



FINDING OF INQUEST

An Inquest taken on behalf of our Sovereign Lady the Queen at Adelaide in the State of South Australia, on the 7th, 8th, 9th 10th and 17th days of August 2006, and the 25th day of September 2006, by the Coroner's Court of the said State, constituted of Mark Frederick Johns, State Coroner, into the death of Peter Malcholm McLeod.

The said Court finds that Peter Malcholm McLeod aged 28 years, late of 3/9 Cuthero Terrace, Kensington Gardens died at the Royal Adelaide Hospital, North Terrace, Adelaide, South Australia on the 1st day of November 2003 as a result of Acute Dopamine Imbalance Syndrome. The said Court finds that the circumstances of his death were as follows:

1. Introduction and reason for Inquest

- 1.1. Peter Malcholm McLeod, who was 28 years of age, died at the Royal Adelaide Hospital on 1 November 2003.
- 1.2. Mr McLeod was born in South Africa where he lived until January 2001. He then came to Melbourne and worked in his occupation of computer programmer. He met his wife in Melbourne and after a return visit to South Africa they arrived in South Australia in March 2003.
- 1.3. Dr Allan Cala carried out a post-mortem examination on 3 November 2003 giving the cause of death as consequences of epilepsy. A macroscopic examination of Mr McLeod's brain was carried out by Dr Grace Scott, the report of which was received as Exhibit C2a in these proceedings. The report indicated brain swelling but otherwise a macroscopically normal brain. A microscopic examination was also

conducted by Dr Grace Scott and the report of that examination was received as Exhibit C2b in these proceedings. That examination reported that microscopy of the brain showed evidence of diffuse hypoxic injury in the cerebral hemispheres, brain stem and cerebellum. This appeared fresh and could be consistent with hypoxic injury secondary to seizure activity during admission. However, Dr Scott reported that macroscopic examination of the brain demonstrated generalised swelling with early bilateral tentorial herniation, and it was possible that some of the hypoxic changes were a consequence of raised intracranial pressure. No evidence was found of pre-existing hippocampal sclerosis, nor were any other epileptogenic structural lesions found on examination of multiple sections from each Ammon's horn. There was no evidence of any underlying infective process in any of the sections examined.

- 1.4. At the time of his death on 1 November 2003, Mr McLeod was detained pursuant to the Mental Health Act 1993. His death was therefore a death in custody as defined in the Coroners Act 2003 and this Inquest was required by virtue of section 21(1)(a) of that Act.
- 1.5. On 26 September 2003 Mr McLeod had a motor vehicle accident. It appears that he had an argument with his wife and left the house in his Daewoo motor vehicle. His home address was 3/9 Cuthero Terrace, Kensington Gardens. As he drove his vehicle down Cuthero Terrace, he collided with a parked Mitsubishi sedan on the north side of Cuthero Terrace outside number 29.
- 1.6. Constable Smith gave evidence of attending at the accident scene and then at the Accident and Emergency Department at the Royal Adelaide Hospital where he spoke with Mr McLeod. Constable Smith recalled that Mr McLeod told him that as he was driving on Cuthero Terrace he had heard voices in his head. These voices told him a variety of things, including a command that he crash his vehicle into the parked Mitsubishi which was ahead of him¹.
- 1.7. Mr McLeod was initially assessed at the Royal Adelaide Hospital by Dr J Huang who made a diagnosis of 'psychosis - acute'.
- 1.8. On 26 September 2003, Mr McLeod was seen by the Psychiatric Registrar Dr Angus who, according to the hospital notes, had quite an extensive interview with

¹ Exhibit C13a

Mr McLeod. He decided to commence Mr McLeod on risperidone (an antipsychotic medication) on an increasing regime of dosage. He also ordered what he referred to in the notes as an 'organic work-up'. This was to determine his physical medical condition. Significantly, Dr Angus referred Mr McLeod for a CAT scan, an EEG, and urine tests.

1.9. The referral for the EEG states under 'Reason for referral':

'First presentation of psychosis. No past history of illness. Wish to exclude organic cause.'²

1.10. During this period, from his first presentation after his motor accident on 26 September 2003, Mr McLeod was detained at Brentwood Ward on the Glenside Campus of the Royal Adelaide Hospital. Brentwood is a closed ward. On 1 October 2003 he was admitted to Ward C3 at the Royal Adelaide Hospital. Ward C3 is a psychiatric ward, but not a closed ward. Although he was under detention he was able to move around the Royal Adelaide Hospital more or less at will, as distinct from the regime which applied at Brentwood which was far more restrictive.

2. Dr Michelle Kiley

2.1. Dr Michelle Kiley, Neurologist, gave evidence at the Inquest. She is Director of Epilepsy Services at the Royal Adelaide Hospital and a Consultant Neurologist at that hospital. She runs the EEG Telemetry Unit. She explained that an 'EEG' is an acronym for electroencephalogram which is a test to measure the electro cerebral rhythms of the brain by means of probes placed on the scalp. It takes approximately 20 minutes to record these rhythms while the patient is in a resting state. Dr Kiley explained that psychiatrists often refer patients for screening tests for first presentation psychosis. She said that the most useful indication for an EEG is patients who have epilepsy but she also said that it is possible to get normal EEGs for epileptics.

2.2. Dr Kiley stated that the purpose of referral of a first presentation psychotic patient is to attempt to exclude any underlying organic causes for their presentation.

2.3. Dr Kiley stated that she reported an EEG for Mr McLeod on 20 October 2003. The test was conducted on 17 October 2003, the preceding Friday. She stated that the

² Exhibit C19

referral was typical of requests received by her Department for exclusion of organic causes in patients with first presentation psychoses. She stated that the EEG technician triages EEG requests and there is often a two to three week delay for routine screens such as this one which was, it will be recalled, first referred on 26 September 2003.

- 2.4. Dr Kiley stated that she could recall independently of the notes her reporting of Mr McLeod's EEG. She noted that during the test on 17 October 2003, Mr McLeod was alert and cooperative. The EEG report stated under the heading 'Conclusion':

'Very abnormal EEG. The background rhythm is abnormal with focal slow and sharp wave activity over the temporal regions (L>R) and frequent paroxysmal generalised epileptiform discharges are evident. The features are more in keeping with a partial seizure focus with secondary generalisation.'³

- 2.5. Dr Kiley stated that in seven years of doing approximately 200 EEG reports per year for first presentation psychotic patients she had never seen such an abnormal EEG in a psychiatric referral of that kind.
- 2.6. She did not think that his presentation 'rang true' as epilepsy. Had she been asked at that time, she would have recommended brain imaging, a CT head scan (she was not aware that a CT scan had already been carried out for his at that stage and had been reported as normal) possibly an MRI scan and a full metabolic and septic screen. She would have also carried out a CSF study (that is a lumbar puncture to obtain cerebrospinal fluid for examination).
- 2.7. Dr Kiley was not contacted by the psychiatric team after the delivery of this abnormal EEG report. Instead, amongst other things, sodium valproate was prescribed by the staff on Ward C3. Dr Kiley stated that this is an anti-epileptic drug which in her opinion should not be prescribed prophylactically. She did note that psychiatrists use sodium valproate for mood stabilisation. She did not know why it had been prescribed in this case.
- 2.8. Dr Kiley was asked whether she contemplated contacting the psychiatrists herself in view of the abnormal EEG report. She stated that she did not because she fully expected to get a call from the psychiatric team to request that she assess the patient clinically. She stated that it is 'protocol' for the referring team to contact the

³ Exhibit C12j

neurologist. She said that if Mr McLeod had been staying in the community or going into the community she would have contacted the psychiatric team. She stated that she was not concerned when she did not immediately get a call from the psychiatric team because it was possible that another member of her Department, including registrars, might have been contacted in her place. Therefore, she thought no more about the matter until Mr McLeod was readmitted on 29 October 2003.

- 2.9. As will be apparent in due course, Mr McLeod was discharged from Ward C3 on 22 October 2003 to the 'Hospital at Home' program for ongoing treatment. However, he was readmitted on 29 October 2003 with a worsening condition.
- 2.10. Dr Kiley saw him on 31 October 2003 to review an MRI scan which had been taken. This was an abnormal MRI. Subsequently a brain perfusion study was conducted with a diagnosis of brain death and on the morning of 1 November 2003 respiratory support was withdrawn, and Mr McLeod was certified deceased at midday that day.
- 2.11. Dr Kiley stated that in her opinion Mr McLeod's cause of death was 'lethal catatonia'. She did not think he had epilepsy. She stated that it can be clinically very difficult to distinguish between epilepsy and catatonia.

3. Dr Shane Gill

- 3.1. Dr Shane Gill is a Consultant Psychiatrist. He is now in private practice but was, in October 2003, Director of Clinical Service, Community Mental Health Service, Royal Adelaide Hospital. He stated that he was not involved when Mr McLeod was at the Brentwood Ward at Glenside Campus. He became involved when Mr McLeod was transferred to Ward C3 at the Royal Adelaide Hospital. Dr Gill was aware that Mr McLeod was suffering from command auditory hallucinations and was under detention with antipsychotic medication.
- 3.2. Dr Gill stated that over the couple of weeks since the motor vehicle accident Mr McLeod had improved in his condition and his paranoia and hallucinations had reduced. By 1 October 2003 he was felt fit to be transferred to an acute ward, namely Ward C3 at the Royal Adelaide Hospital. Dr Gill gave some evidence about the Hospital at Home program. He stated that he was involved in the development of the program. He stated that the patient and the patient's family must consent to

participation in a Hospital at Home program in order for it to be implemented. He stated that there are 12 people on Hospital at Home at any given time.

- 3.3. Dr Gill stated that the abnormal EEG report did not preclude Mr McLeod from participating in the Hospital at Home program. This was because it would not be standard practice to 'arrange admission purely on the basis of an abnormal EEG'⁴.
- 3.4. Dr Gill stated that at the time of his transfer to the Hospital at Home program, Mr McLeod seemed suitable for it. He stated that there was a management plan in place for the abnormal EEG, namely the prescription of sodium valproate. He stated that sodium valproate was not commenced for Mr McLeod because of any psychotic issue but because of the abnormal EEG. It should be noted that sodium valproate is an anticonvulsant as well as a drug for use in addressing mood disorders, and it follows that the sodium valproate, if not being given for mood disorders or a 'psychiatric issue' was being given as a prophylactic for any epileptic seizures. It is notable that Dr Kiley had given evidence that sodium valproate should not be used prophylactically.
- 3.5. Dr Gill stated that the EEG report was not brought to his attention before Mr McLeod's discharge to the Hospital at Home program. He stated that had it been brought to his attention he would have suggested that the neurological registrar be contacted to see what should be done about the EEG report. Dr Gill stated that the sodium valproate would have been prescribed because of the suggestion in the EEG report of partial seizure focus which he stated warranted the prescription of an anticonvulsant medication such as sodium valproate. He stated that this would not normally be done on the basis of an EEG alone, but this EEG used the expression 'very abnormal' and so it happened on this occasion. He stated that it was not necessarily unusual to prescribe the drug without consultation with the neurologist (although he could not say if any such consultation might have occurred between Registrars - the hospital notes do not provide any evidence of a consultation). He stated that on 24 October 2003 he was informed by Dr Kuruvilla of the abnormal EEG report and of the fact that an appointment had been made for Mr McLeod, who was then on the Hospital at Home program, to see the neurological service at the Royal Adelaide Hospital.

⁴ Transcript, Page 108

4. **Dr Latt Latt Aung**

- 4.1. Dr Latt Latt Aung gave evidence at the Inquest. She was the Senior Psychiatric Registrar in the Hospital at Home program in October 2003. She stated that Mr McLeod was referred to the Hospital at Home program on 22 October 2003. She assessed him at his home at Kensington Gardens on 23 October 2003. She spoke to Dr Kuruvilla from Ward C3 to see if a neurological consult had been organised to deal with the EEG report. She also contacted Dr Koopowitz (a psychiatrist in private practice who has a special interest in neurological conditions). However an appointment could not be made with Dr Koopowitz for some four and a half months. Accordingly Dr Aung asked Dr Kuruvilla to organise a neurological consult at the Royal Adelaide Hospital as it could be obtained more quickly through Ward C3 than through the Hospital at Home program. She stated that a reference in the notes on 23 October 2003 showed that a nurse had contacted Ward C3 to request that the C3 treating team obtain a neurology consult.
- 4.2. Dr Aung said that she assumed that the sodium valproate had been started following a discussion between the psychiatric registrar on Ward C3 and the neurological registrar in the Neurology Department. Dr Aung did not think that Dr Kuruvilla had started Mr McLeod on the sodium valproate for his psychiatric condition, but rather for his epilepsy (or suspected epilepsy). She said that she did not see any neurological symptoms when she saw Mr McLeod but she hoped that the C3 arrangements for consultation with the neurologist might occur within two or three days.
- 4.3. Dr Aung stated that on 29 October 2003 she was informed that Mr McLeod's wife Jacqueline had contacted the program earlier that morning to advise that the previous night Mr McLeod had been very disturbed. He had not slept all night, was urinating in the hallway of the house, hyper salivating and was not responsive. On being informed of this Dr Aung cancelled all of her other commitments and urgently arranged to visit Mr McLeod at home. She contacted Ward C3 to inform Dr Kuruvilla that Mr McLeod would probably be returning to hospital that day. When she arrived at Mr McLeod's home at approximately 10am she noted, according to the hospital notes, that he was 'almost catatonic'. She stated that he was making humming noises. He was not totally catatonic because he was able to move and walk.

- 4.4. Dr Aung said in evidence that Mrs McLeod informed her that the previous night she had contacted the regional triage nurse in the Hospital at Home program who had offered to admit Mr McLeod to the hospital in view of his deterioration. However, Mrs McLeod decided to keep him overnight and call in the morning⁵.
- 4.5. Dr Aung stated that in view of Mr McLeod's condition she elected to take him to the Emergency Department rather than Ward C3. She was concerned about any organic problems that he might have. She called Dr Kuruvilla and informed her of this and then called an ambulance. She followed Mr McLeod in the ambulance to the hospital where she conducted a handover to the mental health nurse in the Emergency Department.

5. **Professor Robert Goldney**

- 5.1. Professor Goldney is Professor of Psychiatry at The University of Adelaide and has a long and distinguished career in psychiatry and in medico-legal work. He provided two reports on Mr McLeod's treatment, the first of which was admitted as Exhibit C22 in these proceedings, and the second as Exhibit C22a.
- 5.2. Professor Goldney thought that Mr McLeod had depressive components and schizophrenic components to his condition. Professor Goldney stated that the history obtained over the period of five or six weeks of Mr McLeod's treatment showed that he had had auditory hallucinations for two to three years. Professor Goldney stated that such hallucinations for as little as six months would provide a sufficient basis for a diagnosis of schizophrenia. The severity of the hallucinations was evident from the fact that Mr McLeod obeyed the command to drive his vehicle into parked vehicles, which evidenced a severe schizophrenic disorder.
- 5.3. Professor Goldney stated that the carrying out of the EEG test to exclude organic causes for Mr McLeod's condition was appropriate.
- 5.4. Professor Goldney stated that the EEG report of 20 October 2003 was 'very worrying'. He stated that one has to try to correlate it with the clinical picture at the time. He stated that had he been the treating psychiatrist, he would have wanted to speak to the neurologist about the report. He stated that he would regard it as indicative of an epileptic illness in referring to a 'partial seizure focus'. Professor

⁵ Transcript, Page 158

Goldney stated that it was difficult to say who had responsibility for instituting contact between clinicians in relation to the EEG. He stated on the one hand it might be the psychiatric team on receipt of the report two or three days later, or the neurologist as soon as she wrote the report. He stated that he would like the neurologist to ring him, but on the other hand that the neurologist may not have been as alarmed about the report as he would have as a psychiatrist.

- 5.5. Professor Goldney had some reservations about the institution of sodium valproate by the psychiatric team. He stated that sodium valproate is good as a mood stabiliser but that he suspected that it was motivated by the EEG and that the proper approach would have been to have contacted the Neurology Department to inquire what particular anticonvulsant was recommended. He thought that the sodium valproate was introduced to 'cover all bases' and that in retrospect it should not have happened without a neurology review.
- 5.6. Professor Goldney was aware of the Hospital at Home program and thought that Mr McLeod's treatment and care on the Hospital at Home program was quite impressive. He considered that the level of management was, to use his word, 'excellent'. He would have felt more comfortable if the hospital had spoken to the Neurology Department before sending Mr McLeod on the Hospital at Home program but in the absence of any clinical manifestations of seizure activity he could see why they had not taken that step.
- 5.7. Professor Goldney stated that there was a reference on 27 October to 'thought blocking' and that on that day Mr McLeod appeared 'perplexed'. Professor Goldney thought that this was the first indication of his psychotic condition worsening, but it was not sufficient to warrant readmission to Ward C3 at that stage.
- 5.8. Professor Goldney believed that the cause of death was lethal catatonia. He stated (at T227) that this is a condition which has been known since the nineteenth century and was a clinical description of what happens to some people with a psychiatric disorder. They can become feverish, suffer muscle rigidity, overactivity, a clouding of consciousness and then ultimately death. He stated that this condition has not been observed as frequently since the introduction of antipsychotic medications. What has been observed is the condition Neuroleptic Malignant Syndrome. He stated that some people suggest that Neuroleptic Malignant Syndrome is just a different name for

lethal catatonia. The expression 'neuroleptic' is a reference to antipsychotic medication. Neuroleptic Malignant Syndrome is an extremely adverse response to antipsychotic medication.

- 5.9. Professor Goldney stated that he arrived at the diagnosis of lethal catatonia by exclusion. He stated that Mr McLeod never really had a fever and that for a diagnosis of Neuroleptic Malignant Syndrome one needs to find a fever of more than 37.5 degrees at least twice, and rigidity and increased creatine kinase ('CK').
- 5.10. Professor Goldney stated that there was no real muscle rigidity in Mr McLeod, and that his CK levels were in the normal range. He stated that he had an altered state of consciousness but in the absence of fever, increased CK and muscle rigidity he reached the conclusion that the cause of death was, by a process of exclusion, 'lethal catatonia'.
- 5.11. Professor Goldney stated that if the neurologists had been confident that the cause of death was complex partial status epilepticus, he would have been prepared to accept it. However, he was aware that Dr Hallpike, a Neurologist who also provided opinions for the Court, had not regarded that condition as the cause of death. Professor Goldney stated that Mr McLeod's tragic death reminds us that although we have made some advances in the field of medicine, there are some conditions that just defeat us⁶.
- 5.12. Professor Goldney stated that if one increased the antipsychotic medications one might exacerbate a condition of Neuroleptic Malignant Syndrome because the antipsychotic medications need to be withheld for that condition. But on the other hand, if Mr McLeod had been suffering from catatonia, the antipsychotic medications may have improved his condition. One was therefore on the horns of a dilemma in that for Neuroleptic Malignant Syndrome there was a need to withhold the antipsychotic medication, while on the other hand for catatonia there was a need to increase it.
- 5.13. Professor Goldney was asked whether it would have been prudent to withhold antipsychotic medications immediately following the abnormal EEG report. This would have provided an opportunity to see if the EEG results were attributable to the

⁶ Transcript, Page 232

antipsychotic medication. Professor Goldney's response was that Mr McLeod was improving at that time, and the EEG had been ordered for routine purposes only and not because of any clinical signs of epilepsy. It would not have been a good idea to withhold the antipsychotics at that time in those circumstances as it could have caused a relapse in Mr McLeod's psychiatric disorder.

- 5.14. Professor Goldney stated that there are no specific neuropathological changes from lethal catatonia that would be observed at a post mortem brain examination. He stated that the diffuse hypoxic injuries that were recorded in Mr McLeod's brain were non-specific and did not themselves provide a sound indication of lethal catatonia. He stated that the diffuse hypoxia that was found was because of the fact that Mr McLeod stopped breathing.

6. Dr Jeremy Hallpike

- 6.1. Dr Jeremy Hallpike is Neurologist of 43 years experience. He has a medico-legal consultancy and is the Emeritus Professor of Neurology at the Royal Adelaide Hospital. He has also had much experience in dealing with psychiatric patients.
- 6.2. Dr Hallpike stated that it is perfectly appropriate on the first presentation for major psychiatric illness to refer the patient for an EEG as a screening process. He stated that the yield from such screens can be low, but the benefit high.
- 6.3. Dr Hallpike provided some background in relation to epilepsy conditions. He stated that convulsive epilepticus is the most common manifestation. He stated that generalised convulsive status is the commonest form of epilepsy with probably sixty percent of people falling into that category. He stated that the usual manifestation is generalised seizures with loss of consciousness and convulsions. Successive generalised seizures without intervening periods of consciousness are status epilepticus. This can lead to brain damage and is an urgent medical emergency. The standard treatment is intravenous diazepam.
- 6.4. Dr Hallpike stated that partial seizures are also possible. These are focal seizures where a problem manifests in a particular part of the brain.
- 6.5. He stated that the EEG plays an important role in the diagnosis and classification of epilepsy. However, he stated that where the practitioner sees an abnormal EEG and a

patient with no clinical symptoms of epilepsy, one cannot reach a diagnosis of epilepsy immediately.

- 6.6. Dr Hallpike stated that the EEG report of 20 October 2003 was accurate in describing the result as 'very abnormal'. However, this did not mean that Mr McLeod had status epilepticus because he was, during the test, alert and cooperative. This meant by definition that there was no clinical evidence of epilepsy. The EEG may have been interictal EEG, or in other words, an EEG conducted between seizures. However, it did not necessarily follow that there would be seizures either side of this EEG. Only where such seizures had occurred either side of the EEG could one necessarily diagnose epilepsy.
- 6.7. Dr Hallpike was asked what action should have been taken in view of this abnormal EEG. He stated that he would have regarded it as an unexpected result and would, as the reporting neurologist, have contacted the referring doctor and initiated some form of communication.
- 6.8. Dr Hallpike was asked about Dr Kiley's inclination to arrange, had the matter been left to her, a CT scan, MRI scan, metabolic screen and lumbar puncture investigations at that stage. Dr Hallpike considered it more important to look at the patient clinically from a neurological perspective at that time.
- 6.9. Dr Hallpike stated that the prescription of sodium valproate at that time was a 'weak alternative' to investigating the matter more energetically. In short, he thought that Mr McLeod needed to see a neurologist. He also considered that the neurological investigation had to be carried out before discharge to the Hospital at Home program.
- 6.10. He stated that the Hospital at Home program notes did not show anything that explained the events of 29 to 31 October 2003. He noted that on the second day of the Hospital at Home program, an assessment was done of any potential side-effects of the antipsychotic medication. He stated that these are referred to as extrapyramidal side-effects. He stated that the classic extrapyramidal disease is Parkinson's disease. For that disease, antipsychotic medications are contraindicated.
- 6.11. Dr Hallpike stated that Neuroleptic Malignant Syndrome, catatonia and for that matter Parkinson's disease are all associated with an alteration or imbalance of dopamine levels in the brain. Dopamine is a neurotransmitter. All antipsychotic drugs are

dopamine inhibiting drugs and Parkinson's is a disease where dopamine is in a depleted condition. It is necessary with psychotic patients to inhibit dopamine but with Parkinson's patients to enhance dopamine. Dr Hallpike regards lethal catatonia as 'an obsolescent diagnosis'. He favours the cause of death in Mr McLeod's case as Neuroleptic Malignant Syndrome. He agreed that Neuroleptic Malignant Syndrome is characterised by severe muscular rigidity with a very high CK level from necrosing muscle fibres. He stated that the rate of diagnosis of Neuroleptic Malignant Syndrome is increasing, that people no longer use the term 'lethal catatonia', and that the one has merged into the other. He stated that the basic mechanism in Neuroleptic Malignant Syndrome is dopamine imbalance and that catatonia might be nothing more than the cerebral manifestation of the same thing, but unaccompanied by the muscular rigidity and the heightened CK levels.

- 6.12. Dr Hallpike was critical of the events on admission to the Royal Adelaide Hospital on 29 October 2003. It will be recalled that Dr Aung decided that Mr McLeod should be admitted to the Emergency Department rather than Ward C3. He was in the Emergency Department from approximately 11:00am until 4:00pm for a 'physical examination'. A report in the hospital notes of the Emergency Department record for 29 October 2003 states that the registrar was asked to see Mr McLeod for a physical examination. A Glasgow coma score of 13 out of 15 was recorded. After some time in the Emergency Department he was eventually 'cleared' for transfer to Ward C3. However, the hospital notes show that on readmission to Ward C3 at approximately 4:30pm, Mr McLeod was unresponsive. Further assessment was not possible because of this. The Psychiatric Registrar, Dr Kuruvilla then requested the Neurological Registrar, Dr Leong to attend Ward C3.
- 6.13. Dr Leong, Neurological Registrar, attended to see Mr McLeod at 6:00pm. He noted Mr McLeod to have a Glasgow coma score of 5 out of 15 (it will be recalled that his Glasgow coma score was 13 out of 15 at 11:00am that morning) and Dr Leong instituted a medical emergency team call. Mr McLeod was then admitted to the Intensive Care Unit. He never regained consciousness.
- 6.14. Dr Hallpike was quite critical of the events between 11:00am and 4:00pm that day. He regarded the direction of events as unfocused. He stated that the expression 'medical clearance' or 'physical clearance' is non-specific. As Dr Hallpike noted, Mr McLeod at that time was known to have had an abnormal EEG and to have been

relatively recently introduced to antipsychotic medication. This should have put the clinicians on the alert for Neuroleptic Malignant Syndrome. Dr Hallpike did not regard this as an issue for an intern at all and regarded the unfocused activity in the Emergency Department as a weak point in Mr McLeod's treatment.

- 6.15. Dr Hallpike stated that when Dr Leong saw Mr McLeod at 6:00pm the Glasgow coma score was 5 out of 15. On admission to the Intensive Care Unit, Mr McLeod required ventilation and intubation.
- 6.16. Dr Hallpike stated that the EEG test on 17 October 2003 and the abnormal result it showed could have been explicable as an adverse reaction to the antipsychotic medication Mr McLeod had started on several weeks earlier. It was put to him that the drug dosages were moderate. Dr Hallpike responded that if the reaction was a severe idiosyncratic reaction the fact of a moderate dose is not relevant as such reactions are not dose related.
- 6.17. Dr Hallpike stated that unlike Professor Goldney, after the EEG result was known on 20 October 2003, he would have considered withdrawing the antipsychotic medication to see what effect that had at a further EEG test. It was put to Dr Hallpike that Professor Goldney had expressed the opinion that withdrawal of the medication might have an adverse effect on the psychotic condition of Mr McLeod. Dr Hallpike's response was that he was not suggesting a withdrawal with no monitoring. He was advocating a withdrawal under closely monitored conditions. In any event, no clinician involved in Mr McLeod's treatment on Ward C3 was even considering this as an option at that time, and this was a matter of regret.
- 6.18. Dr Hallpike stated that after that first EEG report he would have been using the EEG procedure 'aggressively' as a diagnostic tool. He would have done this in conjunction with monitoring of the drugs that were or were not being given to Mr McLeod at that time. Dr Hallpike stated that an EEG at the time of Mr McLeod's readmission on 29 October 2003 would likely have been significantly abnormal. It should be noted in this connection that EEGs were in fact performed on 30 October 2003 and 31 October 2003. The first of these showed minimal cerebral activity which may have been attributable to the effect of sedation (at that time Mr McLeod was intubated and sedated on propofol and midazolam). The EEG report for 31 October 2003 was also conducted under the same circumstances of intubation and sedation. It follows that

the opportunity for a useful EEG examination existed between 11:00am and approximately 6:00pm on 29 October 2003.

- 6.19. Dr Hallpike stated that it would have been possible, with a working diagnosis of Neuroleptic Malignant Syndrome, to have instituted at some time after 11:00am on 29 October 2003 the drug bromocriptine in conjunction with re-hydration. Bromocriptine is a dopamine enhancer. Dr Hallpike stated that Mr McLeod's case was not a classical case of Neuroleptic Malignant Syndrome because the CK level was not highly raised (although he considered it was slightly elevated) and there was no increased temperature. In short, Dr Hallpike's view was that he would not rule out the antipsychotic medication in any way as to Mr McLeod's manner of death. He considered that the microscopic and macroscopic brain examination of Dr Grace Scott contradicted the possible theory of death by epilepsy. He considered the effects observed in Mr McLeod over the last twenty-four hours of his life to be extrapyramidal side-effects caused by reaction to psychotropic medication.

7. **Lithium issue**

- 7.1. A curiosity at the Inquest arose from the fact that there is a reference in the hospital notes to a blood test for blood collected at 2350 hours on 29 October 2003 from Mr McLeod showed lithium at the rate of 1.3 millimoles per litre. Such a result, if valid, was regarded by Professor Goldney and Dr Hallpike as highly significant and possibly causative of Mr McLeod's death. A toxicology report of what was referred to as admission serum of blood taken in the Royal Adelaide Hospital (this was taken at 10:00am on 30 October 2003) was the subject of a toxicological analysis by the Forensic Science Centre in November 2003. However, at that time the tests that were conducted did not include screens for lithium.
- 7.2. Accordingly, further tests of those admission serums were conducted by the Forensic Science Centre in March 2006. The March 2006 test results showed the presence of lithium at the rate of 1.36 millimoles per litre. However, at the same time, tests were also conducted of samples of blood taken during the post mortem examination of Mr McLeod (the post mortem date being 3 November 2003). The post mortem sample detected no lithium present.

- 7.3. It should be noted that the admission serums were contained in vials from the Royal Adelaide Hospital, and according to Mr Kostakis, Senior Forensic Scientist at the Forensic Science Centre, the vials containing the admission serums were 'lithium heparin vials'. He stated that most empty vials awaiting blood collection contain some sort of additive, eg. a preservative and/or anticoagulant. He stated that the fact that the vial in which the admission serum was taken and stored was a lithium heparin vial would account for lithium in the blood sample contained within that vial. In other words, the blood sample was contaminated with lithium. Mr Kostakis could not say in evidence at the Inquest that the whole of the amount of lithium detected by the March 2006 testing would be accounted for by the lithium contamination. He stated that the fact that there was no lithium present in the post mortem sample did not necessarily mean that there would not have been lithium present in the blood as at 30 October 2003, the post mortem having taken place some five days later. However, he stated that it was entirely possible that the coating of lithium on the vial entirely accounted for the presence of lithium in the admission blood report for March 2006.
- 7.4. None of this explains the presence of lithium in the blood sample reported in the hospital notes for 2250 hours on 29 October 2003. One possibility is that there was lithium present in the blood at that time. Another possibility is that the vial in which that blood was taken was also a lithium heparin vial, just as the vial in which the bloods taken at 10:00am the following morning were lithium heparin vials.
- 7.5. Each of the medical witnesses involved in Mr McLeod's treatment was specifically asked whether they had prescribed lithium at any stage in Mr McLeod's treatment. None of them had done so. If there was any lithium present in Mr McLeod's body as at 29 October 2003 the evidence at the Inquest did not afford any basis to determine how it came to be present. In my view, the evidence at Inquest was not sufficient to conclude that lithium was in fact present in Mr McLeod's blood on that date. I favour the view that the vial used for the bloods taken at 2350 hours on 29 October 2003 was similar to the vials used by the same hospital the following morning, namely lithium heparin vials. Mr Kostakis confirmed this in his last report to the Court. This provides an explanation for the presence of the lithium in Mr McLeod's body. It follows that lithium was not involved in his cause of death, nor in the circumstances leading up to his death.

- 7.6. Further testing of a sample of blood known not to contain lithium was conducted after storage of the sample in a lithium heparin vial. The testing demonstrated that the lithium levels detected in Mr McLeod's blood could be wholly explained by contamination of the vial.

8. Conclusion

- 8.1. I am left in the position that Professor Goldney's opinion as to cause of death (lethal catatonia) and Dr Hallpike's opinion (Neuroleptic Malignant Syndrome) are apparently different. It may be useful to explore this issue further.

- 8.2. Professor Goldney, in discussing lethal catatonia and Neuroleptic Malignant Syndrome, said 'whether or not you can actually distinguish between the two is very problematic'. He added:

'But more people have suggested that really, perhaps what we are observing as the Neuroleptic Malignant Syndrome may in fact just be lethal catatonia ... Some people have said well, perhaps, with the introduction of the antipsychotic medication, or the neuroleptics, really, we are just giving a different name to what has been known as lethal catatonia since the 19th century.'⁷

- 8.3. Dr Hallpike described the two diagnoses. He said:

'But, the fact is that lethal catatonia doesn't seem to be a diagnostic category that's much recognised now. It was brought in a long time ago. You don't really see much about it. It seems to be obsolescent. Neuroleptic Malignant Syndrome - neuroleptic refers to use of an antipsychotic drug classical or atypical.'⁸

Later he said

'Neuroleptic Malignant Syndrome frequently involves more than purely what one might call cerebral effects ... It can often include severe muscular rigidity with indications that there is some primary muscle component to the condition with a very high serum CK that gets released from recrossing muscle fibres; fever that tends to go with muscle hyper-rigidity.'

- 8.4. Speaking later of the two diagnoses, Dr Hallpike said that the rate of diagnosis of Neuroleptic Malignant Syndrome has been increasing since around 1980, that people are no longer using the expression 'lethal catatonia', and that 'probably the two, or the one, has very substantially merged into the other'. He summarised this with the

⁷ Transcript, Page 228

⁸ Transcript, Pages 267-268

comment that the use of the word catatonia emphasises the cerebral manifestation rather than the muscle rigidity, fever and heightened CK levels.

- 8.5. I took Dr Hallpike to hold the view that a clinician who moves too early to a diagnosis of catatonia may be closing his or her mind to the possibility that the condition is drug induced - a reaction to antipsychotic medication. It was noted that one might generally expect an adverse drug reaction to occur soon after the introduction of the antipsychotic, and not, as in this case, some five weeks later. Dr Hallpike was of the view that this did not necessarily preclude the diagnosis; that an adverse drug reaction might occur after some considerable time.
- 8.6. Dr Hallpike would advocate a preference for a working diagnosis of Neuroleptic Malignant Syndrome rather than catatonia, for the reason that there is a clear explanation for the first which can be treated by withholding the antipsychotic drugs and certain other measures.
- 8.7. Dr Hallpike said that antipsychotic drugs work by depleting the neurotransmitter dopamine⁹. He said that both catatonia and Neuroleptic Malignant Syndrome can be associated with an acute dopamine imbalance. He referred to 'Acute Dopamine Imbalance Syndrome' as a status that would embrace both the possibilities of catatonia on one hand and Neuroleptic Malignant Syndrome on the other. He described the distinction between those two possibilities as 'probably very dubious'¹⁰.
- 8.8. I am persuaded by Dr Hallpike's very scholarly analysis. I am content to describe the cause of death as 'Acute Dopamine Imbalance Syndrome' and I find accordingly.

9. Recommendations

- 9.1. This case shows the need for a close liaison to be established in cases of this kind between the treating psychiatrists and the neurology departments. I recommend, pursuant to Section 25(2) of the Coroners Act 2003, that the Royal Adelaide Hospital re-emphasise the need for full and appropriate interaction between those two disciplines in appropriate cases. All experts in this case were unanimous that there

⁹ Transcript, Page 265

¹⁰ Transcript, Page 277

was a need for a neurological consultation urgently after the EEG report of 20 June 2003. It did not occur.

- 9.2. I recommend, pursuant to Section 25(2) of the Coroners Act 2003, that the Forensic Science Centre should identify, in all toxicology reports, the date and time at which the sample being tested was taken, and that, where in any case that has been the subject of a coronial post-mortem examination, toxicology tests are carried out, a report of those tests be provided to the State Coroner. In this case, certain testing was carried out and I was not even informed of it, let alone advised of the test results. That must not happen again.

Key Words: Death in Custody; Psychiatric/Mental Illness; Acute Dopamine Imbalance Syndrome; Neuroleptic Malignant Syndrome; Abnormal EEG

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

Seal the 25th day of September, 2006.

State Coroner