward and going home. No matter how often or regularly staff explained to Colin the reasons for his detention, he remained preoccupied with leaving the ward. He became increasingly annoyed with his detention and showed signs of aggression and a desire to abscond.'

(Exhibit C6a, pp1-3)

- 4.2. While at Jacaranda Ward, Mr Betts' medication included Prazosin for bladder spasm and high blood pressure, Ranitidine for gastric reflux, and Olanzapine, Clonazepam and Haloperidol for his mental state. Dr Srinivasan commented:

'Despite regular changes to Colin's medication, at times he remained difficult to manage. His mental state could change from being very agitated and trying to abscond, to being drowsy and over-sedated. These extremes in behaviour were often experienced on a daily basis. To counter this problem, PRN (as required) medication was given to calm Colin when agitated, or medication doses were withheld if he was too sedated. His medication needs were therefore reviewed constantly throughout the day to achieve the most appropriate level of sedation. This is reflected in his casenotes.'

(Exhibit C6a, p4)

- 4.3. In the period before his death, the following significant incidents were noted in relation to Mr Betts:

- On 8 April 2001 Mr Betts complained of having fallen in the bathroom. He was examined by Dr Srinivasan who found no sign of injury. She gave the opinion that his condition posed no risk of developing into deep vein thrombosis ('DVT') (Exhibit C6a, p5);
- On 27 July 2001 at 2:30am Mr Betts suffered a seizure. He was seen by Dr Rhett Bosnich who noted swelling to both ankles. Dr Srinivasan saw Mr Betts later in the morning and found no sign of swollen ankles or other swelling. Dr Srinivasan commented that such swelling can sometimes appear and then disappear when the patient lies down. Dr Srinivasan said that she did not suspect DVT at that time (Exhibit C6a, p6);
- Following his seizure, Mr Betts' medication was changed (the Olanzapine was reduced) and Sodium Valproate was prescribed for seizures. He was also prescribed antibiotics as it was suspected he may have a chest infection;

- On 30 July 2001 at about 11:30am Mr Betts was taken to the Royal Adelaide Hospital for a CT scan and chest X-ray as a follow-up to the incident on 27 July 2001. He returned to Glenside Hospital at about 1:30pm. As I mentioned, at about 2pm, he collapsed in the toilet.

## 5. Issues arising at inquest

### 5.1. Quality of psychiatric treatment provided

I received a report from Professor R D Goldney, who is a Professor of Psychiatry at the University of Adelaide and has extensive experience in academic, general and hospital psychiatry as well as the medico-legal aspects of psychiatry.

### 5.2. In his report, Professor Goldney summarised Mr Betts' condition as follows:

'Mr Betts had a severe psychiatric condition, that of alcoholic dementia with a clinical picture known as Korsakoff's Syndrome. That simply refers to the specific pattern of the dementing process, and it is quite typical of chronic alcohol abuse.

In addition there was evidence of Mr Betts having cardiac problems, as indicated by the ECG examination on 27/7/01 and also by the observations of Dr Bosnich, who clinically assessed Mr Betts as having Atrial Fibrillation.

The clinical management of Mr Betts was clearly very challenging. He was insightful, with a tendency to abscond, and there was an appreciable degree of agitation recorded at times. The use of psychotropic medication to control his agitation would appear to have been quite appropriate. Furthermore, it is evident that there had been a markedly reduced dose of medication over the last few weeks of his life compared to that which he had been tolerating in the previous months. It is also evident that there had been clinical concern about the possibility of a chest infection, as the antibiotic, Roxithromycin had been prescribed from 27/7/1.

It is pertinent to note that there is ample documentation of repeated medical assessments of Mr Betts in the last few weeks of his life, with appropriate adjustment of medications.'

(Exhibit C27, pp7-8)

### 5.3. There was some concern expressed by some of the nursing staff that Mr Betts may have been over-sedated. Mental Health Nurse Alan McGowan stated:

'Colin gradually became more and more lethargic and sleepy until he was sleeping for long periods during the day. It got to the stage about one month ago that I had to wake Colin each time I had to give him his medication. In my opinion it was too much and I often withheld his dose if he was too drowsy. If I did this I would advise the CN on my shift that Colin would not be given his dose and make a note on his medication sheet. After my repeated requests to reduce his high doses of medication, his doses were cut back dramatically.'

(Exhibit C8a, p2)

- 5.4. As for the period immediately prior to his death, Mr McGowan said:

'I worked on dayshift on the weekend of 28<sup>th</sup> – 29<sup>th</sup> July 2001. During these shifts, Colin was still very drowsy and his speech was difficult to understand, particularly on the Sunday. On both those days I again withheld his medication because he was too drowsy.

On the Sunday I was told by the CN Regina Loh that I should be giving Colin his medication. I refused, saying that he was ill and shouldn't have his dose. He was so drowsy. I offered for her to give the dose to Colin, but I refused to take responsibility for that. She did not give him the dose either.'

(Exhibit C8a, p3)

- 5.5. Psychiatric Enrolled Nurse Tracey Henery expressed similar concerns (see Exhibit C14a).

- 5.6. Statements from other nurses involved in Mr Betts' treatment did not support these expressions of concern.

- 5.7. Significantly, Registered Mental Health Nurse Robert Allan stated that he accompanied Mr Betts to the Royal Adelaide Hospital during the morning of 30 July 2001 so that he could have a CT scan and chest X-ray. He said:

'Colin on this day was a little drowsy, more so than (than) usual. After I had my lunch break I took Colin in an access cab to the RAH sometime between 11:15am and 11:45am. Colin was taken in a wheelchair because he was unsteady on his feet. At the RAH Colin was fine, he was compliant with all procedures and could help himself more than I had expected given his drowsiness. I remained with him throughout the procedures. After this we returned to Glenside by access cab arriving back at the ward at about 1:30pm or so.'

(Exhibit C13a, p2)

- 5.8. Professor Goldney did not consider that Mr Betts was sedated to an inappropriate extent. He concluded:

'It is evident from some of the reports of nursing staff, particularly from Mr McGowan and Ms Henery, that there may have been differences of opinion about the administration of medication for Mr Betts's reported confusion. Quite clearly Mr Betts presented a clinical challenge, as there is clear documentation even in the last two weeks of his life of him being particularly physically active in terms of his willingness to abscond, and there is a fine line which needs to be drawn in terms of ensuring a patient's safety with appropriate medication, as opposed to inducing drowsiness. That appears to have been recognised not only by the nursing staff, but also by the medical staff. It is of importance that nursing staff do not administer medication if they believe a person is too sedated, and it is also important that they report their concerns to medical staff, as appears to have occurred.

The challenge of knowing whether or not Mr Betts may have been too sedated is illustrated well by the fact that in the hours before his death he was accompanied by a single nurse to the Royal Adelaide Hospital and, over a period of about two hours, he was able to conduct himself in an appropriate manner during further investigations. That is hardly the clinical picture of somebody who is consistently and pervasively over sedated. Similarly, other observations such as Mr Betts trying to abscond from the ward on 26/7/01 are not consistent with a person who is too sedated. Indeed, the doses of medication which he was taking would not be consistent with a clinical picture of over sedation in a person who had previously had much higher doses of the different medications. I could accept that a person who had been naïve to such medication may have been over sedated. However, that is simply not the case with Mr Betts, as he had been on appreciably greater doses of such medications prior to the last few weeks of his life.'

(Exhibit C27, pp8-9)

5.9. Failure to diagnose deep vein thrombosis

When he examined Mr Betts on the morning of 27 July 2001, Dr Bosnich found no signs of DVT. He said:

'I am aware that Colin died on 30<sup>th</sup> July 2001, and that the cause of his death was pulmonary embolism due to left deep vein thrombosis (DVT). The symptoms I would expect to find for this condition would be unilaterally hot, swollen, tender leg. I did not note any symptoms of this type. I noted that both of Colin's ankles were swollen, which is inconsistent with a DVT to one leg. There are many causes of bilateral ankle swelling including heart failure or varicose veins. Swelling under these conditions can fluctuate quite rapidly.

There is nothing to suggest on the basis of my notes that a DVT was present.'

(Exhibit C7a, p3)

5.10. Dr Srinivasan also found no sign of DVT during the morning of 27 July 2001. She commented:

'I am aware that Colin's death on 30<sup>th</sup> July 2001 was caused by pulmonary thromboembolism due to left deep vein thrombosis (DVT). The development of DVT is associated with the slowing of the blood circulation simultaneously with being immobilised for periods which tends to accentuate slowing of the blood stream. These circumstances did not apply in this case at all because, even though he may have had periods of drowsiness and sleepiness, Colin was quite active and had periods of agitation and restlessness until the day he died. There was never any time when he was passively recumbent or immobile for any significant period.'

(Exhibit C6a, p4)

5.11. Professor Goldney accepted the appropriateness of the approach taken by these doctors. He said:

'I accept that over sedation can lead to immobilisation of limbs with an increased susceptibility to pulmonary embolism. However, Mr Betts's periods of over sedation

appear to have been mixed with other periods of relative activity and there is certainly no indication in the case notes of a primary site of a pulmonary embolus having been missed.

As a psychiatrist, I am probably not the most appropriate person to be commenting in detail on a deep vein thrombosis causing pulmonary embolism. However, as a general medical practitioner, it is fair to note that death by pulmonary embolism is not uncommon and can even occur in apparently healthy persons. However, Mr Betts was a man who was far from healthy. He had a history of alcohol dementia; cardiac disease; he had had a convulsion of unknown aetiology; and he was also thought to have had a respiratory condition in the few days before his death.

I have no criticism at all of his clinical management. I consider that both the nursing and the medical care have been excellent. I appreciate that at times some members of the nursing staff have been concerned about the possibility of over sedation, and that is an indication of good nursing care. Equally, there has been good medical care, in the sense that there was continuing observation, monitoring and changing of medication at appropriate times. The clinical challenge was that Mr Betts' clinical condition was on a knife edge, so to speak, of having either too much or too little sedation. The fact that at times medication needed to be withheld is entirely appropriate and consistent with the difficult clinical situation with which the nursing and medical staff were confronted.

In summary, I have no criticism at all about the management of Mr Betts and I consider that the medication and the doses of medication were quite appropriate. In my view they cannot be implicated in his death.'

(Exhibit C27, pp9-10)

- 5.12. I accept Professor Goldney's opinions as outlined above, and have no criticism of Mr Betts' clinical management while he was a patient at Glenside Hospital in the period prior to his death.

## **6. Recommendations**

- 6.1. There are no recommendations pursuant to Section 25(2) of the Coroners Act, 1975.

*Key Words: Death in Custody; Psychiatric/Mental Illness; Deep Vein Thrombosis*

*In witness whereof the said Coroner has hereunto set and subscribed his hand and*

*Seal the 30<sup>th</sup> day of July, 2004.*

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*Coroner*