CORONERS ACT, 1975 AS AMENDED

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 29th, 30th and 31st days of March, and the 1st and 20th days of April 2004, before Wayne Cromwell Chivell, a Coroner for the said State, concerning the death of Michael Jason Taylor (aka Michael Jason Carter).

I, the said Coroner, find that Michael Jason Taylor (aka Michael Jason Carter) aged 27 years, late of Glenside Campus of the Royal Adelaide Hospital, 226 Fullarton Road, Eastwood, South Australia died at Eastwood, South Australia on the 26th day of February 2000. The cause of death has not been determined.

1. Reason for inquest

1.1. On 4 January 2000 the Guardianship Board of South Australia made an order pursuant to Section 13 of the Mental Health Act 1993 detaining Mr Taylor in Glenside Hospital for a period of 12 months. The Board also ordered that Mr Taylor be treated for his mental illness during the period of his detention.

1.2. Accordingly, when Mr Taylor died on or about 26 February 2000, he was 'detained in custody pursuant to an Act or law of the State' within the meaning of Section 12(1)(da) of the Coroners Act 1975, and an Inquest into his death was therefore mandatory by virtue of Section 14(1a) of the said Act.

2. Introduction

2.1. Little is known about Mr Taylor's background and upbringing. A psychological report prepared in 1994 by Forensic Psychologist Dr Jack White (which forms part of the James Nash House clinical record, Exhibit C16) asserts that Mr Taylor was born

on 15 January 1973 in Western Australia. He had lived in Adelaide since 1991. He was the eldest of three children, his parents were separated and he was no longer in contact with his family.

2.2. Mr Taylor left school in year 9 due to 'family problems'. He reported having 'major difficulties' in all school subjects. He worked in various occupations and began getting into trouble with the law, particularly through stealing.

2.3. Dr White assessed that Mr Taylor's cognitive function was 'well below normal for most of his age-related peers'. Dr White concluded that Mr Taylor was of 'borderline intelligence', and wondered whether he had an organic impairment, possibly from frontal lobe brain damage. He recommended further testing to determine the exact nature of his neuropsychological deficits, and also recommended a medical examination (eg. a CT scan) which might further clarify his organic problems.

2.4. On 8 March 1996, Mr Taylor was convicted of armed robbery, damaging property, assaulting police and escaping custody and was sentenced to 8 years and 2 months imprisonment, to commence from 23 October 1995. He was an inmate in prison until 21 November 1996 when he was admitted to James Nash House (JNH), a forensic psychiatric facility at Hillcrest (now Oakden).

2.5. Dr Ken O'Brien, who was then (and still is) the Clinical Director of the Forensic Mental Health Service which includes JNH, reports that Mr Taylor was suffering from a severe mental illness (chronic schizophrenia), and that he had an estimated IQ of 77. He said:

> 'For a prolonged period of time he greatly suffered from the debilitating effects of this illness which required a variety of therapeutic interventions including the prescribing of the antipsychotic preparation Clozapine and the administration of electro-convulsive therapy (for which I made an application to the Guardianship Board on 28 January 1997). Furthermore, he had a history of several suicide attempts and in that respect remained a chronic suicidal risk. He also had a record of non-compliance with medication and attempted escapes from detention. In summary therefore Mr Taylor demonstrated complex pathologies and behaviours whilst frequently his day to day management was challenging.'
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> (Exhibit C, p2)

2.6. Dr O'Brien said that the 21 November 1996 admission was Mr Taylor's fourth to JNH. He was there until 22 October 1999 which, Dr O'Brien said, was indicative of the 'severity and chronicity' of his illness (ibid).

2.7. Mr Taylor was discharged on parole from JNH on 22 October 1999. In the meantime, the Guardianship Board of South Australia had made an order on 4 January 1999 that Mr Taylor be detained for a further period of 12 months. When he was discharged from JNH Mr Taylor was transferred to Glenside Hospital, Kurrajong Ward, which was a closed, secure ward.

2.8. While in Kurrajong Ward, Mr Taylor made what appeared to have been an escape attempt. Had commented to staff that he would jump the fence, and the following day a laceration to his right wrist was noted. This was sutured at the Royal Adelaide Hospital.

2.9. After this incident, Mr Taylor's behaviour was described as 'difficult'.

2.10. On 5 November 1999 Mr Taylor was seen by Dr Raymond Behrens, Senior Psychiatric Registrar, who described him as 'quite psychotic'. Dr Behrens said that he believed that he was receiving messages from 'the family' via the television. The voices were telling him that he was going to burn to death. Dr Behrens assessed that Mr Taylor was an acute risk both to himself and to others.

2.11. On 31 December 1999 Dr Behrens saw Mr Taylor again and commented that although he had made 'significant gains', he remained delusional and continued to require supervision in a 'secure and supportive environment'. Dr Behrens also commented that the transition from a closed ward needed to be done 'gradually and carefully'.

2.12. On 4 January 2000, the Guardianship Board of South Australia made an order for continuing detention for a further period of 12 months, as I have already outlined.

2.13. On 17 January 2000, Mr Taylor was transferred out of Kurrajong Ward to Greenhill Ward. Greenhill was not a closed ward.

2.14. On 5 February 2000 it was noted by nursing staff that Mr Taylor had spent 'lengthy periods' absent from the ward. He was counselled about this, as he was a detained patient and was not entitled to leave. At 8:45pm that evening, Dr Jini Mukherjee, Psychiatric Registrar, was called to the hospital as it was noted that Mr Taylor was unresponsive and cyanotic with difficulty breathing. An ambulance was called and Mr Taylor was transferred to the Royal Adelaide Hospital where naloxone (an opiate

antagonist) was administered. There was a significant improvement in his condition. Upon cessation of the naloxone infusion, his conscious state deteriorated again, and then he slowly improved.

2.15. A blood test taken on 6 February 2000 disclosed the presence of methamphetamine and olanzapine. The presence of these two substances was confirmed by mass spectrometry. Opiates were also detected, but they could not be confirmed in this way so their presence was 'presumptive' only.

2.16. Dr Behrens told me, and I accept, that the symptoms displayed by Mr Taylor, and in particular the response to naloxone, suggests that his collapse was due to an opiate toxicity (T44).

2.17. Mr Taylor remained in the Royal Adelaide Hospital until 9 February 2000 when he returned to Kurrajong Ward at Glenside Hospital. On 14 February 2000 he was returned to Greenhill Ward. This time he was placed in one of the closed 'observation' rooms. On 21 February 2000 he was returned to the open ward at Greenhill.

2.18. From 21 to 26 February 2000 Mr Taylor was on a regime whereby he was required to 'sign in'. Initially, this was every half-hour, and then on an hourly basis. It is noted that for the next five days, apart from several periods when he was noted as being asleep or out shopping, Mr Taylor was largely compliant with this requirement.

2.19. Mr Taylor's apparent overdose on 5 February 2000, and his behaviour in leaving the hospital grounds without permission, were reported to his parole officer by Glenside Hospital staff on 7 February 2000. The Glenside Hospital clinical record states that Mr Taylor was advised on 21 February 2000 that these reports had been made, and that a hearing of the Parole Board, at which these alleged breaches of his parole would be discussed, was imminent.

2.20. On 24 February 2000 Mr Taylor went into the city with two members of Glenside Hospital staff to buy a pair of shoes. He is reported to have enjoyed this outing. On 25 February 2000 he was described as having spent a 'quiet day around the ward' and that he had been compliant with his signing-in obligations.

2.21. A patient, Rodney English, gave a statement to police alleging that Mr Taylor liked to 'mess around with all the drugs that were on offer', and that he and another patient, Ken Wipa, were absent from dinner that evening. Mr English said that at about 7:30pm, Mr Taylor was 'stoned off his tits' (Exhibit C6a, p2). He said that Mr Wipa 'gestured with his hands in an injecting motion' when he was asked what was wrong with Mr Taylor (Exhibit C6a, p2).

2.22. Mr Wipa told police that he also saw Mr Taylor at around 7:30pm on 25 February 2000, and described him as looking 'wasted'. He said that Mr Taylor told him that he had been to the Arkaba (Hotel), and that he was 'on heroin'. He said that after Mr Taylor smoked a couple of cigarettes, he saw him walk over to the lawn and vomit on three separate occasions. When he returned, he told Mr Wipa not to say anything. He said that he saw Mr Taylor vomiting again, at around 8:05pm. He said that he saw him during the evening, and that he seemed to improve as he was drinking water. At around 11:15pm, he came over to Mr Wipa's bed and said that he was still 'wasted' (Exhibit C9a, p3). He said that this was the last time he spoke to Mr Taylor.

2.23. Another patient, Alan Peric, said that he also saw Mr Taylor vomit at some stage during the evening of 25 February 2000 (Exhibit C10a).

3. Cause of death

3.1. A post-mortem examination of the body of the deceased was performed by Dr J D Gilbert, Forensic Pathologist, on 27 February 2000 at my direction.

3.2. Dr Gilbert's conclusion was that the cause of Mr Taylor's death was 'olanzapine toxicity'. He commented:

> '1. Death has been attributed to olanzapine toxicity. The circumstances of the death suggested that the deceased may have used heroin on the evening before his death but only a very low level of morphine was identified in the blood and no monoacetylmorphine was found in the urine. These findings effectively exclude heroin toxicity as the cause of death. The blood contained a therapeutic level of valproic acid, an anticonvulsant and antipsychotic. Although the deceased was prescribed fluvoxamine (an SSRI antidepressant), none was identified in the blood. He was also prescribed zuclopenthixol (an antipsychotic) but Forensic Science currently has no method for its analysis. An olanzapine level of 0.58 mg/L was reported.
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2. Olanzapine may have reached greater than therapeutic levels in the deceased's blood because of interactions with other prescribed medications. Clozapine, a chemically similar antipsychotic, may interact with SSRI antidepressants especially fluvoxamine. It is quite likely that similar problems occur with olanzapine.

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   …

   In summary, if deliberate or accidental overdosage of olanzapine can be excluded, there is a strong theoretical basis to suggest that the blood levels may have become elevated because of interactions with valproate and/or fluvoxamine.

3. Assuming that the deceased was found on top of his bedclothes and clothed only in underpants, the rectal and ambient temperatures noted at the scene were consistent with death occurring approximately 7 hours prior to my scene examination ie. at around 0630 hours 26/2/2000 (+/- 2.8 hours, 95% confidence limits).

4. There were no injuries or other markings on the body to indicate the involvement of another person in the death.

5. No natural disease that could have caused or contributed to the death was identified at autopsy. There was no evidence of renal or hepatic disease that may have affected the deceased's ability to metabolise prescribed medications.'

(Exhibit C3a, pp5-6)

3.3. At Dr Gilbert's request, a toxicological analysis of Mr Taylor's blood was performed by Ms Joanna Rositano, Forensic Scientist, and this disclosed the presence of:

- 0.58mg olanzapine per litre;
- 59mg valproic acid per litre;
- 0.03mg morphine per litre.

(Exhibit C17, p1)

3.4. Ms Rositano also reported that alcohol was not detected in the blood. The urine did not contain monoacetylmorphine, although it did contain morphine. Zuclopenthixol could not be positively identified or quantified, as the Forensic Science SA laboratory was not equipped to test for this substance.

3.5. In a subsequent report Ms Rositano reported that the blood also contained approximately 0.04mg of fluvoxamine per litre (see Exhibit C17a).

3.6. Dr Gilbert issued a further report (Exhibit C3b) in which he found the presence of fluvoxamine significant in that it may well have inhibited the metabolism of

olanzapine, causing the concentration to increase to unexpected levels. He suggested the opinion of a clinical pharmacologist.

3.7. Role of medication in cause of death

I heard evidence from Mr Peter Felgate, the Manager, Toxicology, at Forensic Science SA. Mr Felgate verified the results achieved by Ms Rositano.

3.8. Mr Felgate told me that the screening method adopted by the toxicologists excluded the presence of over 100 common drugs which were listed in Exhibit C17c.

3.9. Mr Taylor was prescribed olanzapine at the rate of 20mg at night. His post-mortem level was 0.58mg per litre, or 580 nanograms per millilitre. The only other time Mr Taylor's blood was tested for olanzapine was on 24 November 1999 when the level was 46 nanograms per millilitre. The combination of drugs he was receiving was the same. Mr Felgate told me that in his opinion, a level of 0.58mg per litre is inconsistent with therapeutic use of olanzapine (T124).

3.10. I heard evidence by video-link from Dr Nick Buckley, who is the Senior Consultant in both Clinical Pharmacology and Toxicology at The Canberra Hospital. Dr Buckley is widely experienced in toxicology and pharmacokinetics, and I have relied upon his expert opinions in a number of previous inquests.

3.11. Mr Felgate said that he read the report of Dr Buckley and he agreed with Dr Buckley's comments (T130).

3.12. Dr Buckley made the following points:

- Olanzapine is an atypical antipsychotic medication which is approved for use in schizophrenia and it has significant anticholinergic and sedative effects, it has a lower incidence of acute and chronic 'extrapyramidal' side effects than typical antipsychotic drugs. Although there have been some reports of myocarditis secondary to olanzapine use, sudden death and cardiac arrhythmia have not been attributed to therapeutic use of olanzapine in the 'mainstream published literature' (Exhibit C18, p2);

- The symptoms of overdose include central nervous system depression (drowsiness, slurred speech, ataxia [loss of coordination], dizziness and coma),

tachycardia (fast heart rate), hypotension, ECG changes, pinpoint pupils and unresponsiveness to naloxone;

- The usual daily dosage is 5mg to 20mg, which results in therapeutic blood concentrations of between 0.01mg to 0.03mg per litre. In deliberate overdose, the concentrations are generally ten-fold higher, and those in the few fatal poisonings have usually been over 1mg per litre;

- It is likely that there would have been some post-mortem redistribution of olanzapine. He explained:

  > 'The post-mortem redistribution is where blood levels rise, or, less commonly, fall, after death ... So, with olanzapine, what has been reported is a two to threefold increase post-mortem. For some drugs, it can be higher, but that's what has been reported for olanzapine'. (T145-146);

- Although there have been some instances of fatal overdose, reports of olanzapine overdose have generally reported few serious toxic effects;

- The product information for olanzapine (Zyprexa) does not report interaction with fluvoxamine. It does report the fact that fluvoxamine caused a 16% increase in the maximum plasma concentration of olanzapine, that metabolism of olanzapine may be increased by smoking thereby lowering the concentration, and carbamazepine therapy will increase metabolism even further;

- Fluvoxamine is a selective serotonin reuptake inhibitor (SSRI), which inhibits a number of drug metabolising enzymes and thus may increase the concentrations of olanzapine. Dr Buckley said that increases in clozapine concentrations as a result of co-prescription of fluvoxamine have been seven to eleven-fold, and that similar increases might be anticipated with olanzapine although 'on the whole these have not been as marked - one report suggests two to three-fold increases were usual' (Exhibit C18, p3);

- The product information for fluvoxamine refers to these interactions and states:

  > 'An increase in previously stable plasma levels of those tricyclic antidepressants (such as clomipramine, amitriptyline and imipramine) and neuroleptics (or antipsychotics such as clozapine and olanzapine) which are largely metabolised through CYP1A2 has been reported when used together with fluvoxamine. <u>The combination of fluvoxamine with these drugs is not recommended</u>.'
  >
  > (Exhibit C18, p3) (my underlining)

3.13. Mr Taylor was also receiving sodium valproate which is an anticonvulsant medication, and a mood stabiliser. Dr Buckley said that it does not have a significant sedative effect.

3.14. As to the presence of morphine in Mr Taylor's blood, Dr Buckley said the concentration of 0.03mg per litre was a relatively low concentration, and was consistent with Mr Taylor having taken heroin to the extent that he was intoxicated, as deposed to by the witnesses English and Wipa, at around 7:30pm the night before. He said:

> 'Heroin and morphine in overdose present with a syndrome, which includes miosis (small pupils), coma, respiratory depression and vomiting. Central nervous system depression is the major clinical manifestation. Lesser degrees of sedation may lead to confusion, incoordination and low blood pressure. Increasing doses lead to increasing degrees of sedation with initial analgesia and sedation, followed by loss of response to verbal stimuli, loss of response to tactile stimuli, loss of control over normal respiration and failure of temperature and blood pressure regulation. Respiratory arrest is the usual mode of death. It is possible that these drug effects (ie. marked sedation) could also lead to prolonged hypoxia and profound neurological damage and thus lead to a delayed death after many hours.
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> Heroin and morphine are μ-opioid receptor antagonists. Stimulation of these receptors in the central nervous systems leads to analgesia, vomiting and profound sedation in a dose dependent manner. Other sedative drugs (eg. alcohol, benzodiazepines) potentiate these effects and the majority of fatal overdoses involve other substances. Non cardiogenic pulmonary oedema occurs in a substantial number of opioid overdoses, however the mechanism behind this is unknown.'
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> (Exhibit C18, p5)

3.15. Dr Buckley said that having regard to the post-mortem concentration, he would not have expected that the concentration of morphine in the system would have reached the levels likely to have caused death, even in combination with olanzapine (T149).

3.16. The further toxicology testing referred to in Exhibit C17b excluded the possibility that other opioid (but non-opiate) drugs played a part in Mr Taylor's death (T150).

4. Issues arising at inquest

4.1. Standard of psychiatric care

Dr O'Brien gave me his expert opinion on the level of care given to Mr Taylor during his stay at Glenside Hospital. He said:

> '(1) In my opinion, the quality of the treatment provided to Mr Taylor whilst at Glenside Hospital was within acceptable limits. Repeatedly, his challenging

pathologies and behaviours had been highlighted and in some respects and with the passage of time his mental illness, prior to his death, was becoming under greater control. Much, though not all, of his challenging behaviours were related to his personality style, his drug seeking behaviour and his low intelligence. It would seem to me that the staff acted cautiously, bearing in mind his historical and current mental and behavioural state. Furthermore, the written records, in my opinion, were appropriately maintained and reflected his progress.

(2) It is a truism that if Mr Taylor had continued to be housed in a well staffed and high secure environment (such as James Nash House) the likelihood of the final adverse outcome might have been reduced. However, even if it had been administratively and legally possible, in my opinion, it would have been an unacceptable invasion of his civil rights and privacy if such intrusive tight security measures had been maintained on a long term basis. Glenside Hospital, in terms of its rehabilitative potential, has a number of significant advantages over James Nash House. Particularly, it has a diverse range of ward areas (sometimes referred to as "step down" facilities) in which patients can be placed either as a result of their improved mental state or, sometimes, to test out their capacity to respond positively to a less intrusive environment. It is my opinion that the staff at Glenside Hospital used their clinical discretion about the placement of Mr Taylor in a cautious and prudent manner. It would seem to me that for the most part each step was carefully considered and, once implemented, monitored. Naturally, not all clinicians necessarily might agree with the timing of each and every transfer. One could perhaps argue, that initially he should have been placed in the closed portion of Greenhill and that on his return to that unit he could have spent a slightly longer period in the closed area of Greenhill but the reasons for transferring him to the open area were persuasive – the only other alternative would have been to return him, yet again, to the unsatisfactory Kurrajong ward. A system of monitoring and signing on was put in place once he was in the more open area.

The police report questions the advisability of detained patients being permitted to use the hospital grounds in an unsupervised manner. A contrary view, is that a psychiatric hospital is a place of "asylum" and that a reasonable amount of freedom of movement should be permitted. It is my understanding that with some patients a system of escorted ground privileges is used but it must be acknowledged that this has significant staffing and resourcing implications that may well result in the paradoxical outcome of a decreased usage of the hospital facilities and grounds, thereby limiting "normality". Therefore, bearing in mind all of these considerations, although I fully appreciate the concern of the police, I do not believe that detained patients at Glenside Hospital should not, under appropriate circumstances, be allowed to use the Glenside Hospital grounds in an unescorted manner.'

(Exhibit C15, pp4-5)

4.2. During the many inquests that I have conducted into the deaths of patients of the mental health system in South Australia, it has become apparent that a patient who is 'detained' pursuant to the Mental Health Act 1993 is not necessarily confined to a

closed ward. Indeed, there are detained patients in certain hospitals in the State where there are no closed ward facilities. This system is so entrenched in South Australian psychiatric care that it seems pointless for me to re-examine it.

4.3. However, the care of a 'detained' patient involves a very high degree of responsibility on the part of the institution concerned to take adequate measures to preserve the safety of the patient and others. After all, that is the point of detention.

4.4. On 5 February 2000, Mr Taylor took an overdose of heroin, and possibly of anti-parkinsonian medication as well, which necessitated his admission to the Royal Adelaide Hospital High Dependency Unit. There is a notation in the Royal Adelaide Hospital clinical record that Mr Taylor had been seen with another patient, Mr Mark Anderson, walking along Unley Road on the evening of 4 February 2000. I was told that Mr Anderson is an alleged drug user and supplier to other patients in the hospital.

4.5. Having regard to that incident, it is surprising to me, notwithstanding Dr O'Brien's comments, that only 20 days later Mr Taylor was able to gain access to the Arkaba Hotel and obtain further heroin. It is also surprising that his obvious intoxication on the night of 25 February 2000 did not come to the attention of the staff at Glenside Hospital.

4.6. To the extent that Mr Taylor's detention was for the purpose of preserving his safety, it was not successful.

5. Conclusions

5.1. Dr Buckley told me that even though the level of olanzapine in Mr Taylor's blood might have been increased by the presence of fluvoxamine and sodium valproate, these medications were also in his blood on 24 November 1999 and yet the concentration of olanzapine was approximately $^{1}/_{12}^{th}$ the post-mortem level. For this reason, Dr Buckley said that it is not possible to explain the post-mortem concentrations in terms of a therapeutic dose of olanzapine having been potentiated by fluvoxamine and/or sodium valproate.

5.2. Dr Buckley said that this left only three possibilities:

- Mr Taylor took a deliberate overdose of olanzapine, illicitly obtained by some means, on the night of 25 February 2000. He estimated that around 20 tablets

would have been required, although the range may be anywhere between 5 and 40 tablets (T157);

- Some (as yet unaccounted for) drug interaction took place. For example, if Mr Taylor had been non-compliant with fluvoxamine in November 1999 but compliant in February 2000, an unexpectedly large potentiation of olanzapine may have occurred (T148);

- The post-mortem redistribution of olanzapine was much higher than the two to three-fold increase which Dr Buckley would have expected (T148).

5.3. In the absence of any evidence to support any of these theories, Dr Buckley described the cause of Mr Taylor's death as a 'puzzle', and led him to speculate that there might be some other mechanism of death. Possibilities include a cardiac arrhythmia or, even less likely, a seizure. He pointed out that such effects have yet to be demonstrated in the literature, but this fact does not exclude these possibilities, since it took more than 40 years to detect side effects associated with Thioridazine (T154).

5.4. Dr Buckley said that he regarded olanzapine toxicity as an unconvincing explanation for Mr Taylor's death, although it was the best of a series of unlikely possibilities (T151).

5.5. In view of Dr Buckley's evidence, I am unable to make a finding, on the balance of probabilities, as to the cause of Mr Taylor's death. Whether he obtained and took some other substance which has not been detected post-mortem which may have been involved in his death, whether his death was caused by an as yet undemonstrated and unexpected side effect of olanzapine or some other of his medications, or whether his death was due to an as yet undemonstrated physical cause such as arrhythmia or seizure, are matters for speculation. Accordingly, I am unable to do more than find that the cause of Mr Taylor's death has not been determined.

6. Recommendations

6.1. Dr Buckley's evidence suggests that the product information which accompanies olanzapine is deficient, in that it fails to warn of the interaction between olanzapine and fluvoxamine, which he described as one of its more significant interactions. He also told me that, in his opinion, the product information for fluvoxamine, although it

does warn of this interaction, does not do so in particularly clear terms and the warning is to be found among the 'fine print' in the product information.

6.2. I agree with this criticism. I recommend pursuant to Section 25(2) of the Coroner's Act that the manufacturers of these medications review their product information with a view to providing a much clearer warning to clinicians about the interactions between these drugs, and that their co-prescription is not recommended.

6.3. Because I am unable to find that heroin played a part in the causation of Mr Taylor's death, I am unable to make recommendations about the security of detained patients at Glenside Hospital.

Key Words: Death in Custody; Medication; Psychiatric/Mental Illness

In witness whereof the said Coroner has hereunto set and subscribed his hand and Seal the 20th day of April, 2004.

Coroner