CORONERS ACT, 2003

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 15th day of July 2005 and the 30th day of August 2005, by the Coroner's Court of the said State, constituted of Anthony Ernest Schapel, Acting State Coroner, into the death of Barbara Ann O'Brien.

The said Court finds that Barbara Ann O'Brien aged 57 years, late of Carmel Court, 39 Myall Avenue, Kensington Gardens, South Australia died at the Royal Adelaide Hospital, North Terrace, Adelaide, South Australia on the 12th day of December 2001 as a result of right bronchopneumonia following hypoxic brain damage secondary to cardiac arrest. The said Court finds that the circumstances of her death were as follows:

1. Introduction and reasons for inquest

1.1. Barbara Ann O'Brien was 57 years of age when she died at the Royal Adelaide Hospital ('RAH') on 12 December 2001.

1.2. The deceased suffered from chronic schizophrenia and non-insulin dependent diabetes mellitus. Prior to her death, she had been a community patient under the care of the Felixstow Continuing Care Unit which is part of the RAH Community Mental Health Service.

1.3. Prior to her admission to the RAH the deceased had been residing at a facility known as Carmel Court at Kensington Gardens. The deceased's psychiatric illness, and her disruptive and self-harming behaviour occasioned thereby, was said to have resulted in her being evicted from the facility on 7 December 2001. The deceased had also collapsed that morning as a result, it was suspected, of hypoglycaemia, the nature of which I explain shortly. She was brought by mental health workers to the office of Dr

Jacqueline Symon who was the deceased's assigned psychiatric registrar. I have received in evidence Dr Symon's statement verified by affidavit (Exhibits C5, C5a). Dr Symon performed an examination of the deceased. The deceased was dishevelled. She had bruises to her ankles, hands, knees and face, all of which appeared to be consistent with self-harming behaviour or with falls. The deceased voiced paranoid delusions about other residents of the facility beating her up. Her insight was poor and her judgment was impaired. Dr Symon concluded that the deceased needed psychiatric admission to reassess her medication and to enable plans for her future care to be made as her current community management was not working despite extensive efforts. Dr Symon was of the view that due to the deceased's current behaviour, she was a risk to herself and potentially to others. Accordingly, Dr Symon detained the deceased pursuant to Section 12 of the Mental Health Act 1993 (the 'MHA').

1.4. In accordance with the detention order, the deceased was later that day admitted to a psychiatric ward of the RAH. Her detention was subsequently confirmed but it lapsed on 10 December 2001.

1.5. In circumstances that I will address in some detail later in these findings, in the early hours of the morning of 8 December 2001 the deceased suffered a cardiac arrest. She was revived, but suffered irreversible brain damage as a result and never regained consciousness. She finally succumbed on 12 December 2001. As of the day of her death, the deceased was no longer detained under the MHA. A three day detention order had lapsed on about 10 December 2001 and any continuation of her detention would have been superfluous in the light of her, by then, vegetative state. Nevertheless, the event that ultimately led to her death, that is to say the cardiac arrest on 8 December 2001, had occurred at a time while the deceased was still detained pursuant to an order under the MHA.

1.6. In those circumstances, in my view this inquest was mandatory for the following reasons. Although this death had been notified pursuant to the provisions of the repealed Coroners Act 1975, it was in my view to be regarded as if it were a notification of a reportable death under the Coroners Act 2003 (see Section 25(3) of the Schedule to the 2003 Act). I have therefore taken the view that all of the provisions of the 2003 Act, including those which define the circumstances in which an inquest under the 2003 Act is mandatory, apply to this death. The deceased's

death was a death in custody as defined in Section 3 of the 2003 Act because the primary cause of the death, namely the cardiac arrest, arose while she was the subject of a detention order under the MHA.

1.7. Accordingly, an inquest to ascertain the cause or circumstances of the deceased's death was mandatory by virtue of Section 21(1) of the 2003 Act. If I am wrong about that, and the repealed Act still applies, an inquest into the deceased's death was in any event mandatory pursuant to Sections 12(1)(da) and 14(1a) of the repealed Act for the same reasons, namely because the primary cause of the deceased's death arose while she was detained in custody as contemplated in the repealed Act.

1.8. A number of issues arose in the course of the investigation of this death that brought into question the quality of the medical attention the deceased received during her time in the RAH. Those issues, in my view, made it desirable in any event to hold an inquest to ascertain the cause and circumstances of the death, quite apart from its mandatory nature.

2. The deceased's physical illness

2.1. When Dr Symon detained the deceased, she gave instructions that the deceased would need to be admitted to the RAH via the Emergency Department so that her medical condition could be accurately assessed in light of her recent hypoglycaemic attack that day. In the event, the deceased was not seen at the Emergency Department but was admitted straight into Ward C3, a psychiatric ward.

2.2. It is pertinent here to describe the deceased's physical illness. She suffered from non-insulin dependent diabetes mellitus. This was a chronic condition. Diabetes is a condition where the body is unable to produce enough insulin to keep the blood sugar level (BSL) within a normal range. Unless treated, an abnormally high BSL, known as hyperglycaemia, will result. Such a level will compromise the health of the person in ways that need no discussion here. In order to combat an abnormally elevated blood sugar level, certain medication may be administered to reduce that level. However, such therapy can reduce the BSL to an abnormally low level. A lack of proper nutrition can also lead to an abnormally low BSL, even in the diabetic. Such a low BSL may be the combined result of the medication and inadequate nutrition. An abnormally low BSL is known as hypoglycaemia. A person suffering from hypoglycaemia may, if the condition is untreated, collapse, lose consciousness or fall

into a coma. As seen, it was said that the deceased had suffered such a hypoglycaemic collapse on the morning of 7 December 2001. In addition, it is also said that she had suffered a hypoglycaemic collapse on 1 December 2001. As well as receiving hypoglycaemic medication, the deceased's nutrition had been compromised by delusions emanating from her psychiatric illness. These combined circumstances may have caused or contributed to the deceased's hypoglycaemia.

2.3. A post-mortem examination of the body of the deceased was performed by Dr Ross James, a Forensic Pathologist then attached to the Forensic Science Centre. In his post-mortem report (Exhibit C3a) Dr James expresses the opinion that the cause of the deceased's death was right bronchopneumonia complicating diabetic hypoglycaemia. He concluded that the deceased's pneumonia was '*clearly a complication of her comatose state following the episode of clinically documented hypoglycaemia*'. Although there can be no doubt that the cause of the deceased's death was right bronchopneumonia, there is now reason to question Dr James' observation that this was a complication of the deceased's diabetic hypoglycaemia. For reasons that I will discuss, another interpretation of the deceased's pathology is that the comatose state that led to the pneumonia was the result of hypoxic brain damage caused by a prolonged period of cardiac arrest that was caused not by hypoglycaemia in itself, but by other complicating parameters. This issue is important because insofar as there is the suggestion that while the deceased was a patient in the RAH her hypoglycaemia was inadequately monitored or treated, such perceived inadequacies may or may not have had any bearing on the deceased's cardiac arrest and consequent death. I deal with that issue in these findings.

3. <u>The deceased's admission to hospital</u>

3.1. As seen, it was Dr Symon's intention that the deceased be admitted to the RAH through the Emergency Department so that her physical condition could be addressed. This did not occur. She was admitted directly to the psychiatric Ward C3 at about 4pm on 7 December 2001. She had been observed in the waiting area of the ward at about 3pm by a Dr Latt Latt Aung, a Psychiatrist who had previously dealt with the deceased at the Felixstow Continuing Care Unit . They had exchanged greetings and from that I infer that the deceased's state of consciousness was not overtly altered. I received in evidence Dr Aung's statement verified by affidavit (Exhibits C6, C6a). The deceased was placed in a bed at about 4pm according to the statement verified by

affidavit of Anna Vukovic, a Registered Nurse in the ward (Exhibits C7, C7a). Not only was the direct admission to the psychiatric ward in contravention of the instruction given by Dr Symon, it was also contrary to what is said to have been the hospital's usual practice, namely to medically clear patients through the Emergency Department (statement of Vukovic). Whether this had any bearing on the outcome is another matter.

3.2. Upon her admission, RN Vukovic obtained a BSL reading from the deceased. She was found to be hypoglycaemic with a level of 2.1mmol/L. A BSL should normally be 5mmol/L or above. In order to raise her BSL, she was immediately given a drink of sugar infused Milo and was given another at about 5pm. She was given food at about 6pm, but only consumed half a tomato. She demonstrated a reluctance to eat the rest of the food presented.

3.3. Dr Aung made an assessment of the deceased sometime between 6pm and 7pm. He was aware of a history of hypoglycaemic attacks and of her diabetes. Dr Aung was also aware of the low BSL that had been taken earlier. In the light of that, he requested that her BSL be checked at six-hourly intervals, that her oral intake of food be encouraged and that her diabetic medication, which would have a tendency to exacerbate hypoglycaemia, be withheld for the time being. He also made a request that an on-call medical doctor be called if her physical condition deteriorated. Dr Aung explains in his statement that he did not conduct a medical review at that time. Although aware that it was the usual practice for patients to go through the Emergency Department before admission to a psychiatric ward, he claims that well known patients like the deceased were sometimes admitted directly to the ward. He was unaware of Dr Symon's specific instruction that she go through the Emergency Department.

3.4. Although the deceased had a low BSL that afternoon and evening, and stated that she was tired, it is important to observe that there is no suggestion that her level of consciousness was affected. Dr Aung describes the deceased as suspicious and somewhat hostile towards him and she appeared to be preoccupied with grief over the death of her father thirty years ago and with the wealth that he had left her, no doubt all in keeping with the deceased's psychiatric condition, but she was nevertheless '*well conscious and orientated* (sic)'. Her BSL was next taken at 8:45pm. It had risen slightly to 2.4mmol/L. At 9pm the deceased took her medications, with the

exception of her withheld diabetic medication. RN Vukovic checked on her at 10pm and 11pm when the deceased was observed at the latter time to be asleep in bed. At 11:15pm she was observed by Registered Nurse Jennifer Elliott to be asleep in bed and '*breathing normally*' (statement verified by affidavit, Exhibits C9, C9a). RN Elliott made a similar observation at 12:20am. This appears to have been the last observation of the deceased before she was located at about 1:28am on the floor of her room. The last BSL taken was that taken at 8:45pm, the level of 2.4mmol/L. It was anticipated that in accordance with Dr Aung's instructions it would have been re-taken at about 2am, according to RN Elliott's statement. In spite of the low BSL recorded at 8:45pm, there is again no suggestion of a compromised state of consciousness, as opposed to sleep, until her collapse some time before the discovery at about 1:28am.

4. The deceased's collapse and resuscitation

4.1. At about 1:28am Registered Nurse Chris Hall was performing his rounds of the ward when he discovered the deceased lying face down on the floor of her room. She was not breathing and was in cardiac arrest. Cardio-pulmonary resuscitation was initially administered. The Cardiac Arrest Team arrived and achieved a spontaneous cardiac rhythm. The deceased was then transferred to the Intensive Care Unit.

4.2. During the course of the deceased's resuscitation, RN Hall took a BSL. He made no written note of the reading but recalled that it was '*fairly high at about 17mmol/L*'. This level represented a significant increase from the previously recorded BSL of 2.4mmol/L at 8:45pm.

5. The deceased's treatment and death following her collapse and revival

5.1. There is no doubt that the deceased had suffered a significant hypoxic brain injury consequent upon her cardiac arrest. An hypoxic brain injury results from oxygen deprivation in the tissues. This injury was diagnosed before she died. I received in evidence the statement verified by affidavit of Dr Robert Young (Exhibits C10, C10a). Dr Young states that a CT scan showed a loss of grey-white differentiation consistent with early cerebral oedema secondary to hypoxic brain injury. The deceased had poor neurological function with no response to stimuli. Dr Young assessed her as having suffered a severe hypoxic brain injury secondary to cardiac arrest with a prolonged down time, that is to say the duration of the period during

which her brain had been deprived of oxygen, of at least 25 minutes. Her prognosis for neurological recovery was assessed by Dr Young and Drs Finniss and Koopowitz as hopeless. After discussion with members of the deceased's family, it was decided that further aggressive treatment would cease. Individual members of the deceased's family appear to have adopted different attitudes to this decision, but I need not discuss that issue further. The deceased died at about 9:30am on 12 December 2001.

6. The cause of the deceased's death

6.1. At post-mortem examination, Dr James found that there was consolidation of the right lung consistent with established pneumonia in its middle and upper lobes. The left lung appeared to be normal. Dr James describes this pathology as the primary cause of the deceased's death, that is '*right bronchopneumonia*', but that it complicated diabetic hypoglycaemia, more specifically that the right sided pneumonia is a complication of the comatose state following hypoglycaemia. It is true that hypoglycaemia, if left untreated, may lead to coma if Dr James' report is to be read in that fashion. However, that there was a clear diagnosis made during life of a severe hypoxic brain injury following immediately upon a well documented cardiac arrest gives rise to the conclusion that the coma was the direct consequence of the cardiac arrest. I now deal with the possible causes of the cardiac arrest.

6.2. I received in evidence the report of Professor Anne-Maree Kelly who is an emergency physician (Exhibit C18). Professor Kelly is a medical practitioner in Victoria. She is currently Professor and Director of Emergency Medicine at Western Hospital in Melbourne. She has had 15 years' experience as an emergency specialist. Professor Kelly also gave evidence before me. She has been asked to give an overview of the deceased's treatment and has also given an opinion as to the likelihood of various mechanisms leading to death.

6.3. Professor Kelly expressed the view, both in her report and before me, that the most likely cause of the cardiac arrest was a cardiac arrhythmia resulting from a number of factors that placed her at a higher risk of arrest. Those factors do include hypoglycaemia, but in her view that condition did not in itself lead to cardiac arrest. Rather, other factors including the action of her antipsychotic medication and significant coronary artery disease had also to be considered. That the deceased had a susceptibility to cardiac arrhythmia was, in Professor Kelly's view, supported by

reports of the deceased's sister, Gail Holland, whose statement verified by affidavit I received in evidence (Exhibits C1, C1a), that the deceased had reportedly recently complained of palpitations. Professor Kelly concluded that if hypoglycaemia, caused either by an overdose of hypoglycaemic medication or without any such overdose, was responsible in itself for the cardiac arrest, coma followed by cardiac arrest as a late event would have been the likely presentation (T17). There was no evidence of coma prior to the cardiac arrest in this case. As seen earlier, the deceased had been conscious and responsive. Although she was observed sleeping from about 11pm onwards, there is no suggestion that she was in any way, or at any time, comatose. The fact that the deceased could remain in an unaltered conscious state with a BSL between 2mmol/L and 3mmol/L added weight to the view that her hypoglycaemia was having little or no effect on her level of consciousness. Given that the deceased was at all material times conscious, interactive and alert, there was in Professor Kelly's view nothing to suggest severe hypoglycaemia in her clinical presentation. If severe hypoglycaemia was present, Professor Kelly would have expected to see confusion, drowsiness or unconsciousness of which there was no evidence. Professor Kelly had never experienced cardiac arrest from hypoglycaemia without a preceding coma (T20). Professor Kelly described the BSL of the deceased as evidencing moderate hypoglycaemia and that cardiac arrest is a very uncommon event and usually only occurs when the BSL is very low and then only after a protracted period of something like 6, 8 or 12 hours.

6.4. As to the BSL of about 17mmol/L taken at the time of the resuscitative measures, Professor Kelly opined that the significant increase could to a degree reflect the sudden physiological insult occasioned by the cardiac arrest but in any event was an indication that the deceased was not hypoglycaemic at the time of her collapse (T17).

6.5. Professor Kelly discounted other possibilities such as myocarditis caused by medication or a head injury from a fall as leading to cardiac arrest because there was simply no evidence of either at autopsy. In my view, such possibilities can in fact be discounted for that reason.

6.6. Professor Kelly considered that the greatest risk of cardiac arrest was posed by the medication that the deceased was taking, placed as it was against a background of diabetes and chronic hypoglycaemia (as opposed to an acute severe hypoglycaemia of which there was little evidence in her view) and of coronary artery disease. The

deceased was taking Clozapine, Lithium and Chlorpromazine for her psychiatric illness. Sudden cardiac death is reported to have a rate of about 7 in 1,000 in relation to Clozapine administration. The risk associated with the taking of Clozapine combined with other antidepressants was heightened. Professor Kelly thought that the deceased:

> '.. had an arrhythmia of her heart contributed to by her antipsychotic therapy, her underlying diabetes, with perhaps a lesser contribution being her established coronary artery disease and potentially her chronically low level of blood glucose.' (T23)

As to the contribution of the diabetes and chronic low blood glucose, over apparently a sustained period of time, Professor Kelly pointed out that both could have contributed to abnormal CT parameters of heart function, that is the electrical patterns of heart function. Naturally, this would not have helped if heart function had been adversely affected by her antipsychotic medication.

6.7. As to the deceased's earlier reported collapses, namely on 1 December 2001 and the morning of 7 December 2001, the day of her admission, Professor Kelly doubted that they were hypoglycaemic episodes. She stated:

> 'Firstly starting with Mrs Holland's statement, Ms O'Brien has complained to her of palpitations. Palpitations are very commonly associated with abnormalities of heart rhythm. Ms O'Brien had also had a number of episodes of collapse or near collapse from which she recovered spontaneously. In a hypoglycaemic situation she would have needed therapy to improve, whereas with short episodes of heart rhythm it would correct spontaneously, and I think there are at least two or three episodes evident in her record, one on the day of her admission and one the week before.' (T14)

Professor Kelly was of the view that the more likely cause underlying these earlier episodes was an abnormality of heart rhythm rather than an abnormality of glucose.

6.8. Having considered Professor Kelly's evidence in conjunction with the opinions expressed by Dr James, in my view it could not be said on a balance of probabilities that the cardiac arrest suffered by the deceased some time before 1:28am on 8 December 2001 was caused in itself by a low BSL. The fact that the deceased did not demonstrate any decreased level of consciousness at any point in time when she was said to be hypoglycaemic points very much in my view to there being no real and immediate connection between her BSL and the cardiac arrest. In this regard, I have considered very carefully the possibility that there was, as Professor Kelly said there

would be if hypoglycaemia had played a role in the arrest, a period of coma prior to that arrest but which was not detected by nursing staff. Although the deceased was seen to be awake at 10pm, this was the last occasion on which she was so observed. Her collapse was not detected until 1:28am and observations in the intervening period recorded her as being asleep. The possibility that the deceased had lapsed into a coma and had not merely fallen asleep needed to be considered. Professor Kelly acknowledged that it was sometimes difficult to tell whether a person is asleep or in a coma if they are not woken up, and there was no attempt by nursing staff to wake up the deceased. However, she doubted from her perusal of the clinical record that the deceased was comatose before her detected collapse (T21) and it seems to me that to conclude that the deceased was comatose would be to draw a long bow and in any case to indulge in speculation. Again, everything in my opinion pointed to the fact that the deceased remained conscious and responsive in spite of her low BSL. Her hypoglycaemic medication was stopped and she was given glucose enriched substances that had resulted by 8:45pm in an increased BSL. In those circumstances, it is impossible to say that hypoglycaemia led to cardiac arrest, especially when there is an extremely plausible alternative explanation, that is to say, the possible effects of the deceased's medication against the background of known disease as Professor Kelly as postulated. The possible role of Clozapine was also postulated by Dr Koopowitz, a Psychiatrist who did not treat the deceased but who was involved in the decision to stop further aggressive treatment of the deceased. He states in Exhibit C11a that a recognised complication from Clozapine use is that any level of it can affect heart rhythm.

6.9. What is clear is that the deceased died from right bronchopneumonia. Professor Kelly would not dispute Dr James' conclusion in that regard. It is also clear to me that the bronchopneumonia was consequent upon the hypoxic brain injury resulting from the prolonged period of cardiac arrest. In the circumstances, for the reasons discussed above, I am not prepared to extrapolate from there in order to assign a cause for the cardiac arrest. The cause of death can adequately be expressed as right bronchopneumonia following hypoxic brain damage secondary to cardiac arrest.

7. Criticisms of the deceased's care at the Royal Adelaide Hospital

7.1. There are said to have been a number of shortcomings in the deceased's care.

7.2. She was not admitted via the Emergency Department but straight to the psychiatric ward thereby bypassing what conceivably would have been a more thorough medical examination.

7.3. The deceased was not given frequent BSL tests. The only tests that occurred were administered at 4pm and 8:45pm. Dr Aung ordered six-hourly tests when, it is said, she should ideally have had them hourly until her BSL was seen to have increased into the normal range (report of Professor Kelly, Exhibit C18, paragraph 2).

7.4. The deceased was not given an IV dextrose infusion when it was apparent that she was only prepared to accept nutrition with some reluctance (Exhibit C18, paragraph 2).

7.5. I do not need to pass any judgment or comment upon those criticisms for two reasons. Firstly, it will be noted that those criticisms in the main relate to the management of the deceased's hypoglycaemic condition. Professor Kelly said in her report:

> 'In summary, it is my opinion that an arrhythmia is the most likely cause of Mrs O'Brien's cardiac arrest, potentially contributed to by coronary artery disease, hypoglycaemia and her psychiatric medications. It is not possible to judge whether closer attention to her BSL levels could have avoided her death, but closer monitoring and specific intervention would have been desirable.'
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> (Exhibit C18, p3)

She said in evidence:

> 'I think that – these cases are always difficult, patients with psychiatric illness and coexisting medical problems are always difficult to manage and choosing the right setting to manage is a fine line. Trying to judge what's best for them; what is going to bring the best outcome, but also is going to be less upsetting for them and upsetting for others. I think that certainly there should have been closer monitoring of her blood sugar, but even with that I doubt that the cardiac arrest could have been avoided. I think it would have occurred if she'd still been in the Emergency Department or in a medical ward of if they'd been checking her blood glucose every hour. I don't think that setting would have stopped it happening.' (T23)

I agree with those observations and there is in my view insufficient evidence to conclude on a balance of probabilities that the deceased's acute hypoglycaemia played a role in the deceased's collapse on the morning of 8 December 2001 and her

death on 12 December 2001. Secondly, and in any event, I am informed that the RAH conducted what is called a Sentinel Events Review in relation to the deceased's death, made certain recommendations consequent upon its findings and implemented those recommendations. All of this is explained in the statement of Raili Tanska, a Quality Improvement Coordinator of the RAH Community Mental Health Services (Exhibits C13, C13a) and of Marc Cunningham who was at the time of the holding of this Inquest the Clinical Nurse Consultant of Ward C3 at the RAH (Exhibits C15, C15a). I need say no more than that. Those statements seem to me to adequately address the issues raised in the Inquest.

8. Conclusions

8.1. The deceased was admitted to the RAH on 7 December 2001 having been detained by Dr Symon pursuant to Section 12(1) of the MHA. I find that the detention was lawful in all of the circumstances.

8.2. I am informed in the statement of the investigating police officer, Christopher Scott Walkley (Exhibits C14, C14a) that the original detention order was confirmed by a Dr Beckwith at about 10:30am on 8 December 2001. This took place, of course, after the deceased's collapse and placement in the Intensive Care Unit in her comatose state. I need not comment as to whether the confirmation in all of those circumstances was necessary or indeed lawful. Suffice it to say for current purposes, the deceased remained at least in de facto detention from that time until the order expired a day or two before her death.

8.3. The deceased suffered a cardiac arrest some time before 1:28am on 8 December 2001.

8.4. The deceased's period of cardiac arrest resulted in her sustaining a severe hypoxic brain injury that ultimately led to her contracting right bronchopneumonia.

8.5. I am unable to find on the balance of probabilities that the cardiac arrest was caused by the deceased's hypoglycaemic state as it was diagnosed on 7 December 2001. Although the alternative explanation for the cardiac arrest postulated by Professor Kelly is an attractive one, I make no positive finding that it provides the explanation for the arrest.

8.6. I find the cause of the deceased's death to be right bronchopneumonia following hypoxic brain damage secondary to cardiac arrest.

9. Recommendations

9.1. In the light of my findings and the steps taken by the RAH to rectify any perceived shortcomings in the deceased's treatment, I do not consider it necessary or appropriate to make any recommendations pursuant to Section 25(2) of the Coroners Act 2003.

Key Words: Psychiatric/Mental Illness; Hospital Treatment; Hypoglycaemia

In witness whereof the said Coroner has hereunto set and subscribed his hand and Seal the 30th day of August, 2005.

Acting State Coroner

Inquest Number 16/2005 (3289/2001)