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 14th and 15th days of March 2007 and the 20th and 29th days of June 2007, by the Coroner's Court of the said State, constituted of Anthony Ernest Schapel, Deputy State Coroner, into the death of Michael Philip Cockburn.

The said Court finds that Michael Philip Cockburn aged 40 years, late of the Glenside Campus of the Royal Adelaide Hospital, 226 Fullarton Road, Eastwood, South Australia died at the Royal Adelaide Hospital, North Terrace, Adelaide, South Australia on the 25th day of December 2002 as a result of respiratory failure due to Adult Respiratory Distress Syndrome (ARDS). The said Court finds that the circumstances of his death were as follows:

1. Introduction and reason for Inquest

1.1. Michael Philip Cockburn was 40 years of age when he died at the Royal Adelaide Hospital ('RAH') on 25 December 2002. He had been admitted to the RAH on 14 December 2002 having been transferred from the Glenside Campus where he was detained pursuant to a continuing detention order issued under Section 13 of the Mental Health Act 1993. The Mental Health Act creates a legislative regime that provides for the detention of persons who have a mental illness and who should be detained in the interests of their own health and safety and/or for the protection of others. The Section 13 detention order, if lawfully imposed, was still in effect as of the date of the deceased's death. This would mean that it was a death in custody in respect of which an Inquest would be mandatory. A question has arisen as to whether

in the particular circumstances of this case the detention order had been lawfully imposed. I deal with the lawfulness of the deceased's detention below.

1.2. Mr Cockburn suffered from what had been diagnosed many years before his death as a deep seated schizophrenic illness, a disorder characterised by the suffering of delusions. Complicating the matter was the fact that he had a well understood and well documented propensity to react adversely to antipsychotic medication. He had suffered dangerous side effects from some such medications, including breathing difficulties.

1.3. The deceased's psychiatric disorder had been long standing, existing since his early adulthood. His medical history makes it plain that he had been an energetic and resourceful young man before he was struck down by this debilitating illness. Mr Cockburn is described as having been a very pleasant and jovial fellow when well. By 2002 the deceased was to be described as a completely insightless and frequently very psychotic and violent man when otherwise. The absence of suitable medications for his psychosis made treating him very difficult. He became more and more suspicious and distrusting of Glenside staff and he had a history of violence towards members, even those with whom he had developed positive relationships. The deceased was regarded as unpredictable and dangerous. At the time of his death he was 187 centimetres in height and weighed 145 kilograms.

1.4. The deceased also suffered from other physical complaints including chronic obstructive airways disease and a chronic suspected gastro-intestinal bleed with associated acute episodes of anaemia. He had also been hospitalised earlier in December 2002 for a suspected deep vein thrombosis and pulmonary embolism. The post mortem examination of the deceased revealed previously undiagnosed heart disease.

1.5. On 14 December 2002, the deceased was admitted to the RAH because those responsible for his medical treatment at Glenside had noticed a significant drop in his haemoglobin level and that he was anaemic. It was suspected that this was reflective of blood loss through gastrointestinal bleeding. It was determined that this blood loss would probably necessitate a blood transfusion and that this could properly be facilitated at the RAH. I pause here to observe that this was not the first occasion the deceased's haemoglobin level had been compromised and where medical intervention had been considered necessary for that reason.

1.6. The difficulty of dealing with the deceased manifested itself on the evening of 14 December 2002 when the attempt was made to administer the blood transfusion at the RAH. He became volatile and agitated and refused to take oral sedative medication. In the event, he had to be restrained and antipsychotic medication including the drug Haloperidol, which was said to have caused the deceased to experience severe and dangerous side effects in the past, was administered by intra muscular (IM) injection. Not long after its administration, the deceased suffered a respiratory collapse from which he never fully recovered and he died on 25 December 2002. At autopsy, the deceased was found to have died from respiratory failure due to Adult Respiratory Distress Syndrome (ARDS). I have found this to be the cause of death and I return to the detail of this later. An issue for investigation is whether the medication that he was given on the first day of admission had been appropriately given, especially in the light of his documented resistance to antipsychotic medication, and whether this may have triggered his collapse or played a role in the causation of his death.

1.7. An Inquest to investigate those issues was clearly indicated, but quite apart from that, an Inquest into the cause and circumstances of a death in custody is mandatory.

1.8. Mr Cockburn remained in the RAH between 14 December 2002 and 25 December 2002, the day of his death. At all material times in that period the deceased remained under the detention purportedly imposed pursuant to the provisions of the Mental Health Act. 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.

1.9. The definition of a death in custody contained within Section 3 of the Coroners Act 2003 is as follows:

> ‘means the death of a person where there is reason to believe that the death occurred, or the cause of death, or a possible cause of death, arose, or may have arisen, while the person—
>
> (a) was being detained in any place within the State under any Act or law, including any Act or law providing for home detention (and, for the purposes of this paragraph, a detainee who is absent from the place of his or her detention but is in

the custody of an escort will be regarded as being in detention, but not otherwise); or

(b) was in the process of being apprehended or was being held—

(i) at any place (whether within or outside the State)—by a person authorised to do so under any Act or law of the State; or

(ii) at any place within the State—by a person authorised to do so under the law of any other jurisdiction; or

(c) was evading apprehension by a person referred to in paragraph (b); or

(d) was escaping or attempting to escape from any place or person referred to in paragraph (a) or (b)'

It will be seen that this definition includes a death where there is reason to believe that it has occurred, or the cause of death, or a possible cause of death, arose, or may have arisen, while the person was in any of the listed custodial situations. I do not need to decide here whether as a matter of law this provision in the strict sense only applies to custody that has been lawfully imposed, although the point can be made that it would be extraordinary if the coroner's scrutiny could be avoided if the custody that the person died in was, although ostensibly lawful, in fact unlawful. Be that as it may, the provision clearly bites where there is reason to believe that the death occurred in any of the listed custodial situations and also where there is reason to believe that the cause of death may have arisen in any of those circumstances. Whether the Section 13 order in this case was lawfully imposed or not, for reasons that will become obvious this was clearly a death where at the very least there was reason to believe that it occurred, or its cause may have arisen, while the deceased was in lawful detention as contemplated by the definition of death in custody. Accordingly, an Inquest to ascertain the cause or circumstances of Mr Cockburn's death was mandatory by virtue of Section 21(1) of the Coroners Act 2003.

2. <u>The lawfulness of Mr Cockburn's detention and treatment at the RAH</u>

2.1. The lawfulness or otherwise of the deceased's detention on 14 December 2002 and in the period between that date and his death has been questioned by the member of SAPOL who investigated this death on behalf of the State Coroner. I refer here to Detective Seneca's analysis of the matter in his statement, verified by affidavit, Exhibits C7 and C7a at pages 27-30.

2.2. Detective Seneca very properly raises an issue as to whether the continuing detention order of the Guardianship Board made on 21 August 2002, from which the lawfulness of the deceased's detention and treatment at the RAH on 14 December 2002 and following is said to derive, was a valid order or not.

2.3. In my view, the lawfulness or otherwise of the deceased's detention and treatment under compulsion during that detention is one of the circumstances surrounding his death and is therefore the proper subject of inquiry in this Inquest.

2.4. The relevant chronology is as follows:

a) On 2 July 2002, Drs Kent and Beckwith at the RAH made a detention order pursuant to Section 12(6) of the Mental Health Act 2003 (the Act). The maximum duration of such an order by law is 21 days. The duration of the detention is not specified in their order and it is postulated that this was a defect that rendered the order, and the detention that it purported to impose, null and void

b) On 17 July 2002, Dr O'Moore at Glenside makes a detention order pursuant to Section 12(1) of the Act. By virtue of that provision, this order will expire 3 days after it was made if not revoked earlier. This order is made in the belief that the order of Drs Kent and Beckwith made on 2 July 2002 was null and void because of its failure to specify the duration of the detention. If the order of 2 July was valid in spite of the identified defect, the prohibition in Section 12(7) of the Act would come into play. Section 12(7) prohibits the making of an order under Section 12(1), such as Dr O'Moore's order, if a patient is already being detained pursuant to an order under Section 12(6), such as Dr Kent and Beckwith's order.

c) On 18 July 2002, Dr Narielvala confirms Dr O'Moore's detention order of the day before. This confirmation is made pursuant to Section 12(4) of the Act which requires Dr O'Moore's order to be confirmed within 24 hours of admission to the approved treatment centre.

d) On 20 July 2002, Dr Asokar makes a first 21 day detention order pursuant to Section 12(5) of the Act. This is effected because Dr O'Moore's 3 day order of 17 July 2002 is about to expire.

e) On 10 August 2002, Drs Litt and Hustig impose a second 21 day detention order pursuant to Section 12(6) of the Act.

f) On 21 August 2002, the Guardianship Board makes its continuing detention order pursuant to Section 13 of the Act. It is this order that is said to be in existence at

the time of the deceased's death and which is said to have formed the lawful basis for the deceased's detention and treatment between 14 December 2002 and 25 December 2002, the day of his death. The jurisdiction of the Board to make this order is based upon the lawfulness of the series of orders commencing with that of Dr O'Moore of 17 July 2002 and upon the lawfulness of the detention imposed pursuant to those orders.

2.5. The difficulty with all of the above arises from a perception that the orders commencing with that of Dr O'Moore of 17 July 2002 may have been invalid because of the existence of the order of Drs Kent and Beckwith of 2 July and the operation of the prohibition in Section 12(7) of the Act. In my opinion, this perception is incorrect.

2.6. The Guardianship Board's continuing detention order purports to have been made pursuant to Section 13 of the Mental Health Act 1993 (the Act). Section 13(1)(a) of the Act makes it plain that the Guardianship Board may lawfully make a continuing detention order only where the person who is the subject of the order is already being lawfully detained pursuant to the Act. Previous decisions of the District Court Administrative Appeals Tribunal express the same view - **Romancuks v Guardianship Board** DCAAT-98-134; **P v Guardianship Board** DCAAT-99-135 [1999] SADC 97. Unless the already existing detention was lawful, the Guardianship Board has no power to impose the continuing detention order pursuant to Section 13. It is both the fact of the already existing detention and its lawfulness that enlivens the jurisdiction of the Board. If the Board had no jurisdiction to impose the continuing detention order, a nice question arises as to whether that order was void right from the beginning. If it was so void, the deceased's detention was unlawful. If the order, although lacking in jurisdiction, was not to be regarded as void from the beginning, it seems to me that the detention would have been lawful unless the detention order was quashed, which it never was. In the event, I do not need to decide whether the Board's order was void from the beginning because in my opinion it was valid in any case because the deceased was already being lawfully detained when the Board's continuing detention order was made. In my opinion, the previous detention orders pursuant to Section 12 of the Act, made with a view to remedying the Kent/Beckwith order, were all valid. They were all valid orders because in my view the earlier order of Drs Kent and Beckwith was patently null and void because of its failure to specify the duration of the detention. Drs Kent and Beckwith had to consider two distinct

matters. Firstly they had to consider whether detention was appropriate, and secondly they had to consider the duration of the detention, given that the 21 days is a maximum. The order bears no evidence of the consideration of the second question, and in particular, whether the purposes of the detention could be achieved within a period of detention shorter than the maximum 21 days. Thus the Section 12(7) prohibition referred to above is irrelevant. Dr Kent and Beckwith's order was null and void, the orders commencing with that of Dr O'Moore of 2 July were valid as was the order of the Board of 21 August 2002.

2.7. It is to be observed that the Board's continuing detention order contained a treatment order that enabled treatment to be given to the deceased for both mental illness and any other illness notwithstanding the absence or refusal of consent to the treatment. Therefore in my view both the detention and treatment of the deceased between 14 December 2002 and 25 December 2002 was authorised by the Board's order and was lawful.

2.8. I add here that there is no suggestion that any of the orders made under the Act were not made in good faith or not appropriately made on their merits. In addition, there is no suggestion that any of the deceased's psychiatric or medical treatment was not administered in good faith.

3. The deceased's psychiatric medical history

3.1. No less than twelve volumes of clinical records from Glenside exist in relation to the deceased. They date back to 1984 when the deceased was first diagnosed with schizophrenia. It is said that the deceased's psychosis was treatment resistive. This psychosis continued to exist and manifest itself in the deceased's behaviour right up to the time of his death. He experienced florid psychotic delusions and was the subject of a number of detention orders under the Mental Health Act.

3.2. I have already referred to the type of behaviour that the deceased was capable of.

3.3. Throughout the course of the deceased's treatment at Glenside, he was from time to time placed on antipsychotic and sedative medication.

3.4. The administration of Haloperidol, one of the antipsychotic drugs that the deceased would be given on 14 December 2002, is recorded as having taken place on a number of occasions in 1984. There are many references in the Glenside clinical records

relating to this man's adverse reaction to antipsychotic medication in general and to identified antipsychotic drugs in particular. It can be seen that on the deceased's first admission to Glenside in January and February 1984 there was such an adverse reaction. On 9 February of that year, the deceased was placed on a regime of Haloperidol medication, namely 20mg, three times daily. Prior to that date, he had been administered other drugs. His medication chart for this admission reveals that from 9 February onwards, Haloperidol was the sole antipsychotic medication that he was being administered. On 9 February at 1pm and 5pm (possibly 6pm)[1] he was given Haloperidol. It is recorded that he was also given Benztropine at 3pm and 5pm for a dystonic reaction. On the night of 17 February 1984 it is documented that the deceased complained of neck muscle rigidity, a well recognised dystonic reaction to Haloperidol. He had taken that drug at 8am, 1pm and at either 5pm or 6pm on that day. In order to combat this side effect, 2mg of Benztropine was administered to the deceased and the situation was resolved. The deceased was discharged on 20 February 1984. I infer from the above that the deceased had suffered a dystonic reaction from Haloperidol during the course of this admission. Neck muscle rigidity as a dystonic reaction to antipsychotic medication can be associated with laryngeal spasm which can cause breathing difficulties. However, there is no evidence of laryngeal spasm on these occasions and the dystonic reactions can be considered mild.

3.5. I do not understand there to be any further evidence of an adverse reaction to Haloperidol in the course of the deceased's history. Although there are references in documentation such as discharge summaries which suggest that the deceased had demonstrated an aversion to that drug as well as to others, specific documented instances of the administration of Haloperidol with adverse consequences between the deceased's first admission in 1984 and 14 December 2002, the occasion of his collapse, are not readily apparent from the clinical records.

3.6. However, it is important to observe that as well as the adverse reaction that was recorded in respect of Haloperidol in February 1984, there were a number of recorded instances of adverse reactions to other antipsychotic medication, from which an inference is available that the deceased had an aversion to this type of medication in general. I mention some examples of this to illustrate the point.

[1] Clinical record indecipherable

Occasion	Medication where known	Adverse reaction
April 1984	Fluphenazine	Dystonic
April 1985	Fluphenazine	Severe laryngeal spasm with occasional apnoeas and loss of bowel control
April 1985	Fluphenazine	Muscle dystonia and laryngeal spasm
April 1985	Fluphenazine	Truncal and neck dystonia
April 1985	Fluphenazine	Acute dystonic attack
April 1985	Fluphenazine	Recurrence of laryngeal spasm with severe dyspnoea and inability to take a breath
April 1985	Pimozide	Severe laryngeal spasm followed by observations at the QEH
August 1993	Remoxipride	Symptoms of dystonic reaction including laryngeal spasm

3.7. In addition to the above, it was also recorded that the deceased suffered facial oedema as an adverse reaction to Chlorpromazine, an antipsychotic medication. It is also said that the deceased was at risk of pulmonary embolus from Chlorpromazine.

3.8. It was also recorded in the deceased's clinical record that he had been treated with a drug called Thioridazine, apparently without any adverse consequence, but in any case with little beneficial effect.

3.9. The fact that the deceased's intolerance to antipsychotic medications is well documented throughout the entirety of his long history leaves no doubt in my mind that those responsible for his treatment at Glenside well understood the risks involved in administering any of the following antipsychotic medication to the deceased, namely Haloperidol, Fluphenazine, Pimozide and Remoxipride. A case summary compiled on 14 August 2002, four months before the deceased's collapse, refers to the 1985 episodes of severe dystonic reaction, laryngeal spasm, oculogyric crisis, loss of bowel and bladder control with oral Fluphenazine, dystonic reaction and laryngeal spasm on Haloperidol, and relatively poor response to Thioridazine. The summary also refers to the 1993 experience with Remoxipride which had been unsuccessful and which had also elicited a dystonic reaction. The document also referred in general terms to a number of drugs that were said to have elicited '*severe at times life threatening side effects*'. They were Fluphenazine, Primozide, Haloperidol, Remoxipride, Chlorpromazine and Clozapine.

3.10. The statement verified by affidavit of Dr Andrew Short[2], a trainee in psychiatry at Glenside at the time of the deceased's death, and now a specialist in psychiatry, makes it clear that the deceased's multiple sensitivity to antipsychotic medication was notorious and that the consequent problems in treating his disorder were well understood. Dr Short states that the options for treating psychotic illness when antipsychotic medication cannot be used are very limited. Almost every medication that would normally be considered in the treatment of the deceased's disorder were not available to him because of his sensitivities to that medication. Even the medications that had not been tried in his treatment were to be ruled out because of probable adverse reaction to those as well. Consideration was also given to using the drug Clozapine. The use of this drug caused those treating the deceased some anxiety because of the possible complications from its use, but it was considered beneficial because of its well known property to decrease aggression.

3.11. I heard evidence about the relationship of individual antipsychotic drugs to each other. Dr Gilbert, who performed the post mortem examination with respect to Mr Cockburn, said that Haloperidol and Fluphenazine were closely related drugs and would be expected to have a similar pattern of side effects with respect to dystonic reactions. So that if the deceased had a documented history of dystonic reactions to Fluphenazine, which he did, then one would reasonably expect him to show a similar propensity to dystonic reactions with Haloperidol. Professor Goldney, a psychiatrist who gave expert evidence, was to say something quite similar. These observations would lead one to conclude that Dr Short was not overstating the position when he said that even untried psychotic medication could be problematic to the extent that they would not be an option. However, a demonstrated propensity to exhibit a dystonic reaction to an antipsychotic drug does not necessarily imply an inevitability that on each and every occasion of its administration that such a reaction will occur. Professor Goldney told me it was wrong to view these reactions as allergic, where there is a stimulus and an almost inevitable reaction. Rather, even in a situation where there has been a recognised propensity in a person to react adversely to a particular antipsychotic medication, there may or may not be an adverse reaction on a given occasion. However, it is also pertinent to observe that antipsychotic medication such as Haloperidol will usually have a sedative effect on the patient. A sedative effect may have some impact on a patient's respiratory capabilities.

[2] Exhibits C5 and C5a

3.12. At the time of the deceased's admission to the RAH on 14 December 2002, he was on a prescription for Clozapine and other sedating and/or mood stabilising medications, as well as a recently released antipsychotic called Amisulpride.

3.13. A number of documents had been created at Glenside in relation to the deceased's history of adverse reaction to antipsychotic medication. I am here referring in particular to pages 1-3 of Volume 12 of the Glenside clinical records[3]. These documents were clear in their terms and, as will be seen, were made available to medical staff at the RAH upon the deceased's presentation on 14 December 2002. The first document at page 3, the date of which is unclear, and which is headed '*ATTENTION*', recommends the discontinuance of '*all antipsychotics stat*'. The term 'stat' refers to the administration of a drug on an ad hoc, possibly once off basis. It is in contradistinction to the administration of drugs '*PRN*' which refers to regular or intermittent administration. The document also refers to the drugs Fluphenazine and Pimozide as having given rise to severe dystonic reactions including episodes of laryngeal spasm and cyanosis and identifies particular episodes of adverse reaction to each drug in April 1985. Although the document does not refer to Haloperidol as having caused an adverse reaction, it does, as seen, recommend the discontinuance of all antipsychotics of which Haloperidol is one. The other relevant recommendation contained within the document states that any institution, which would obviously be taken as re-institution, of antipsychotic agents should be gradual and that Cogentin (which is Benztropine) should be administered at the commencement of any such therapy. Benztropine is a drug designed to ameliorate adverse side effects of antipsychotic medication. It had been used to good effect during Mr Cockburn's first admission in 1984. The second document, undated but seemingly compiled after 4 February 2002, is also headed '*ATTENTION*'. It refers to adverse reactions to Fluphenazine, Pimozide, Risperidone and Remoxipride, facial oedema and rash with Chlorpromazine and pulmonary embolus with Clozapine. The document states:

> '4 Treatment with conventional antipsychotics and <u>ALL</u> of the abovementioned antipsychotics should be avoided at all times.'[4] *(the word <u>ALL</u> is in capitals and is underlined in the original)*

This document also recommends the use of Cogentin (Benztropine) in conjunction with the institution of any other antipsychotic agent. The third document brought into existence and dated 17 June 2002 is a proforma South Australian Mental Health

[3] Exhibit C7d
[4] Exhibit C7d, Volume 12, Page 2

Service document which states - '*This form is to be used when a client has a condition that is potentially dangerous to the client…*'[5] and contains space for the listing of allergies (including allergic drug reactions), sensitivities, serious infectious diseases and contra-indications (to the administration of drugs). The document, under the heading '1 *ALLERGY*' states:

> '* Note: severe dystonic reactions to antipsychotic medications, including episodes of laryngeal spasm and cyanosis
>
> * Fluphenazine, Pimozide, Risperidone, Remoxipride
>
> * Chlorpromazine (facial oedema & rash) Haloperidol
>
> * Pulmonary embolus – Clozapine.'[6]

3.14. These three documents travelled with the deceased to the RAH on 14 December 2002. It does not appear that any of the three documents were present in the RAH clinical record at the time of an earlier admission at the RAH in June 2002. However, it appears that on that earlier occasion the deceased's aversion to antipsychotic medication had been recognised. A Dr Beckwith has noted in the clinical record of 17 June 2002 that the deceased:

> '… cannot have IM antipsychotics (except Olanzapine for which there are no stocks in Australia yet)' [7] (IM means intra-muscular)

In addition, the deceased's PRN medication chart for that admission at the RAH lists a number of drugs, including Fluphenazine and Haloperidol as those that might give rise to 'Adverse drug reactions', with the added comment that such reactions could be '*Severe ++ Life threatening*'.

3.15. Of the documented adverse reactions to antipsychotic medication, laryngeal spasm (or laryngospasm) is the most relevant as far as the issues in this Inquest is concerned. There is at least a suspicion that a laryngeal spasm was a precipitating factor in the collapse of the deceased on 14 December 2002. A laryngeal spasm involves the vocal chords coming together involuntarily with the result that the airway becomes blocked. If not reversed, the obvious consequences, such as a respiratory and cardiac arrest, will likely occur. Drugs such as Benztropine and Diazepam (Valium) were known to alleviate the condition in the deceased's case.

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[5] Exhibit C7d, Volume 12, Page 1
[6] Exhibit C7d, Volume 12, Page 1
[7] Exhibit C7d, Volume 11, Page 135

4. The deceased's relevant physical medical history

4.1. The deceased suffered from a number of physical deficits. He suffered from chronic obstructive airways disease (COAD) and was morbidly obese. I have already referred to his weight of 145 kilograms.

4.2. As seen, the deceased had been hospitalised at the RAH in June 2002 for suspected gastro-intestinal bleeding giving rise to anaemia. He was given a number of units of blood. This condition was said to have existed chronically, and was also the reason he was admitted on 14 December of that year. The site of this bleeding was never identified, even at post-mortem.

4.3. The deceased also had a history of the development of deep vein thrombosis and associated pulmonary embolus, a life-threatening condition.

4.4. Although undiagnosed at the time of his death, the deceased also suffered serious cardio vascular difficulties revealed only at post-mortem examination. I return to this issue later and in particular to its possible impact on the cause of death.

4.5. Much of the deceased's relevant physical history is described in the statement verified by affidavit of Dr Paul Drysdale, a Consultant Physician at the RAH. Dr Drysdale recounts that the deceased had suffered from anaemia and iron deficiency in the past, the cause of which was suspected as being chronic blood loss but which had been investigated without success on a number of occasions. Acute episodes of anaemia had been treated with blood transfusions.

4.6. Dr Drysdale also reports that the deceased had what he describes as '*very precarious lung function*'. He describes his normal lung function as poor due to smoking related disease and that in such a context, respiratory function often does not recover. The deceased's chronic obstructive airway disease was long standing and he had a history of previous admissions for respiratory failure.

4.7. Dr Drysdale states that the deceased had been admitted to the RAH with a respiratory deterioration earlier in December 2002. On this occasion investigations were conducted regarding a possible DVT (or clot) in his leg. A significant thrombus was excluded, but a minor clot was located in the gastrocnemius vein. The deceased was also anaemic and in respiratory failure, both of which were chronic conditions. On

this occasion, the deceased had refused inhalers and had also refused a blood transfusion.

4.8. Dr Drysdale did not have any role to play in the events of 14 December 2002. Nevertheless he suggests that the medication administered to the deceased could have triggered laryngeal spasm, although he notes that the intensive care doctors had placed a tube down into his trachea which would have enabled him to breathe. He states that laryngeal spasms could have contributed to the deceased's respiratory arrest. Dr Drysdale suggests that it would have been better if the deceased had not been given the medication that he was given on 14 December 2002, but remains unconvinced that this solely compromised the deceased's respiratory condition.

5. The events of 14 December 2002

5.1. The events at Glenside, before the deceased was transferred to the RAH, are described in the statement of Dr Short to whom I have already referred. Dr Short was the on-call duty doctor at Glenside when, at about 9:25am, he was asked to see the deceased. The deceased's haemoglobin level had dropped significantly. He was anaemic, dizzy, short of breath, confused and belligerent. Dr Short formed the view that the deceased needed a medical review at the RAH and a blood transfusion.

5.2. Dr Short prepared a handwritten letter to the RAH Emergency Department and this, together with other documentation relative to the deceased, travelled with him to the RAH. The letter referred to the deceased's history of violent behaviour in the past and warned that he should be treated with caution. The letter was attached to, and made reference to, what was believed to be the deceased's latest drug chart at Glenside and requested the recipient of the letter to note his '*multiple allergies*'. The letter was addressed to the Emergency Department Doctor. In addition to the drug chart, a number of other documents accompanied Dr Short's letter. They were the three documents that are described in paragraph 3.12 above and which, inter alia, collectively warned of the potential pitfalls of the deceased being administered antipsychotics and which recommended the discontinuance of all antipsychotics stat.

5.3. The drug chart that was included in this bundle of documentation from Glenside did not relate to the deceased at all. It was the drug chart of a patient by the name of Nancarrow. This chart described among other things a twice-daily regime of the administration of Clozapine, an antipsychotic that was to be given at 8am at a dosage of 100mg and at 9pm at a dosage of 400mg. As it happened, Mr Cockburn himself

was on a daily regime of that drug, but at a dosage of 175mg morning and night. It is recorded that he had received a dosage of 175mg at Glenside that morning. That dosage was of course in accordance with his own regime, but because of the mix up with Nancarrow's chart, he was given a further 400mg of Clozapine at 9pm at the RAH. This was 225mg in excess of his normal nightly dosage. The Clozapine regime for the deceased had only been recently reintroduced and was being gradually increased. It had been thought that the drug may have had a propensity to cause pulmonary embolus in the deceased.

5.4. On 14 December 2002, the deceased was taken from Glenside to the Emergency Department of the RAH where he was seen shortly after 11am by a Dr Tadros. The Nursing Assessment form compiled at about that time records in handwriting the words '*some psychotropic drugs*' in a section intended to record his allergies. Psychotropic is presumably intended to mean antipsychotic.

5.5. When the deceased came to be admitted, he was seen by a Dr Nicholas Kasmeridis who was one of two admitting medical registrars on duty that day. Dr Kasmeridis saw the deceased just before he was admitted at about 3pm. It is obvious that Dr Kasmeridis had access to the documentation that had travelled with the deceased from Glenside.

5.6. It is clear on the evidence that a PRN drug chart was prepared by Dr Kasmeridis for the deceased's admission which included two significant entries that in the main were distillations of the written information that had arrived at the RAH that day with Mr Cockburn. The PRN chart perpetuated the erroneous information about the Clozapine regime that was thought to relate to the deceased but which in fact related to the patient Nancarrow. Consequently, the PRN chart recorded a regime of 100mg in the morning and 400mg at night for the deceased. In a section of the chart marked *'Adverse drug reactions'*, the following drugs are included, namely Fluphenazine, Pimozide, Risperidone, Remoxipride, Chlorpromazine and Haloperidol. This information is taken seriatim from one of the documents that accompanied the deceased to the RAH, namely the document that I have already referred to in paragraph 3.13. herein, ie. the document headed '1 *ALLERGY'*.

5.7. The PRN chart and the documentation that had arrived with the deceased from Glenside I find was sent to Ward Q8, the ward at the RAH to which the deceased was admitted. I heard a lot of evidence as to the method by which this documentation was

transmitted and kept within the ward. I do not need to go into the fine detail of that because there was no dispute that the documentation was available to nursing staff on the ward and to medical staff. This availability is illustrated by the fact that the PRN chart was acted upon in the ward, and in particular when the erroneous dose of the 400mg of Clozapine was administered at 9pm, as well as Valproate syrup and 2mg of Clonazepam. It will be remembered that this chart listed the six drugs to which the deceased was said to have exhibited adverse reactions.

5.8. The blood transfusion commenced some time during the evening. The circumstances leading up to the administration of the deceased's antipsychotic medication are described by a registered nurse, Ms Rosslyn Stafford, who gave evidence before me. Her notes of the incident, as contained in the RAH clinical record, appear to provide a better record of these events than her unaided memory. She describes that at about 10:10pm while he was being given his second unit of blood he became verbally aggressive and demanded to be taken outside for a cigarette. He threatened to remove the jelco and made threatening gestures with the drip stand. Urgent restraint was called for. An intern by the name of Dr John Youssef-Boghdadi (Dr Boghdadi), who also gave evidence before me, came to the ward and ordered oral sedative medication for the deceased. The order was for 5mg of Olanzapine and 1mg of Lorazepam. This was a conservative and appropriate approach and could not possibly attract criticism. When staff attempted to give this to him, it is recorded that '*he flatly refused to accept medication, swearing and utilising inappropriate words*'[8]. Urgent restraint was again called for and the deceased was shackled by four security guards. It was common ground among those who witnessed these events that the deceased was quite intimidating. Dr Boghdadi was recalled to the ward. He described the deceased as very agitated and he was conscious of the fact that he was such a big man. The security guards had hold of him in the corridor and he was being violent. He had to be physically returned to his room. Dr Boghdadi told me that he had felt threatened by the situation and was a little scared as he was inexperienced, which I took to mean inexperienced in such an adverse clinical setting. Without consultation with any other practitioner, he prescribed two medications, namely Haloperidol at the maximum dosage of 10mg and Clonazepam at 2mg. Nurse Stafford administered this by IM injection at 10:45pm. Gradual relaxation in the deceased was then observed and the transfusion was recommenced at 10:50pm. Nurse Stafford's final note about the

[8] Exhibit C7c, Volume 3, Page 29

deceased that evening was that he was '*currently lying quietly in bed*'. Nurse Stafford's shift concluded officially at 11pm.

5.9. Neither Nurse Stafford nor Dr Boghdadi had any previous dealings with the deceased. In spite of the significant amount of written material that existed suggesting that the deceased should not be given medication of the kind he was given, neither claims to have known anything about that.

5.10. By 11:50pm another nursing shift had commenced. At about that time Registered Nurse Kristie Head was in the deceased's room making a routine check. What took place there at that time is described in two statements made by her which have been tendered before me - a statement verified by affidavit being Exhibits C6 and C6a and a further statement verified by affidavit being Exhibit C6b. In addition, there is a nursing note compiled by Nurse Head at 12:10am on 15 December 2002 that also describes what occurred at that time.

5.11. Nurse Head's second statement on oath, taken over two years after these events, unsurprisingly makes it plain that her recall of detail, especially in relation to an interpretation of what is contained in her notes, is not good. I did not see the need to trouble Nurse Head to give oral evidence. It is as well to set out her notes in their entirety:

> '33# called 2351 hrs as p^t going "blue" and gasping for air, Guedel inserted + EAR started · / bag + mask. P^t recently had IM Clonazepam + Haloperidol 2245 by late duty staff for severe agitation. See Med Reg / ICU note for further details. CNC in attendance.' [9]

The reference to 33# is to an emergency pager number. The abbreviation EAR is expired air resuscitation. P^t means patient. The abbreviation CNC is Clinical Nurse Consultant.

5.12. In Nurse Head's first statement, taken on 2 March 2005, she says that when she entered the deceased's room he still seemed agitated and was moving around in the bed. He did not say much, his eyes were open and he was looking around. She states that she knew that there was '*something not quite right with him*'[10]. She says that she had been in the room for about 5 to 10 minutes when she noticed that he had stopped breathing. He went blue in front of her. I do not know whether her nursing note on

[9] Exhibit C7c, Volume 3, page 30
[10] Exhibit C6a, Page 3

the RAH file was made available to Nurse Head when she made this statement, but it makes no mention of the '*gasping for air*' recorded in that note. Nurse Head's more recent statement taken in March of this year was taken to ascertain whether she could provide any more detail about the deceased's presentation, particularly in light of what was recorded in her note. His presentation at that time, as will be seen, is relevant to what may have triggered his respiratory collapse. In her recent statement Nurse Head states that she had not noticed anything unusual about the deceased that she did not attribute to the fact that he had been quite agitated earlier. She knew this from the handover. However, when asked to explain what she had meant in her first statement when she had said that there was something not quite right with the deceased, she was unable to elaborate. She did say, however, that the reference in her note to gasping for air meant that she had observed that his breathing had changed from apparently being able to breathe normally to '*attempting to take breaths but appearing to be struggling to get air*'[11]. She did not recall whether he made an audible noise in association with his breathing difficulty. This statement very much suggests that the deceased was awake and conscious before his breathing difficulty manifested itself because she refers to him looking around the room at her and other objects such as the pictures on the wall.

5.13. Nurse Head says that she called for an emergency resuscitation team which duly arrived. She had inserted a Guedel airway in the deceased's mouth. This is a device designed to prevent the tongue from blocking the air passage. In her more recent statement Nurse Head says that she does not recall having problems when she inserted the airway. The insertion of a Guedel airway is to be distinguished from intubation of a patient which is a different and more difficult procedure.

5.14. A resuscitation team arrived. Dr Stephen Lam, then a medical registrar, was in charge. The deceased was in both cardio and respiratory arrest and Dr Lam had noted cyanosis, that is blueness due to lack of oxygen in the tissues, and '*agonal respirations*'. Dr Lam, now a consultant at the Flinders Medical Centre, gave evidence in the Inquest. He did not have any independent recollection of these events. However, he was shown his note of the incident and was asked to interpret what he had written. He has noted that he was called at 2351 hours that evening. He surmised that his notation that the deceased was suffering from agonal respirations was from what he was told on his arrival at the ward because his own observations, as recorded

[11] Exhibit C6b, Page 2

in his notes, were that the deceased had no pulse and that he was cyanotic. Resuscitative measures including intubation and the administration of adrenalin and atropine restored the deceased's circulation. One of the more significant features of Dr Lam's involvement was that he was able to intubate the deceased by placing an endotracheal tube into the deceased's airway and was able to do so with ease. Dr Lam in evidence told me that this was an indication that at the time he intubated the deceased there was no evidence of laryngeal spasm that might have blocked or significantly restricted the deceased's airway. However, Dr Lam conceded the possibility that before his arrival there may have been a laryngeal spasm that had already resolved. Following the deceased's resuscitation he was taken from the ward into the Intensive Care Unit for further observation and treatment.

6. The deceased's treatment between 14 December 2002 and the day of his death

6.1. Dr Drysdale, to whom I have earlier referred, indicates in his statement that following the deceased's resuscitation and transfer to the Intensive Care Unit he continued to have respiratory difficulties despite intubation and ventilation. Further investigation indicated that the deceased was still anaemic and remained in respiratory failure. Dr Drysdale suggests that the deceased never improved and succumbed ten days later. Dr Drysdale also makes an observation that the deceased did not regain consciousness at any time after being resuscitated. However, the clinical record would suggest that in the early days of Mr Cockburn's time in Intensive Care there was a level of consciousness as reflected by entries describing the deceased from time to time as being '*combative and agitated*', '*aggressive and unmanageable*'[12], awake and obeying commands and smiling at his mother and sister. There is also a reference on 16 December 2002 to the deceased refusing investigations for anaemia. Be that as it may, there was a clear and well documented deterioration in his respiratory condition.

6.2. There is a reference in the clinical record on 20 December 2002 to a diagnosis of ARDS and again on 21 December 2002. References to episodes of consciousness as far as the deceased is concerned end on 18 December 2002.

6.3. There are also references in the clinical record to the possibility of the deceased suffering from a pulmonary embolus.

[12] Exhibit C7c, Volume 3

6.4. A notation of 24 December 2002 refers to '*severe ARDS*[13]' and a '*combined image of septic shock and pulmonary embolism*'[14]. It is recorded that the deceased's eyes were shut and that there was no response to voice on that day. Mr Cockburn was pronounced deceased at 1:45am on 25 December 2002.

7. Cause of death

7.1. A post-mortem examination that included an autopsy was conducted by Dr John Gilbert, a Forensic Pathologist. I received in evidence Dr Gilbert's post-mortem report[15]. As well, Dr Gilbert gave evidence before me.

7.2. Dr Gilbert states the cause of death as respiratory failure due to ARDS (adult respiratory distress syndrome). In his report Dr Gilbert refers to the clinical history between 14 December 2002 and the day of the deceased's death.

7.3. The stated cause of death has to be examined in the light of clarifying comments made by Dr Gilbert, both as expressed in his report and in his evidence before me. Although Dr Gilbert attributes death to respiratory failure largely due to ARDS, he expresses the view that his death was also contributed to by patchy early bronchopneumonia and focal, relatively minor thromboembolism. Another relevant finding was that the deceased had severe narrowing of the left anterior descending coronary artery with areas of old ischaemic scarring in the anterior and posterior walls of the left ventricle. As well, the deceased's myocardium adjacent to the old ischaemic scarring showed very occasional microscopic foci as well as other irregularities that indicated that there had been microscopic ischaemic injury a few days before death and agonally. Dr Gilbert has described a situation where the deceased had on top of everything else undetected coronary artery and ischaemic heart disease with evidence that there had been myocardial infarction in the past. In his evidence Dr Gilbert suggested that the deceased's underlying ischaemic heart disease put him at risk, particularly when under stress, of having heart attacks or cardiac arrhythmias, either of which could cause death[16]. Dr Gilbert went so far as to say that the coronary artery disease was akin to a ticking time bomb as far as the deceased was concerned, in that it could have caused sudden unexpected death at any time. Dr Gilbert said that the blocked arteries could have led to a cardiac arrhythmia

[13] Exhibit C7c, Volume 3, Page 42
[14] Exhibit C7c, Volume 3, Page 43
[15] Exhibit C12
[16] Transcript, page 146

or a cardiac arrest without there necessarily being any evidence of overt infarction of the heart muscle. He said that although one could reasonably exclude the deceased as having experienced a major heart attack as a contributor to his death, what he could not exclude was the existence of a cardiac arrhythmia that may have contributed significantly to his respiratory arrest and cardiac arrest on 14 December 2002[17].

7.4. In ascribing the deceased's death to respiratory failure due to ARDS, Dr Gilbert provided this explanation to the Court:

> 'It's a non-specific condition in the lungs characterised by amongst other things, congestion, oedema or leakage of fluid into the lungs, loss of the lining cells in the small airways in the lungs, formation of membranes, of fibre within the airways and later by proliferation of fibrous tissue or scarring of the lung tissue. It obviously progresses in stages and I have more or less outlined the evolution of it. It's a response to a variety of different injuries. The lung only has a limited way of responding to various kinds of insults and that kind of pattern can be seen following cardiorespiratory arrest as a result of lack of oxygen to the lungs. It can be seen in infection, it can be seen as a side effect of certain drugs, it can be a response to radiation for example. It's a very non-specific but fairly well characterised pattern of injury to the lungs.' [18]

Dr Gilbert elaborated on the above. He also said this in relation to the syndrome:

> 'The failure of the lungs to bring oxygen in and the failure of the blood to bring oxygen to the lung tissues and transport away carbon dioxide causes damage to the small capillaries in the lungs and the cells lining the airspaces in the lungs and that's what sets off that cascade of that fairly non-specific but clinically fairly characteristic condition of adult respiratory distress syndrome. With respect to the amount of time it takes, or how long one has to have respiratory and cardiac compromised to develop it, I think that would be very variable and obviously it depends on factors like how long it takes to restore breathing and circulation as part of the resuscitation effort and obviously the presence of other complicating factors like infection. In his particular case I've got no particular views about - I haven't seen any particular problems with the timeliness and the effectiveness of his resuscitation. I'm really not sure why in the end he developed that but I've nominated some of the obvious risk factors for the department (sic) of ARDS in his case.' [19]

7.5. As to the possible contribution by a minor thromboembolism, as expressed in his original report, Dr Gilbert told me in evidence that during the deceased's time in intensive care a limited degree of pulmonary embolus had been demonstrated and that was borne out by the autopsy[20]. However, Dr Gilbert was of the view that pulmonary embolus was not sufficient to be identified as the sole cause of his significant

[17] Transcript, pages 178 and 179
[18] Transcript, page 130
[19] Transcript, page 176 and 177
[20] Transcript, page 141

respiratory failure nor indeed the sole cause for the respiratory arrest in the first instance on 14 December 2002.

7.6. Dr Gilbert also suggested that an infection that had been identified whilst the deceased was in intensive care could also have been a contributing factor in his death.

7.7. Although there may have been other contributing factors, I accept Dr Gilbert's evidence that the cause of death is to be attributed to respiratory failure due to ARDS (adult respiratory distress syndrome) and I find that to be the cause of death in this particular case.

7.8. As to how the deceased came to acquire ARDS, Dr Gilbert states in his report that '*the major contributing factor to the development of ARDS was probably the respiratory arrest on 14* December'. In evidence he expressed the view that although he could not be entirely certain as to what the initiating factors were, he said that the deceased's respiratory failure all seemed to follow on from the cardio respiratory arrest on 14 December 2002. Dr Gilbert explained that after that point in time the deceased required management in the Intensive Care Unit and there were changes developing in his lungs, that on a clinical and X-ray basis, appeared to resemble ARDS on 20 December 2002. This was confirmed by Dr Gilbert's findings at autopsy[21].

7.9. Dr Gilbert went on to state in the deceased's particular case that he suspected that the cardio pulmonary arrest on 14 December 2002, a period of poor oxygenation damaging the lungs and then possibly the evidence of infection and sepsis that was later identified were contributing factors to the development of ARDS[22]. While on the subject of infection, the source of the infection was never determined. Dr Gilbert suggested that it was probably related to the deceased's intensive care environment where, with intubation and ventilation, it is very common to develop pneumonia as a complication of that. Dr Gilbert suggested that there is nothing particularly uncommon about the development of infections in an intensive care setting.

7.10. On the subject of the connection between the cardio respiratory arrest on 14 December 2002 and the development of ARDS, Professor Robert Goldney, whose evidence in detail I will come to in due course, and who is a psychiatrist, at one point suggested that the clinical picture of the deceased between 14 December 2002 and the

[21] Transcript, pages 130 and 131
[22] Transcript, page 131

day of his death did not quite fit with such a connection. Professor Goldney was there speaking of the deceased's apparent alertness and level of consciousness at different points in time in that period. Dr Boghdadi also made reference to this in his statement. However, as earlier observed, the evidence of alertness, consciousness and responsiveness on the part of the deceased after his episode on 14 December 2002 is very limited and seems to be confined to the period prior to 18 December 2002. In any event, clearly at all material times between 14 December and 25 December 2002 he was being treated for respiratory failure and that remained, according to Dr Drysdale, the salient feature of the deceased's deterioration. There is no doubt in my mind that there is a significant connection between the events of 14 December 2002 and the deceased's death. I accept Dr Gilbert's evidence that a major contributing factor in the deceased's development of ARDS was the respiratory arrest on 14 December 2002. There may have been other contributing and predisposing features at work, and in addition, the question of a pulmonary embolus and infection cannot be ignored when one looks at the overall picture. However, the development of ARDS and its usual method of development as described by Dr Gilbert fits very well with the fact that the deceased had a respiratory arrest on 14 December 2002 and with his gradual decline after that date. This perceived break in the 'nexus' between the collapse on 14 December and the deceased's ultimate demise from ARDS arose in another context that I deal with below.

8. The cause of the deceased's cardio respiratory arrest on 14 December 2002

8.1. Another issue, however, concerns what it was that precipitated the respiratory arrest and cardio arrest on 14 December 2002. Dr Gilbert was asked about a possible connection between the administration of the drugs that the deceased had received on the evening of 14 December 2002 and his respiratory collapse later that evening. Counsel for the Department of Health, Mr Ralph Bonig, objected to Dr Gilbert providing an opinion as to the possible cause of the respiratory arrest on 14 December 2002, on the basis that Dr Gilbert was not qualified to offer such an opinion. I had no hesitation in overruling that objection. I found that Dr Gilbert was perfectly qualified to offer an opinion in relation to the cause of the respiratory arrest on 14 December 2002. However, it is fair to say that Dr Gilbert was guarded in relation to this particular issue. In his post-mortem report Dr Gilbert said this:

> 'The major contributing factor to the development of ARDS was probably the respiratory arrest on 14 December 2002. As noted clinically, the arrest was precipitated by sedation

> with haloperidol and clonazepam and was probably contributed to by the deceased's morbid obesity and pre-existing chronic obstructive pulmonary disease and may have been exacerbated by the deceased's clinically undetected ischaemic heart disease.' [23]

8.2. Dr Gilbert elaborated on this in evidence. Dr Gilbert described obvious risk factors with the deceased; firstly the sedation with a number of central nervous system depressant drugs, secondly the deceased's morbid obesity which made the work of breathing more difficult for him. Thirdly the deceased had chronic obstructive pulmonary disease which again compromised his respiration to some extent and fourthly at autopsy there was the identified significant coronary artery disease[24]. As far as a pulmonary embolism is concerned, Dr Gilbert repeated that in this context there was no clinical or post-mortem evidence that the deceased had a major and significant episode of pulmonary embolism on 14 December 2002 and suggested that there was little evidence that it would have been a substantial factor in his collapse. Dr Gilbert said that although he may have had a small pulmonary embolus at that stage, and it that it might have tipped the balance as far as his collapse was concerned, he suggested that it could not have been a substantial factor. I found those two propositions somewhat hard to reconcile at first, but later in his evidence he stated that there was no evidence of any massive pulmonary embolism of the kind that usually produces cardiac or respiratory arrest. He said that it remained a possibility that a small pulmonary embolus could initiate a cascade of events that result in respiratory and cardiac arrest. However, Dr Gilbert while recognising that possibility, was I think more impressed by other predisposing factors such as the deceased's obesity, his chronic obstructive pulmonary disease and his underlying and undiagnosed heart disease.

8.3. Dr Gilbert suggested that of all his illnesses and predispositions to cardio respiratory arrest, the one most likely to produce an unexpected adverse result was the coronary artery disease. Of course, this is not to say that this was necessarily the defining cause of the collapse in my view. The obvious temporal connection between the administration of sedative medication and the collapse cannot be ignored. The medication was administered at 2245 hours and the collapse was witnessed at 2351 hours. As earlier reported, Dr Gilbert suggested in his post-mortem report that, as noted clinically, the arrest was precipitated by sedation with Haloperidol and Clonazepam. Dr Gilbert maintained in his evidence that the sedative effects of the

[23] Exhibit C12, page 6
[24] Transcript, page 135

drugs still had to be considered as a possible contributing factor[25]. Dr Gilbert when asked as to whether he would put the sedative effects of the drugs at the bottom end of the scale of contributing factors, said that he would not necessarily agree with that proposition, but nevertheless did agree that observations that the deceased appeared to be awake and conscious just prior to his observed collapse tended to suggest that he was not that heavily sedated.

8.4. I think it is fair to say that what Dr Gilbert was content to postulate was that although sedation was not necessarily the precipitating factor in the deceased's collapse, it was one of a number of factors. I asked him:

> 'Q. I mean, if someone was to ask you this question: But for the administration of the sedation, would he have this event, are you able to say yes or no to that.
>
> A. I couldn't absolutely exclude that possibility because he had other problems. I mean, if you take away haloperidol and clonazepam, he's still got three sedating-type drugs, one of which, clozapine, has been given in a dose higher than had been prescribed; he's got significant coronary artery disease that no-one knows about; he's morbidly obese; and he's got chronic obstructive pulmonary disease.' [26]

8.5. The possible sedative effect of the medication that the deceased had been given is but one matter. There is also the suggestion that the antipsychotic medication that he had taken, namely the Haloperidol, in respect of which it was said that he had suffered adverse reactions in the past, as well as having suffered adverse reactions to other antipsychotic medications in the past, may have caused a dystonic reaction such as a laryngeal spasm. Although Dr Gilbert's post-mortem report does not deal with that possibility, he raised it in his evidence. I pause here to explain, if it is not clear already, that a laryngeal spasm could to a very large extent block off the deceased's airway. It would not be a question of sedation. It would be more a question of his mechanical ability to get air in and out of his lungs. Dr Gilbert suggested that a dystonic reaction can occur suddenly[27]. Dr Gilbert, while acknowledging that there was evidence that the deceased had been observed by Nurse Head to have been gasping for air, suggested that a more telling observation for a laryngeal spasm would have been a peculiar noise called laryngeal stridor which is caused by the difficulty involved in getting air past the larynx. However, later evidence would suggest that one would not necessarily observe this in a patient who had a complete airway blockage from laryngeal spasm. In addition, Dr Gilbert acknowledged that laryngeal

[25] Transcript, page 171
[26] Transcript, page 156
[27] Transcript, page 168

spasm might not involve stridor because of the sedation that the deceased in any event was under.

8.6. When Dr Gilbert's evidence is carefully considered I did not understand him to be advocating the existence of a laryngeal spasm as a cause of or contributing factor in the deceased's collapse on 14 December 2002, particularly when it is considered that Dr Gilbert was very much advocating other more obvious causes and contributing factors such as sedation from the medication and chronic obstructive airways disease, not to mention the undiagnosed ischaemic heart disease. It would not be possible on the evidence of Dr Gilbert alone for me to say with any conviction that laryngeal spasm, precipitated by antipsychotic medication, was a cause or contributing factor in the deceased's arrest on 14 December 2002.

8.7. Dr Gilbert exhibited a reluctance, quite understandably, to comment too pointedly on the effects of antipsychotic medication. Dr Gilbert would obviously defer to the opinions of a psychiatrist in that regard. It was for that reason that I caused the papers in this matter to be sent to Professor Goldney for his consideration. The Inquest was adjourned for that purpose.

8.8. As well, I thought it prudent to discover from Dr Lam what he meant precisely in his notes when he referred to the ease of intubation and whether that carried any implication in terms of the possibility of laryngeal spasm. Before dealing with Professor Goldney's evidence, it is well to record that, as early observed, Dr Lam saw no evidence of laryngeal spasm but did not entirely remove the possibility from the table because he conceded that it could have been in existence prior to his arrival on the ward.

8.9. Dr Lam told me that the ease with which the Guedel's airway had been inserted by Nurse Head before his arrival did not carry any implication as to whether or not the deceased had been experiencing any laryngeal spasm at that stage. Dr Lam said that he would expect that there would still be the ability to insert a Guedel's airway even with laryngeal spasm[28].

8.10. Dr Lam also said that one would not necessarily detect stridor in someone who was experiencing a completely blocked airway as a result of a laryngeal spasm.

[28] Transcript, pages 253 and 254

8.11. Dr Lam acknowledged that a respiratory collapse is sometimes associated with sedative effect of medication. Although Dr Lam did not himself witness the deceased gasping for air, or witness anything that Dr Lam would describe as agonal respirations, he told me that a person who was making a large respiratory effort due to a blocked airway may gasp. On the other hand, if their respiratory compromise was purely due to sedation rather than a blocked airway, then he would not expect to see gasping. He would not discount the possibility that there could be evidence of gasping in the situation where someone who has been sedated is endeavouring to get air, but it usually occurs in the context of a person who has been sedated but has an obstructed airway[29]. He told me that agonal respirations could still be consistent with sedation in that it means that the patient has not been sedated to a point where they have lost respiratory drive. Dr Lam was asked to consider the scenario described by Nurse Head of what appears to have been a level of consciousness followed by struggling for air and attempts to take breaths. In Dr Lam's long and considered response to this question he suggested that there are a number of possibilities to explain such a scenario. Having suggested that gasping associated with noisy breathing usually means that there is an obstruction, he went on to describe a number of competing possibilities. One possibility that he suggested was low oxygen being delivered to the brain, whether as a result of low circulation and blood pressure or due to poor levels of oxygen in the blood due to respiratory insufficiency or lung disease. He also suggested that it could be explained by the sedative medication gradually taking effect, with the deceased progressing from being agitated to sedated to over-sedated. Dr Lam suggested that the myriad of explanations for the scenario as described by Nurse Head very much gives rise to a large measure of speculation. The effect of Dr Lam's evidence is that what Nurse Head observed as far as the deceased's struggle to take a breath was concerned might be more in keeping with an obstruction of his airway as opposed to a reaction to sedative medication. However, clearly Dr Lam's evidence does not rule out the possibility that the picture painted by Nurse Head was one in which there had been an adverse reaction to the deceased's level of sedation as opposed to laryngeal spasm. Moreover, on Dr Lam's evidence there is simply no clinical support for the proposition that there was a laryngeal spasm. He inserted the endotracheal tube with ease. The airway was patent at the time he treated the deceased. It would be to draw a long bow to elevate the bare possibility of the existence of a laryngeal spasm before Dr Lam's arrival to fact.

[29] Transcript, page 262

8.12. It is against the background of all that material that Professor Goldney came to give evidence. Professor Goldney is now the Head of the discipline of Psychiatry at the Adelaide University based at the RAH. His report was tendered at the Inquest and is Exhibit C13.

8.13. Professor Goldney did not favour the scenario of the deceased having suffered laryngeal spasm giving rise to a respiratory arrest. Professor Goldney much preferred the scenario that Dr Gilbert had preferred, namely that there was a probable reaction to the sedative effect of the drugs. Professor Goldney proffered a number of reasons as to why he would eschew the suggestion of a laryngeal spasm. One of those reasons was that a much more violent and distressed reaction would have been expected on behalf of the deceased if his airway had been compromised. For example, there may have been an expectation that he would clutch his throat in distress, whereas there was no such reaction here as described by Nurse Head. Another reason that Professor Goldney would not have preferred laryngeal spasm as an explanation for the respiratory collapse was the clinical picture following his collapse being one where there appeared to be a measure of recovery, at least in the early days of his time in the Intensive Care Unit, as evidenced by his reacting with his relatives. This led Professor Goldney to conclude that there had been a break in the nexus between the respiratory arrest and his ultimate succumbing to ARDS. I had difficulty understanding that observation insofar as it was restricted to a consideration of whether or not a laryngeal spasm may have accounted for the respiratory collapse on 14 December 2002. The observation made by Professor Goldney would seem to apply equally to a respiratory arrest occasioned by an adverse reaction to the sedative effect of medication. In other words, it is difficult to understand why the mechanism by which the respiratory arrest occurred has anything to do with the way in which the deceased declined over the next 11 days. Looked at in another way, it also seemed to me that if the cause of death as expressed by Dr Gilbert is correct, and his view that that had been precipitated by the respiratory arrest on 14 December 2002 is also correct, what difference did it make in the final analysis as to whether the respiratory arrest was caused by one thing as opposed to the other. When that view of the matter was put to Professor Goldney he said he was prepared to take that point on board[30]. However, Professor Goldney suggested that one would have to carefully examine the clinical picture for the period between 14 December 2002 and 25 December 2002. He also suggested that perhaps the clinician would have been more qualified than Dr

[30] Transcript, page 233

Gilbert to offer an opinion about any nexus between the respiratory collapse and the development of ARDS and death. I do not accept that observation. Dr Gilbert is an experienced and highly qualified pathologist who quite clearly had access to and had examined all of the clinical notes. I have no hesitation in accepting Dr Gilbert when he says there was a probable connection between the respiratory arrest and the development of ARDS. There is nothing in the clinical picture that would suggest that there had been any significant level of recovery on the part of the deceased at any stage after that respiratory arrest. He remained intubated for several days after the collapse and there is really nothing in my view to suggest that his decline was anything other than the natural progression of his episode of 14 December. Although Dr Drysdale may have overstated the position to say that the deceased never regained consciousness, the fact of the matter was, as Dr Drysdale observes, that the deceased was in the Intensive Care Unit for the entire time and continued to have respiratory difficulty in spite of his intubation and ventilation. Furthermore, Dr Gilbert describes in my view a clear pathological connection between a respiratory collapse of the kind that the deceased suffered and a subsequent development of ARDS, particularly when it is borne in mind that the deceased already had a compromised ability to breathe from his chronic obstructive airways disease. To my mind there is a clear connection between the respiratory collapse on 14 December 2002 and the development of ARDS and the deceased's death.

8.14. The question remains whether it can it be shown to the necessary degree that the respiratory collapse was due to one thing as opposed to another.

8.15. As far as the contribution of the sedative effect of the medication is concerned, Professor Goldney referred to a number of matters that appeared to him to indicate that this had been the contributing factor to Mr Cockburn's respiratory collapse. In respect of the contribution of Haloperidol in particular, he said this:

> 'I think it did contribute but I'm not sure that it contributed with a dystonic reaction but one cannot deny the probability that any sedation associated with the haloperidol would have contributed along with a number of clinical factors to his respiratory distress.' [31]

8.16. The other clinical factors that Professor Goldney had in mind there were the combined effects of the Haloperidol together with the Clonazepam that he had been given at the same time, the fact that he had been given Clozapine on two occasions earlier in the day, remembering of course that he had been given 275mg of that

[31] Transcript, page 212

medication over and above what he should have received at that time, and he also referred to the contributing factors of the chronic obstructive airways disease, anaemia, and his undiagnosed cardiovascular disease. In the final analysis, aside from the differences between Professor Goldney and Dr Gilbert as far as any break of the 'nexus' was concerned, they both held the firm view that a dystonic reaction such as a laryngeal spasm was not the likely explanation for the respiratory collapse, and that the more likely role of the medication that had been administered to the deceased was its sedative effect. In spite of the fact that to my mind the scenario described by Nurse Head is not inconsistent with a dystonic reaction such as a laryngeal spasm, it is very difficult to reject the opinions of Dr Gilbert and Professor Goldney on the issue, notwithstanding the difficulty I had in understanding some of the reasons why they did not favour laryngeal spasm as the explanation for the deceased's collapse. It is also to be borne in mind that the very experienced clinician Dr Lam suggested that there were possible explanations other than spasm that are consistent with the effect of sedation. In addition, a laryngeal spasm is by no means the inevitable outcome even in patients who have exhibited severe reactions to antipsychotic medications in the past.

8.17. In my view, while the evidence is clear that the medication that was administered to the deceased was a contributing factor in his collapse on 14 December 2002, the evidence is insufficient to establish on a balance of probabilities that the mechanism giving rise to that respiratory collapse was a dystonic reaction to the antipsychotic medication in the form of a laryngeal spasm. Whilst there are some indications of a physical reaction that might be in keeping with the laryngeal spasm, and while recognising that there is a level of suspicion that this is what did occur here, there is insufficient clinical or expert evidence of it to the point where I could say confidently that it was more likely than not.

8.18. It is not possible to apportion the contribution of the medication to the deceased's respiratory collapse on 14 December 2002. The deceased had many co-morbidities. He was a very sick man, he was morbidly obese, suffered from disease of his airway; he had underlying ischaemic heart disease and anaemia, and the possible role of a pulmonary embolus cannot be overlooked if one accepts Dr Gilbert's evidence about that which I do. It is of particular note that the deceased's coronary artery disease and ischaemic heart disease on Dr Gilbert's evidence could have at any point in time triggered a cardiac arrhythmia and then cardiac and respiratory arrest. The possibility

of that arising in the deceased's case was so marked that Dr Gilbert told me that if there had been no history of the administration of the medication that we know about, and if he had not succumbed to ARDS, on a post-mortem examination Dr Gilbert would have been content to conclude that the deceased had died of ischaemic heart disease. However, one thing that cannot be overlooked is the fact that the deceased's respiratory collapse occurred about an hour after he was administered the Haloperidol and Clonazepam, and both Dr Gilbert and Professor Goldney were of the view that there was a probable connection between the level of sedation occasioned thereby and his collapse. I accept their evidence on this issue. I am persuaded that the probability is that the medication had a part to play in the deceased's collapse and that the role was more likely to have been its sedative effect on the deceased in combination with his co-morbidities. However, the precise level of contribution by the medication to the collapse is not clear.

9. Should the deceased have been given the medication

9.1. It has been established that the deceased was given 400mg of Clozapine at 9pm. We have seen already that this was in excess of his usual dosage. The Haloperidol (10mg) and Clonazepam (2mg) were administered at 2245 hours. I think it is fair to say that Dr Boghdadi conceded that if he had been made aware that the deceased had documented adverse reactions to antipsychotic medication, he probably would not have administered Haloperidol[32]. There was considerable debate in the Inquest about what the deceased's clinical records truly revealed as far as adverse reactions to Haloperidol in particular was concerned. But it is clear to me that the deceased had quite a history of adverse reaction to antipsychotic medications in general. Both Dr Gilbert and Professor Goldney were of the view that if there had been a demonstrated propensity to exhibit adverse reactions to other antipsychotic medication, such a Fluphenazine, then the administration of a drug like Haloperidol would be contraindicated. Professor Goldney went so far as to say that the administration of Haloperidol with the deceased's history was '*unwise*' in the circumstances[33]. Nevertheless, Professor Goldney was not otherwise critical of what had transpired on 14 December 2002 for the most part taking into account the adverse circumstances in which the decision to administer the medication was made. For my part, if I had found that the deceased's respiratory collapse had been precipitated by a laryngeal spasm brought on by the antipsychotic medication to which the deceased had a well

[32] Transcript, page 101
[33] Transcript, page 216

understood aversion, there would have been obvious scope for a significant level of criticism to be directed at what had transpired here. However, as seen, the preferable explanation is that the effect of the medication was sedative and that the level of sedation was a contributing factor.

9.2. Even though the deceased's collapse on 14 December 2002 cannot be ascribed to a dystonic reaction, the fact of the matter is that the deceased was given inappropriate medication, and that this involved a significant risk to his well being. He had been given a dose of Clozapine that was significantly in excess of his usual dosage. That dosage would have contributed to the level of sedation that he experienced. He was given an antipsychotic that he had a documented aversion to. All of this occurred because an inappropriate level of scrutiny was accorded to the documentation that was available in the hospital, if not in the deceased's ward, which plainly set all of this out.

9.3. However, there had been a clear need for the deceased to be brought under control. There were two reasons for this; firstly the treatment that he was being given, that is the transfusion, had not concluded and secondly, it does not require much imagination in these circumstances to conclude that the deceased was presenting a particular danger, not only to others, but to himself. There was a need for the deceased to be sedated quickly. There was no outward sign that the medication that he had been given earlier, even with the larger than usual dose of Clozapine, had resulted in the deceased experiencing any meaningful sedative effect from it. Professor Goldney told me in hindsight that it would have been better if the deceased had been administered something other than Haloperidol, such as another intramuscularly delivered medication. He suggested that Diazepam (Valium) given intramuscularly would have been the medication of choice. While I agree that there was a clear necessity for the deceased to have been given sedative medication, I do not agree that the matter should exclusively be examined in hindsight. There was clear information available at the time from which the only conclusion to be drawn was that Mr Cockburn should not be given Haloperidol.

9.4. However, it should be recorded that a possibility remains that it would not have mattered what particular sedative medication he was given. In Professor Goldney's opinion the outcome may well have been the same, whether he had been given Haloperidol that has a sedative effect, or whether he had been given Diazepam, with its sedative effect. Some might say, therefore, that it is somewhat immaterial that the

deceased was given the drug Haloperidol in these circumstances. In my view the matter cannot be viewed so simplistically. Whilst nobody can say with certainty that the administration of any sedative drug in any amount would have dictated the same outcome, by the same token it can never be known whether a more conservative approach to subduing the deceased's behaviour might have changed the outcome. In addition, the role of the excessive dose of Clozapine at 9pm will also never be fully understood.

9.5. Dr Boghdadi candidly acknowledged that before he ordered the deceased to be given Haloperidol and Clonazepam, he should have referred to the written material that had been provided to the RAH by Glenside which explained the deceased's adverse reactions to antipsychotic medication. If he had seen Haloperidol listed on the PRN chart under the heading '*Adverse drug reactions*', he would not have prescribed that medication. Similarly, Dr Boghdadi agreed that if he had known that the deceased had a history of unfavourable reactions to antipsychotic medications such as laryngeal spasm, he probably would have sought out advice from a psychiatric registrar about the appropriateness of using such medication. I agree with Professor Goldney that it was an unwise course for Dr Boghdadi to take without referring to any documentation that was available on the ward on that issue. Dr Boghdadi told me that normally he would seek out and look at the PRN chart because that would be the chart that he would use to prescribe drugs. He agreed that he would do that even in a situation involving urgency. As to why he did not do that in this case, he told me that the urgency of the situation, coupled with his other workload in other wards, were the probable reasons. There was much to admire about Dr Boghdadi's candour in this Inquest and it has to be recognised that he was in a very awkward situation on the night in question. His quite appropriate strategy of prescribing oral sedative medication had not worked. He was confronted with a set of circumstances that were adverse to say the least, but they were circumstances in which he was required to act quickly.

9.6. The other worrying aspect of the matter is that nursing staff had not examined the PRN drug chart either. Nurse Stafford, who was the person who actually physically administered the drugs, told me that she had no recollection of seeing the chart, although the evidence is plain that it was available on the ward. Like Dr Boghdadi, she also told me that if the PRN chart had been referred to, and if she had seen the warning on it under the heading '*Adverse drug reactions'*, as she inevitably would

have, she would not have given the drugs including the Haloperidol. She told me that because of the urgency of the situation, she only looked at the stat chart which did not have any drug alert on it.

9.7. However, it has not been demonstrated on a balance of probability that any shortcoming in respect of the failure to examine the documentation about the deceased's adverse drug reactions caused the deceased's death because I am unable to conclude firstly that there was a dystonic reaction to the medication and secondly, that any alternate strategy that Dr Boghdadi may have adopted in order to sedate the deceased would necessarily have dictated a different outcome.

10. Recommendations

10.1. Pursuant to section 25(2) of the Coroner's Act 2003 I am empowered to make recommendations that in the opinion of the Court might prevent, or reduce the likelihood of, a recurrence of an event similar to the event that was the subject of the inquest.

10.2. Mr Bonig on behalf of the RAH tendered two affidavits from members of RAH staff relating to changes in procedures as far as the creation of medication alerts are concerned. The procedures include the automatic transfer of medication alert forms from one volume of a patient's file to the current file so that it sits at the front of every current volume of a patient's clinical record.

10.3. In addition, the RAH have changed their PRN medication charts. There have been other changes to procedures that in the main relate to the creation and transmission of written information that might make the existence of important information such as a medication alert more obvious to nursing and medical staff.

10.4. These measures may have changed things in Mr Cockburn's case because the medication alerts may have been more obviously displayed on the file, but it has to remembered in this case that the PRN drug chart, prepared at the time of his admission, and which clearly identified the drugs to which Mr Cockburn might have an adverse reaction, was present on the ward. Nurse Head recognised that in a patient such as Mr Cockburn, coming from where he did and with his history, the existence of such a document in his file was virtually inevitable and would be assumed. However, she told me that she would not have looked at the PRN chart because of an erroneous assumption that the stat chart that she said she did look at should have

reflected all of what was on the PRN chart. It has to be borne in mind here that all the charts in the world are of little use unless they are accurately compiled and actually looked at. One would have thought that even in a situation of urgency there is a clear need for written information to be examined carefully, particularly where medication that is known to cause adverse side effects is prescribed. This is not the first occasion that a failure to examine clinical records thoroughly has been the subject of coronial comment.

10.5. It seems that much of what occurred here may have been avoided, and I refer here to the prescription of the Haloperidol and Clonazepam, if Dr Boghdadi had time to consult a superior. He was after all an intern who had been called upon to make a snap decision in very unfavourable circumstances, and where his first strategy of prescribing less dangerous oral medication had not borne fruit. Dr Boghdadi referred me to an RAH protocol that suggests that a prescription of Haloperidol and Clonazepam combined is a standard response to the kind of situation that presented itself here, and he said that he acted in accordance with the protocol after the deceased had refused to take oral sedative medication. That may well be the case, but a more experienced practitioner, who had examined the clinical record, and in particular the PRN drug chart, to my mind probably would have acted differently. In his witness statement, Dr Boghdadi suggests that it would be appropriate to recommend that the on duty medical or psychiatry registrar attend all the restraint calls at all hours. It is difficult to argue with the wisdom of that. I do not know how readily or otherwise such a procedure could be implemented, but I would suggest that it is a measure that at least ought to be given consideration by the RAH.

10.6. I was also troubled in this case by the fact that Glenside provided information to the RAH that was simply incorrect. I refer here to the substitution of the deceased's Glenside drug chart with that of Nancarrow. I do not know how this occurred.

10.7. I make the following recommendations:

a) That the RAH formally remind all nursing and medical staff of the need to carefully examine a patient's file for the existence of and contents of any medication alerts before medication that is known to produce adverse side effects is administered;

b) That the RAH consider implementing measures to ensure that medical practitioners of the level of registrar or above are preferentially called to restraint

situations, or are at least made aware of the existence of a restraint situation and are tasked to be ready to provide advice to junior practitioners if necessary;

c) That the RAH formally impress upon its nursing and medical staff, especially interns, to consult more senior staff if in doubt about the appropriateness of prescribing particular medication;

d) That Glenside implement measures to ensure that a substitution of one patient's documentation for that of another does not happen again.

Key Words: Death in Custody; Mental/Psychiatric Illness; Adverse Reactions; Adult Respiratory Distress Syndrome

In witness whereof the said Coroner has hereunto set and subscribed his hand and Seal the 29th day of June, 2007.

Deputy State Coroner

Inquest Number 4/2007 (3569/2002)