Coroners Act 1996 [Section 26(1)]



Western

Australia

RECORD OF INVESTIGATION INTO DEATH

Ref No: 13/18

I, Evelyn Felicia Vicker, Deputy State Coroner, having investigated the death of **Stanley KING** with an Inquest held at Perth Coroners Court, Court 51, Central Law Courts, 501 Hay Street, Perth, on 6-9 March 2018 find the identity of the deceased was **Stanley KING** and that death occurred on 22 May 2015 at Rockingham General Hospital as the result of Coronary Artery Disease in a man with Laryngeal Dystonia, Severe Psychosis (treated) and Obesity in the following circumstances:-

Counsel Appearing:

Ms F Allen assisted the Deputy State Coroner

Ms J Hook (State Solicitors Office) appeared on behalf of the South Metropolitan Health Service, Department of Health

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INTRODUCTION

Stanley King (the deceased) was admitted to Mimidi Park, Mental Health Inpatient Unit (Mimidi Park), Rockingham General Hospital (RGH) on 12 May 2015 with a severe relapse of his paranoid schizophrenia. Following assessment by his psychiatrist, Dr Sunny Varghese on 13 May 2015, the deceased was made an involuntary patient under the provisions of the *Mental Health Act 1996* (WA).

During his admission the deceased appeared to stabilise but experienced periods of breathlessness. It was believed he suffered a dystonic reaction to his antipsychotic medication and he was treated with benztropine. He continued to experience difficulties with breathing and all his antipsychotic medication was withheld. He was reviewed during the evening of 21 May 2015 and appeared stable.

In the early hours of 22 May 2015 the deceased was noted to be in distress to the extent he became incontinent. He took himself to the shower and the duty medical officer (DMO) was called. On the attendance of the DMO the deceased walked out of his shower towards the DMO who intended to take him to the treatment room. He collapsed and cardiopulmonary resuscitation (CPR) was commenced and the medical emergency team (MET) were called. Unfortunately the deceased could not be revived despite aggressive resuscitation and was declared deceased shortly thereafter. The deceased was 45 years of age.

By the provisions of the *Coroners Act 1996* (WA) involuntary patients under the *Mental Health Act 1996* (WA) are "persons held in care". This mandates the holding of a public inquest, section 22 (1) (a), to examine the circumstances of the death, and a coroner holding that inquest must comment on the quality, supervision, treatment and care of that person while held in that care.

During the course of the inquest the evidence comprised of the written documentation providing the brief of evidence, Exhibit 1, the oral testimony of a number of persons involved with the deceased around the time of his death, and review by an independent respiratory physician, Dr Quentin Summers.

BACKGROUND

The Deceased

The deceased was born on 18 February 1970 as Robert Stanley James Leahy in Middle Swan, Perth, and was one of three children having a brother and sister. He later had two half-brothers. The deceased's parents separated when he was young and the deceased changed his name by deed poll to Stanley King once he became an adult.¹

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¹ Ex 1, tab 10

The deceased experienced a seizure at six months of age which resulted in a brain injury causing a short attention span, limited literacy skills, anger management issues and a probable intellectual disability.

Due to his early childhood intellectual disability the deceased was known to the disability services and was in receipt of a disability pension. He reported a traumatic childhood for himself and his sister to psychiatric services, with domestic violence issues and a history of depressive illness with self-harm, while his brother had significant drug and alcohol issues.

The deceased met and married a woman suffering from cerebral palsy and intellectual disability in Perth and they had a child. He and his wife moved to Brisbane where they had another three children. All children became wards of the state and the deceased and their mother divorced in 2002, after which the deceased moved back to Perth and lived with his mother.

His mother reported the deceased was initially relatively stable but there were still issues with his behaviour and his violence escalated to the extent he required admission into mental health facilities and fortnightly depot injections.

The deceased first presented with psychotic symptoms in 2008 and was later diagnosed with schizophrenia. He was

cared for by Peel and Rockingham Kwinana Community Mental Health Service (PaRK) and his GP.

The deceased became increasingly angry about his contact with mental health services and was attempting to regain custody of his children, while refusing his depot injections.² He was contesting the Public Trustee's control of his finances.

At the time of his death the deceased was on a disability pension, his finances were controlled by the Public Trustee and he had a medical history of high cholesterol, obesity, sleep apnoea, appendectomy and tonsillectomy and was reported to be allergic to penicillin.

Psychiatric History

Aside from the deceased's early diagnosis of an intellectual disability he also experienced periods of psychosis and, while in Brisbane, he received treatment while an inpatient without difficulty with his medication.³

It is unclear when exactly the deceased was diagnosed with schizophrenia, but in Western Australia he was admitted to Royal Perth Hospital (RPH) in June 2013 with a relapse of psychotic symptoms. He was treated with olanzapine

² Ex 1, tab 15 & 16

³ Ex 1, tab 10 & 4

without issue and he was discharged into the community on the same medications to be managed by PaRK.

The deceased's treating psychiatrist, Dr Sunny Varghese, He had first reviewed the deceased in September 2013. been admitted to RGH ED due to his level of agitation and from the ED was admitted as an involuntary patient to the locked ward at Mimidi Park under the Mental Health Act 1996 on 15 September 2013 due to his aggression and risk of absconding. He had been medication non-compliant and was recommenced on olanzapine tablets, changed to response zuclopenthixol with а better and no extrapyramidal side effects. He progressed well under the altered medication and was discharged back to live with his mother for community follow up. His medication on discharge was zuclopenthixol 1/2 10 mg tablet in the morning, 1 tablet at night, with benztropine 2 mg at night, to prevent side effects to the antipsychotic medication.

He was reviewed on 8 October 2013 when he was resistant to the medication and not happy for follow up in the community.

In June 2014 the deceased presented to the ED with psychotic symptoms. On this occasion he was given Acuphase, zuclopenthixol acetate, a fast acting antipsychotic medication given where patients are aggressive and violent to decrease their level of arousal. The maximum dose is 400 mg which is given over a period of time in three separate doses. It is necessary a patient be monitored when taking Acuphase to ensure they are tolerating the drug adequately. He was also provided with intramuscular (IM) clonazepam.

While medicated the deceased's mental state improved as did his vital signs and at the conclusion of the course of Acuphase it was decided to commence the deceased on zuclopenthixol depot injections. Although he had improved, the deceased was still aggressive and managed on the closed His ongoing olanzapine was ceased and oral ward. zuclopenthixol commenced. The deceased was given open ward access but continued to have low grade, grandiose, He appeared more settled. paranoid delusions. His zuclopenthixol was increased for the night dose, but due to the return of extrapyramidal side effects comprising gait change, slurring of speech and changes in voice quality, it was reduced to 10 mg per day. The deceased was recommenced on benztropine 2 mg bd.⁴

The deceased was commenced on his zuclopenthixol depot, 100 mg IM every two weeks, and was discharged back into the community on a community treatment order (CTO) on 14 July 2014 to live with his mother.

⁴ Ex 1, tab 14 & 18

When in the community the deceased was treated by the community mental health staff and generally managed well on his depot injections. The deceased was attempting to regain custody of his children and his finances from the Public Trustee. He would become agitated with the process and start to be noncompliant with his medications. His depot zuclopenthixol was increased and he was advised to zuclopenthixol, reduce his oral but continue with benztropine. The deceased remained compliant with his medication regime through August to November 2014 with regular reviews which indicated he was doing well, his mental state was stable with no ongoing psychotic symptoms.⁵

December 2014 the deceased ceased his In depot medication and by January 2015 his mother contacted PaRK concerned the deceased was no longer prepared to take medication. He attended a CTO appointment on 19 January 2015 when he was reviewed by Dr Varghese and a psychiatric registrar. It was noted he had ongoing psychotic symptoms, was mildly agitated, had little rapport and underlying irritability. He had persecutory and grandiose delusions and denied any imminent danger to himself or others. He did not wish to continue with oral He continued to be angry with the Public medication. Trustee and have delusions about property. He refused to

⁵ Ex 1, tab 14

have depot injections voluntarily and was continued on a CTO, however, refused depot injections.

The deceased was not breached and was reviewed through his GP who felt he was not in need of the depot injections and did not give them.⁶

The deceased continued to be reviewed and because he seemed to be reasonably stable his CTO was allowed to lapse in March 2015. His mother understood she could contact PaRK if she needed. The deceased continued to appear to be relatively stable.

The deceased presented to Fiona Stanley Hospital (FSH) on 23 April 2015 complaining of chest pain. Following investigations it was diagnosed as anxiety due to State Administrative Tribunal (SAT) hearings relating to his children and finances controlled by the Public Trustee. Dr Varghese was required to provide a report as to the deceased's mental health for the purposes of the SAT hearing.

By May 2015 the deceased was experiencing stressors as a result of his delusions and the SAT hearings. He continued to be noncompliant with antipsychotic medication.

The deceased began to relapse.

⁶ Ex 1, tab 15

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FINAL ADMISSION

On 12 May 2015 the deceased presented to PaRK in a highly agitated state. He was very angry with Dr Varghese for the report he had been required to write for the SAT hearing. The deceased was threatening both Dr Varghese and the clinic staff. He had been noncompliant with medication for some time and was having a relapse of his psychotic symptoms.⁷

As a result of the deceased's behaviour in PaRK reception the police were called and the deceased was taken to RGH ED under the *Mental Health Act 1996*.

In the ED the deceased was reviewed by the medical team and a consultant psychiatrist. He was very aggressive and was given IM injections of olanzapine and lorazepam, a fast acting benzodiazepine. He was also provided with 100 mg of Acuphase IM. He remained in the ED through 13 May 2015 until he was cleared by the medical team for transfer to the locked ward at Mimidi Park.

On his admission to Mimidi Park the deceased was reviewed by Dr Varghese and Dr Law at approximately 3.30 pm on 14 May 2015. Although sedated he was still very agitated, with slurred speech, and threatening towards Dr Varghese which led to him being restrained by security staff. Dr Varghese diagnosed the deceased with a relapse of his schizophrenia

⁷ t 07.03.18, p79

on a background of intellectual impairment.⁸ He admitted the deceased as an involuntary patient.

Dr Law recorded the initial management plan to continue with the two additional doses of Acuphase and provide the deceased with regular lorazepam morning and night. His medical review on admission to Mimidi Park was delayed until he had settled. Dr Law asked for an ECG and noted his bloods were relatively normal. Dr Law also documented the deceased was allergic to penicillin in the admission notes, adverse responses medication chart.⁹

Dr Sekhon, Consultant Psychiatrist, reviewed the deceased on 15 May 2015 when he was provided with his second Acuphase injection. He was also offered olanzapine orally which he refused. He was provided with the third and final Acuphase on 17 May 2015.

The deceased was next reviewed by Dr Varghese on 18 May 2015. On this occasion he was cooperative and appeared settled. While still not happy with Dr Varghese's report to SAT, he was pleasant and polite, although it was clear he was having hallucinations with respect to seeing ants and property ownership. He had been provided with olanzapine in the morning and following Dr Varghese's review his olanzapine was ceased and changed to oral zuclopenthixol at night. He was also provided with chlorpromazine at

⁸ Ex 1, tab 24

⁹ t 06.03.18, p 12, Ex 1, tab 18

10.25 pm to settle. The deceased did not complain of any difficulties and was not noted to be suffering any respiratory distress.¹⁰

His blood tests were normal and although he was breathing loudly his chest was clear. He was prescribed a salbutamol inhaler to use as needed. There was a suggestion he had asthma.

On 19 May 2015 the deceased remained breathless and the nurses noted he was using his nasal spray.

20 May 2015

During the early hours of 20 May 2015 the deceased complained of breathlessness as a result of a blocked nose and was advised to use his salbutamol inhaler. He was given lorazepam before he settled back to sleep and the nursing staff requested a medical review in the morning.

The deceased was seen at approximately 9.50 am by Dr Varghese and the psychiatric team. He was breathing heavily and was charted for regular benztropine twice daily, to treat possible stridor secondary to a dystonic reaction from the antipsychotic medication he was using. The deceased was concerned about his breathing, however, it was not noted to be due to respiratory distress.

¹⁰ Ex 1, tab 14

The deceased still had his psychotic delusions and was still resistant to depot injections. Although he denied wanting to harm Dr Varghese he still wanted to sue him. Due to his improved behaviour it was suggested he could try open ward access with a one to one nursing special.¹¹

At 1.30 pm the deceased again complained of shortness of breath, but his oxygen saturations were normal and the nursing staff again requested a medical review. This occurred at 4.20 pm with the medical intern. The deceased was noted to have a stridor/wheeze and a chest X-ray, ECG and blood tests were requested. The medical team suggested an asthma prevention medication, but it was never charted. He was again provided with salbutamol, chlorpromazine and lorazepam to settle him for the evening.

At 10.30 pm the nursing staff were concerned because the deceased was distressed, restless and short of breath. He was seeking reassurance. They asked the duty medical officer (DMO) to attend. When contacted the DMO advised he was busy, but would attend as soon as possible.

The duty DMO on night shift overnight 20-21 May 2015 was Dr Oyewopo. He was a psychiatric registrar. Dr Oyewopo recalled the request from the nursing staff on the ward as occurring at approximately 10.30 pm and he attended at 11.30 pm. He noted the deceased's breathing was audible

¹¹ Ex 1, tab 14

on inspiration and was concerned there was a problem in his upper airways.¹²

Dr Oyewopo had called at the nurses station prior to his review to obtain the deceased's history and discovered he was known to have asthma. Dr Oyewopo was also advised the breathing difficulty had been present for some time. Dr Oyewopo was concerned as to whether the difficulty for the deceased was asthmatic, anaphalactic or an extrapyramidal effect of his antipsychotic medication, acute laryngeal dystonia.

Dr Oyewopo advised asthma usually produced difficulty on expiration while an allergy, or anaphylactic reaction, would be immediate rather than having continued for some time with no obvious cause. Dr Oyewopo was concerned he was looking at an asthmatic problem or laryngeal dystonia. Laryngeal dystonia would create difficulty on inspiration and Dr Oyewopo knew the deceased had received Acuphase. Although laryngeal dystonia was a very rare side effect Dr Oyewopo had prior experience with its presentation. This was not the evidence of other clinicians called at inquest.¹³ On examination Dr Oyewopo confirmed the deceased's breathing problem appeared to be in the upper airway and that air into the deceased's lungs was reduced bilaterally.

 $^{^{12}}$ t 06.03.18, p28 – Note the transcript is inaccurate and does not differentiate accurately between inspiration and expiration. 13 t 06.03.18, p29

When asked to review the deceased Dr Oyewopo had been told the deceased had a residue on his tongue, normally a form of infection, but on physical review Dr Oyewopo believed his tongue appeared swollen. Dr Oyewopo suspected it was a protrusion, secondary to the upper respiratory dystonia, rather than an anaphylactic reaction.

The deceased's respiration was elevated at 24 and although his heart rate was slightly elevated it was not of concern. Dr Oyewopo noted the deceased had good oxygen saturation and that his chest appeared clear. He viewed the earlier ECG, which he considered to be normal and that confirmed for him it was likely the deceased was experiencing extrapyramidal side effects of the Acuphase in the form of laryngeal-pharyngeal dystonia.¹⁴

The residue on the deceased's tongue was indicative of oral candida. Dr Oyewopo ordered the deceased be provided with benztropine IM at 2 mg rather than 1 mg. He believed it may be diagnostically useful in that if there was a response to the benztropine it was more likely to be a dystonic rather than anaphylactic reaction.

Dr Oyewopo described that laryngeal dystonia can be quite idiosyncratic in its presentation and is not necessarily dose related. It is a rare condition. Benztropine is the accepted medication and when looking at the deceased's medication

¹⁴ t 06.03.18, p31

chart Dr Oyewopo noted that apart from the zuclopenthixol, the deceased had also had chlorpromazine. He considered the administration of the chlorpromazine medication appeared to correspond with increased distress for the deceased and was at about the time at which he was first asked to review the deceased. He considered 1 mg of benztropine was probably not enough which was why he had prescribed the additional dose of benztropine, and that the chlorpromazine be ceased. He also asked that the deceased's zuclopenthixol be reviewed in the morning by his treating team.

In evidence Dr Oyewopo explained that both chlorpromazine and zuclopenthixol are antipsychotic drugs belonging to the typical or first generation antipsychotics known to cause dystonic reactions. The deceased was due not zuclopenthixol for another 24 hours which meant he would provided with any of the older generation not be antipsychotics prior to his review by the treating team in the morning. Dr Oyewopo did not wish to have a major impact in his mental state treatment.¹⁵

21 May 2015

Dr Oyewopo advised the court he stayed with the deceased for approximately an hour to monitor him and ensure the benztropine provided an effective response to the deceased's respiratory difficulties. It was due to the DMO spending

¹⁵ t 06.03.18, p31~32

time with the deceased his note was not written until after midnight on 21 May 2015. Dr Oyewopo was concerned that if the deceased did not respond to the benztropine it may not be appropriate for him to stay in the mental health unit overnight and he would need more support with his airway and require transfer.¹⁶

After being satisfied the deceased had improved following additional benztropine, Dr Oyewopo left the ward. He was recalled approximately an hour later by the nurse concerned the deceased had again deteriorated and was very agitated.

Dr Oyewopo was concerned the benztropine, although holding the deceased for a while, had not been successful and he knew the deceased would need intensive intervention. He advised the nurse on the telephone that a medical emergency team (MET) call needed to be made and he then attended at the unit again himself.¹⁷

Dr Oyewopo advised that on his return to the unit the deceased's breathing appeared even more laboured than it had previously.¹⁸

On Dr Oyewopo re-attending the unit the MET call had been made and the documentation of events at that stage then belonged to the leader of the MET call as the leading

¹⁶ t 06.03.18, p32

¹⁷ t 06.03.18, p34

¹⁸ t 06.03.18, p33

clinician, so Dr Oyewopo did not document his second attendance at the unit with respect to the deceased.

The MET team lead clinician was the ED registrar, Dr Sally Wickham. Dr Wickham advised the court she had no experience with laryngeal dystonia.¹⁹ It was her perception the deceased was suffering from anaphylaxis due to the extent of the deceased's respiratory distress, or an acute asthmatic attack. The deceased was treated with adrenaline and hydrocortisone which did not have an immediate effect.

It was Dr Oyewopo's recall the medical registrar suspected dystonia and that extra benztropine should be considered, but Dr Wickham did not support that view. Dr Oyewopo, however, was adamant that as far as he could recall, benztropine was administered to the deceased before he was moved to ED. Certainly the ICU registrar, who was present, and consultants later involved with the deceased appeared to believe he had been treated with benztropine while in the mental health unit, although it had not been charted.

It was Dr Oyewopo's recall that after the benztropine had been administered, the deceased started to improve in terms of his breathing, but it was the general consensus it was not safe to keep him in Mimidi Park which did not have the same life support facilities available as did the ED. Due to the fact the Acuphase had been given, which is very long

¹⁹ t 06.03.18, p53

acting, there was a concern he may have another attack and it was decided it would be better he be moved to the ED.²⁰ The deceased was transferred to ED by Dr Wickham who described the deceased as suffering both inspiratory and expiratory stridors.²¹

Dr Wickham advised the inquest she was concerned the way the deceased presented represented a bronchospasm or upper airway problem and that his protruding tongue was an anaphylactic reaction or other unknown reaction. The deceased had been treated for anaphylaxis and asthma and she agreed he improved but she was not certain of the reasons for his improvement. Dr Wickham considered it prudent the deceased be moved to the ED where he could be closely monitored. She did not recall the administration of benztropine.

An X-ray performed in the ED excluded pneumothorax²² and the deceased was only in the ED for an hour before he was transferred to the intensive care unit (ICU) after consultation with the ICU registrar who was part of the MET team. Dr Wickham believed the deceased had improved as a result of the treatment for anaphylaxis and/or asthma.²³

²⁰ t 06.03.18, p35

²¹ t 06.03.18, p48

²² t 06.03.18, p57

²³ t 06.03.18, p51

Once in ICU the deceased did not receive any treatment other than had been administered as part of the MET call prior to his transfer at approximately 2.40 pm.

Dr Oyewopo, at the conclusion of his shift, visited ICU to see how the deceased was presenting. He stated the deceased appeared to be a different person with no breathing difficulties. The change from his presentation in the early hours of that morning was quite dramatic. Dr Oyewopo recalled the deceased as sitting up and having breakfast in bed.

Dr Oyewopo said the deceased was about to be reviewed by the consultant intensivist and Dr Oyewopo discussed with the intensivist the issues to do with the deceased. He advised the intensivists of his experience with laryngeal dystonia and provided them with some recent literature which discussed the misdiagnosis of laryngeal dystonia with Dr Oyewopo anaphylaxis.²⁴ At the time left ICU practitioners were waiting for the results of a tryptase test to determine whether there was an anaphylactic reaction.²⁵

Dr Oyewopo ensured he did a personal handover to the psychiatric team in the morning before he left because he concerned about the laryngeal dystonia was and benztropine. He wanted the team to consider stopping zuclopenthixol providing and new generation а

²⁴ Ex 3 – A & B

²⁵ t 06.03.18, p37

antipsychotic which may not have the same extrapyramidal side effects.²⁶

Dr Ravikiran Sonawane was the consultant intensivist in the ICU on the morning of 21 May 2015. Dr Sonawane advised the inquest he was aware of a possible dystonic reaction from the ICU registrar overnight and at the time Dr Sonawane reviewed the deceased he was fine and all his observations had returned to normal. Dr Sonawane thought that was because the deceased had been treated and that one of the treatments was effective. Dr Sonawane did not believe it was anaphylaxis, there had been an inconclusive tryptase test, and in evidence believed it was probably a dystonic reaction.²⁷

Dr Sonawane advised the ICU is not a good environment for patients generally, especially mental health patients, because it is a chaotic environment due to the monitors and machinery. From ICU's perspective the deceased was well enough to return to the mental health unit and on discussion with the consultant psychiatrist, it was recommended the deceased be returned to the psychiatric unit.²⁸

Dr Law attended ICU in the morning to discuss the deceased's return to Mimidi Park. Prior to attending ICU Dr

²⁶ t 06.03.18, p36

²⁷ t 07.03.18, p64~65, 69

²⁸ t 07.03.18, p77

Law had reviewed a chest X-ray on the computer which she felt showed some consolidation in his lungs and was concerned he may be developing pneumonia. She went to ICU to see how the deceased was in view of her review of that chest X-ray.

Once in ICU Dr Law was advised the deceased was medically cleared and able to return to the ward. She saw he was in the process of transfer before she had an opportunity to go and assess him.²⁹ Dr Law noted there was limited capability in the mental health unit to manage physically unwell patients. She was advised ICU did not believe there was a problem and there was no reason he should stay in ICU.

Dr Law next saw the deceased at approximately 1.20 pm for a physical examination in the treatment room. The deceased said he felt short of breath and was very worried he would end up returning to ICU. She noted he was breathing fast and his breath made a high pitched sound audible from a distance, but was a not a typical wheeze. She described it as an upper airway sound that she had not heard before and has not heard since.³⁰

The deceased's respiratory rate was elevated to 24 and that in conjunction with her preview of the chest X-ray made her concerned there may be a number of issues facing the

²⁹ t 06.03.18, p8

³⁰ t 06.08.18, p22

deceased. Due to her perception the consolidation seen on the X-ray may indicate pneumonia she asked if the medical registrar could come and review the deceased. She also considered there may be an issue with asthma and laryngeal dystonia as separate issues. Dr Law was advised the registrar was busy and he would attend as soon as he could.

Dr Law prescribed the deceased Augmentin duo forte. She cannot explain her prescription of that antibiotic when she should have been aware the deceased was allergic to penicillin as she had written it on the adverse medication chart. She prescribed it for administration at 8.00 am and 8.00 pm. At no time was her prescription questioned despite the nurses administering it having to enter it upon the same medication chart.³¹

She also prescribed a dose of benztropine.

In evidence Dr Law also noted the deceased had been charted for benztropine earlier on 21 May 2015 and she believed that was part of the MET call overnight. This certainly supports the giving of benztropine in the unit prior to transfer to ED, however, it may also relate to the fact Dr Oyewopo wrote his 11.30 pm attendance on 20 May 2015 up after midnight on 21 May 2015.³² Dr Law advised she prescribed the benztropine on his return to the unit

³¹ t 06.03.18, p13

³² t 06.03.18, p15

because she had been advised by ICU they believed one of the problems may be laryngeal dystonia. She also wrote the deceased up for a regular salbutamol inhaler, because that had been perceived as being effective in ED. She described the diagnosis as being unclear as to precisely what was causing the deceased's respiratory problems, especially the high pitched sound. In view of his mixed history it was thought appropriate to try and treat for asthma as well as laryngeal dystonia.³³

Dr Law again reviewed the deceased with Dr Varghese and Dr Ho at approximately 4.00 pm. At that review Dr Varghese prescribed the deceased benztropine IM and advised the deceased all his antipsychotics would be withheld. The deceased had not had zuclopenthixol for that day. The deceased seemed settled enough and it was recommended he could be transferred to the open ward.³⁴ Dr Law felt the deceased was much more settled during that review and she was less anxious about the lack of medical review.³⁵

Dr Law handed over to the overnight psychiatric DMO and asked that he review the deceased sometime during the night.

³³ t 06.03.18, p16 ³⁴ t 06.03.18, p18-19, 23

³⁵ Ex 1, tab 18

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At the conclusion of Dr Law's shift the nursing observations indicate the deceased's respiratory rate had decreased back to normal, his oxygen saturations were normal and he appeared to be more settled. There was no reaction recorded to the Augmentin administered at 2.40 pm.³⁶

The clinical nurse coordinator for the ward afternoon shift on 21 May 2015 was Maeve Morrison. CN Morrison allocated the deceased to herself due to the general concern as to the deceased's presentation and her knowledge of him over many years. Her shift commenced at 1.00 pm and she finished at 9.30 pm.³⁷

CN Morrison confirmed the deceased's vital signs were stable throughout her shift and she had no concerns about his observations. She provided him with Augmentin at 8.00 pm and there appeared to be no adverse reaction. CN Morrison recalled the deceased as being very anxious due to his experience with ICU the previous night and he was worried something would happen to him when he went to sleep. She felt he was reassured by the frequent observations and medical reviews.

CN Morrison noted the deceased had a hoarse voice and complained of a sore throat, but she did not notice anything which she thought could relate to a dystonic reaction. She considered his condition to be the same throughout the

³⁶ Ex 1, tab 18 ³⁷ Ex 1, tab 19

shift. He was anxious and short of breath, but was able to talk and all his observations were stable. She concluded her shift at 9.30 pm shortly after the deceased had been reviewed by Dr Visweswar, the night medical DMO, and noted to be stable.

The psychiatric DMO Dr Law had requested review the deceased during his shift was Dr Zoran Simovik. Dr Simovik was advised the deceased had been provided with Acuphase injections which the treating team believed had caused a dystonic disorder affecting his breathing, but without a definitive diagnosis. He was told the deceased had been provided benztropine and was charted for benztropine IM twice daily, with a request Dr Simovik check on him during his shift to see how he was doing physically, Dr Simovik understood from that history the main concern was the deceased's breathing.³⁸

Dr Simovik attended at the unit at 7.00 pm to review the deceased and described him as very relaxed with a good mental state. Dr Simovik could discern no sign of any mental health disorder and that physically he appeared well. The deceased was breathing, walking, talking and smiling. He described him as very friendly and polite, but he did notice the deceased's voice was husky.³⁹ However, there was no sign of any physical distress. Dr Simovik did not take any observations because they had already been

³⁸ t 07.03.18, p134

³⁹ t 07.03.18, p135

noted and he could see there was no concern with the deceased's observations.⁴⁰ Dr Simovik had not intended to see the deceased again because he knew there was a request for the night medical DMO to provide the deceased with a medical review overnight.

That medical review was undertaken by Dr Chandan Visweswar. He noted in the progress notes the deceased had stable observations when he reviewed him at 9.00 pm and that his chest was clear.⁴¹ Dr Visweswar saw Dr Simovik later as he was leaving the unit and they briefly discussed Mr King to see whether there were any concerns. Dr Simovik was advised the medical registrar had no concerns about the deceased's presentation.⁴²

22 May 2015

The shift ward coordinator overnight was Collier Marimba. He is a registered mental health nurse and although not allocated to the deceased observed him while he was doing routine visual observations on the ward at approximately 1.30 am.⁴³ The deceased was very anxious and not sleeping. He was in the dining room area having sips of water, so RN Marimba went with the deceased into the television lounge to reassure him and comfort him. RN Marimba monitored the deceased's physical observations and then handed the observations to the allocated nurse to

⁴⁰ t 07.03.18, p137

⁴¹ Ex 1, tab 18

⁴² t 07.03.18, p138

⁴³ t 07.03.18, p122

document. He gave the deceased his salbutamol inhaler because he had wheezy breath. RN Marimba observed the deceased became incontinent. RN Marimba asked a nurse to call the DMO to review the deceased, while he cleaned the floor and the deceased took himself to his shower.⁴⁴

DMO Simovik received a call requesting he go to the ward to review the deceased because they were concerned about his breathing. The request was urgent. Dr Simovik stated he was only about 50 metres from the ward so he was there within a minute or so and on his arrival he was told the deceased was in the shower and so Dr Simovik waited outside the deceased's room in the corridor for him to finish his shower.⁴⁵

The recall of RN Marimba and Dr Simovik differ slightly at this point, but really it is so marginal it is not of concern. RN Marimba described the deceased as walking out of his shower into the bedroom with erratic and wheezy breathing, then being escorted into the treatment room where he was met by Dr Simovik.⁴⁶ Dr Simovik's memory is rather that he was present when the deceased came out of his bedroom into the corridor and he looked pale but was not appearing to struggle to breathe although he only observed him for about 5 seconds before the deceased stumbled in the corridor and collapsed.

⁴⁴ t 07.03.18, p123

⁴⁵ t 07.03.18, p140

⁴⁶ t 07.03.18, p125

"He just walked. There was no wheeze." No laboured breathing at all. You know, when people struggle to breathe they use the axillary muscles, or back. You can see they're trying to help. It wasn't the case at all."47

Dr Simovik said that once the deceased collapsed, he and the nurses took the deceased into the treatment room and chest compressions were started immediately when it was obvious the deceased was not breathing or had a pulse. Dr Simovik took over maintaining the deceased's airway and asked a nurse to call a code blue. To Dr Simovik it appeared the deceased had sustained a cardiovascular arrest such as a stroke or a heart attack because the collapse was so sudden and complete.⁴⁸ Dr Simovik was clear CPR was commenced in 5-10 seconds and that the deceased never revived. Whatever had occurred was an immediate fatal event.49

The doctors comprising the MET call in the early hours of 22 May 2015 were Dr Visweswar, Dr Wickham and Dr Ragab. All of whom had seen the deceased before.⁵⁰

The progress notes record the code blue as being called at 1.57 am with a defibrillator being used at 2.00 am. The crash team arrived at 2.07 am and took over with CPR,

⁴⁷ t 07.03.18, p141 ⁴⁸ t 07.03.18, p142

⁴⁹ t 07.03.18, p142~143

⁵⁰ Ex 1, tab 18, 28, 30

oxygen was already in situ. From 2.07 am until 2.41 am aggressive resuscitation was conducted on the deceased, however, there was no response and CPR was ceased at 2.41 am.⁵¹ On her attendance with the MET team Dr Wickham noted she recognised the deceased from the previous morning. She observed he had a swollen tongue and that on the second attempt the ICU registrar had obtained good airway, bilaterally. The deceased remained in asystole and after 40 minutes with no change in his presentation CPR was ceased. She noted that at 2.40 am there was no cardiac output or respiratory effort and that the deceased was left in the treating room in the ward.⁵²

The notes in the integrated progress notes are undecided as to the aetiology of the deceased's sudden collapse. Again there was the query as to whether it was an anaphylactic response or a coronary response with a query as to laryngeal dystonia. In evidence Dr Wickham believed the deceased's final cardiorespiratory collapse was likely to be a combination of factors.⁵³

The reason for the deceased's sudden collapse remained unknown as far as the clinicians were concerned.

Psychiatry did not consider a laryngeal lingual dystonic reaction on the morning of 22 May 2015 was responsible

⁵¹ Ex 1, tab 18

⁵² Ex 1, tab 30

⁵³ t 06.03.18, p53

due to the delay since the deceased had been treated with antipsychotics and his treatment with benztropine.⁵⁴

ED practitioners considered the collapse in the early hours of 22 May 2015 may have been for a different reason than that of 21 May 2015. The nursing staff were all concerned the deceased had been very anxious overnight 21-22 May 2015, despite his observations being within normal limits. The deceased's anxiety was clear and he was very restless.

POST MORTEM EXAMINATION

The post mortem examination of the deceased was undertaken on 28 May 2015 by Dr D Moss, Forensic Pathologist, PathWest Laboratory of Medicine WA on 28 May 2015.⁵⁵

Post mortem examination of the deceased revealed an obese male with focally severe hardening and narrowing of the blood vessels over the surface of the heart (coronary artery atherosclerosis) and an enlarged heart. While generally there was atherosclerosis with mild stenosis, there were focally severe areas of stenosis of approximately 75% occlusion within the mid portion of the left anterior descending (LAD) branch of the left coronary artery. The liver appeared fatty and the lungs were heavy and fluid laden.

⁵⁴ t Ex 1, tab 14

⁵⁵ Ex 1, tab 7, 8, 9

confirmed focally Histology severe coronary artery atherosclerosis likely fat secondary to and emboli resuscitation attempts in the lungs. Bronchopneumonia was not identified. There were no changes suggestive of acute or even chronic asthma in the lungs. There were fatty changes in the liver with the kidneys showing changes in keeping with hypertension. There was no evidence of acute infarct within the heart.

Microbiology did not reveal specific pathogenic any organisms and there evidence of sepsis. was no Neuropathology revealed a small recent haemorrhage within the body of the corpus callosum, in keeping with a mild concussive head injury.

Toxicology revealed the presence of the antipsychotic medications chlorpromazine, olanzapine and zuclopenthixol although at low levels, confirming they were still present in the deceased's system despite all antipsychotic medication being withdrawn over 24 hours before death. Benztropine was present in the urine, while alcohol and other common drugs were not detected. Tests for anaphylaxis with respect to the administration of penicillin were equivocal and of uncertain significance.

Despite extensive investigation Dr Moss was unable to come to a definitive conclusion regarding the death of the deceased. He noted that while the enlarged heart and focally severe coronary artery atherosclerosis provided a possible explanation for death, they did not adequately explain the circumstances surrounding death, which at the time Dr Moss was investigating the matter indicated a reluctance by some clinicians to accept the deceased's collapse on the morning of 22 May 2015 was triggered by lingual laryngeal dystonia.

Dr Moss believed the issues regarding the deceased's breathing difficulties were not adequately explained at post mortem examination. He considered the issue of upper airway obstruction may be an explanation for death, given the deceased appeared to have had a respiratory arrest as the initiating event of his final collapse and there was no evidence at post mortem examination of asthma.

Dr Moss considered the views of a respiratory physician would be useful regarding the nature of the upper airway problems for the deceased and their potential contribution to death. Dr Moss believed an ICU report covering the previous morning's collapse may provide useful insights.

With respect to the ICU report, the ICU clinicians believed the deceased's death may be due to anaphylaxis, but were more inclined to consider the collapse on 21 May 2015 was due to laryngeal dystonia.⁵⁶ None of the clinicians, other than Dr Oyewopo, were comfortable with laryngeal dystonia

⁵⁶ Ex 1, tab 21

being the trigger for the final collapse on 22 May 2015 due to the withdrawal of all antipsychotic medication and ongoing treatment with anticholinergic therapy.

At the conclusion of the post mortem examination Dr Moss concluded the cause of death for the deceased was unascertained.

Evidence of Dr Summers

The Office of the State Coroner (OSC) sought the opinion of a respiratory physician. Dr Quentin Summers was asked to provide his opinion as to a likely explanation for the death of the deceased in the early hours of 22 May 2015.

Dr Summers noted the deceased's history of severe psychosis required sedation which caused a drop in his oxygen saturations, together with antipsychotic medication which was known to produce extra pyramidal side effects for the deceased. Dr Summers also noted the clinicians dealing with the deceased had noted a change in his voice on occasions when zuclopenthixol doses had been increased. He noted the deceased had been treated for dystonic reaction with benztropine in the past.

With respect to the deceased's admission as an involuntary patient on 14 May 2015 Dr Summers noted the deceased was initially treated with olanzapine which was changed to zuclopenthixol due to side effects. The deceased was treated with benztropine due to a dystonic reaction on a change in medication.

Dr Summers noted the deceased's relapse into respiratory distress in the early hours of 21 May 2015 with an apparently swollen tongue and hypoxic breathing on room air, with a very low oxygen saturation. There had been some discussion there was an anaphylactic reaction, although the source for that was not clear, or laryngeal dystonia. It was the view of the psychiatric clinicians the incident in the early hours of 21 May 2015 was the result of dystonia. The deceased's laryngeal antipsychotic medication was ceased and he had not been provided with antipsychotic medication since the afternoon of 20 May 2015. He was provided with benztropine.

Following the deceased's stay in ICU he was reviewed, appeared well which indicated one of the therapies used appeared to have worked, probably the benztropine, and he was returned to the mental health unit. The intensivists confirmed they believed it was more likely to be a dystonic reaction to antipsychotic medication.

Following the deceased's management with regular benztropine he was reviewed a number of times during 21 May 2015 and appeared to be well although there was note of a high pitched upper airway noise. Dr Summers explained in evidence the high pitched noise described was probably on inspiration which Dr Summers described as being an important distinguishing feature for laryngeal dystonia.

Dr Summers was clear breathing audible on inspiration indicated an upper respiratory tract issue which suggested laryngeal dysfunction.⁵⁷ Dr Summers stated in evidence it was important that people taking observations when there was а concern with extrapyramidal side effects of antipsychotics, note not only the difficulty in breathing, but whether it is on inspiration or expiration because it is diagnostic for upper airway issues and therefore more related to lingual laryngeal dystonia.58

The deceased's improvement after being treated with extra benztropine seemed to have satisfied clinicians the incident in the early hours of 21 May 2015 was due to the extrapyramidal side effects of the antipsychotics in the form of laryngeal dystonia.

The deceased's observations were generally stable but there were frequent notes he had an unusual pitch when breathing and a change in his voice was also noted. The deceased had been reviewed at both 7.00 pm and 9.00 pm with complaint of a sore throat and husky voice but was not in respiratory distress and his oxygen saturations were within normal range.

⁵⁷ t 08.03.18, p160

⁵⁸ t 08.03.18, p169~170

There was an ongoing issue of noisy breathing and a change in voice, however, that was not considered to be of significance until, in the early hours of 22 May 2015, the deceased was restless and very anxious about his breathing. Following a period of incontinence and retreating to the shower, Dr Summers believed that when the deceased came out of the shower he was in respiratory distress. He then suffered a cardiorespiratory arrest, which was fatal.

Dr Summers was quite clear he thought this second episode in the early hours of 22 May 2015 was also the result of dystonic laryngeal and lingual reaction to his drug therapy. Dr Summers did not agree with other clinicians there were additional contributing factors to the episode on 22 May 2015.

Dr Varghese, the deceased's treating psychiatrist, was initially of the view the deceased's second collapse was not due to laryngeal dystonia but after hearing the evidence of Dr Oyewopo, who had experience with laryngeal dystonia and its erratic and sometimes intermittent presentation, as described in the literature, agreed in evidence he now thought the trigger for the final and fatal event for the deceased was laryngeal dystonia.

Dr Summers believed the deceased had been appropriately treated with anticholinergic therapy (benztropine) and noted the deceased's psychosis was severe and it was necessary he be treated with antipsychotics. Dr Summers believed all the deceased's symptoms were consistent with a problem in his upper airway and inconsistent with an anaphylactic reaction. Dr Summers pointed out that treating patients who exhibit extrapyramidal side effects to antipsychotic medication may be very difficult if they have severe psychosis which necessitated antipsychotic treatment.

I was provided with literature outlining antipsychotic drug induced acute laryngeal dystonia by Dr Oyewopo and Dr Varghese. One of the features of the rare cases of life threatening laryngeal dystonia as a result of antipsychotic therapy reflected it frequently occurred when there had been a change or an increase in older generation antipsychotic drug use. The cases were rare and even more rare were cases reported for the newer antipsychotics.⁵⁹

During the course of evidence I put to both Dr Varghese and Summers Dr the extent of the deceased's focal atherosclerotic stenosis in the LAD and his other comorbidities. with the proposition the deceased's underlying comorbidities would have contributed to his death by reducing his ability to compensate for the physiological insult to his system. Generally there was agreement with this proposition and when I proposed a cause of death including the deceased's coronary artery disease, obesity, severe psychosis requiring treatment with antipsychotics triggering a laryngeal dystonic reaction, all clinicians and Dr Moss, as the forensic pathologist were comfortable with that as a reasonable cause of death.⁶⁰

MANNER AND CAUSE OF DEATH

On all the evidence with respect to this matter I am satisfied the deceased was a 45 year old obese male suffering severe psychosis when noncompliant with ongoing medication. This necessitated his treatment with sedatives and antipsychotic medication. He also had underlying moderately severe coronary artery disease.

While medicated the deceased was reasonably able to survive in the community, especially when observed and cared for by his mother. However, the deceased frequently became non-compliant with his medication and became agitated and extremely difficult to manage. As a result of his psychosis he had delusions about his financial capabilities and could become quite aggressive when antagonised.

When he became severely psychotic it was necessary he be sedated and treated with antipsychotics. According to the literature it is often in these circumstances extrapyramidal effects in the form of laryngeal lingual dystonia become apparent and require treatment with anticholinergic therapy such as benztropine. Laryngeal dystonia has been recorded

⁶⁰ Communication from Dr Moss Ex 4, t 09.03.18, p99-100, 181

as being erratic in its responses to intervention, often inconsistent with dosage and may appear intermittently.

This would appear to have been the case for the deceased, who in the early hours of 21st and 22nd May appears to have suffered the onset of serious respiratory difficulties as a result of laryngeal dystonia arising from his necessary sedation and treatment with antipsychotics. Although the antipsychotic medication was ceased when it was recognised there was a problem this does not appear to have resolved the issue due to the erratic nature of his reaction.

The evidence would suggest the deceased suffered ongoing laryngeal dystonia noted as unusual breathing which was not readily identified by those not experienced with laryngeal dystonia. Dr Oyewopo recognised it but his diagnosis remained unconfirmed once the deceased's antipsychotic medication was discontinued and clinicians were concerned there may be other explanations for his ongoing presentation.

I find the deceased was given benztropine in the mental health unit in the early hours of 21 May 2015 when the MET call was made as recalled by Dr Oyewopo and that resulted in his improved presentation while in ED and ICU.

I find the Augmentin administered to the deceased on 21 May 2015 was prescribed and provided in error, but did not cause the deceased to suffer an anaphylactic reaction following its administration. He had not been treated with antibiotics as the time of his first collapse, and his collapse in the early hours of 22 May 2015 was similar to that in the early hours of 21 May 2015 and quite dissimilar to his usual anaphylactic reaction to penicillin.⁶¹

The lack of asthmatic changes at post mortem examination would suggest the deceased's observed ongoing breathing difficulties were due to the extra pyramided effects of his treatment with antipsychotics affecting his upper airways rather than the suspected asthma.

I am satisfied the nature of the deceased's voice change and the inspirational nature of his breathing, while noted, was not fully appreciated as indicating an ongoing laryngeal dystonic reaction. The deceased's system appears to have been able to compensate to some extent until the early hours of 22 May 2015 when he suffered severe respiratory arrest. From my perspective the final difficulty for the deceased was his underlying comorbidities for which his system was no longer able to compensate. He suffered a fatal cardiorespiratory arrest at approximately 2.00 am on the morning of 22 May 2015 and, despite prompt and effective resuscitation, could not be revived.

⁶¹ t 07.03.18, p115

I find the deceased died as the result of his coronary artery disease and severe psychosis necessitating treatment with antipsychotics, the extrapyramidal effects of which required anticholinergic therapy, and his obesity. While use of antipsychotics induced the extrapyramidal effects of lingual laryngeal dystonia, his psychosis was severe and it was necessary he be treated. All his underlying co-morbidities were naturally occurring.

I find death occurred by way of Natural Causes.

CONCLUSION

It is clear that when well the deceased was able to live successfully in the community. Unfortunately his psychosis was severe and as is often the case he had little insight into the ongoing need for appropriate medication compliance. The deceased became noncompliant with his medication while in the community and suffered serious relapse. The extent of his relapse often led to aggressive and violent confrontations requiring significant medication. Unfortunately, as a result of that need for medication the deceased's usually polite and compliant demeanour became completely eroded.

The deceased required admission to facilities capable of monitoring him during these events, however, his development of laryngeal dystonia was erratic and it was unexpected he would continue to suffer, especially with an acute episode, once he had been withdrawn from antipsychotic medication and medicated with benztropine.

I appreciate the occurrence of dystonic laryngeal lingual reactions to antipsychotics to the extent there is a fatal outcome is extremely rare. There are very few cases recorded in the literature overall.

I am unable to determine whether earlier appreciation of the erratic and severe nature of the deceased's laryngeal dystonic reaction by the provision of life support before his final collapse could have prevented his death. In view of the fact he was provided with immediate competent resuscitation it is likely his arrest was unsurvivable in the circumstances in which it occurred. Had he been on life support or given more benztropine prior to arrest the outcome is unknowable.

I am satisfied the deceased's supervision, treatment and care was reasonable in all the circumstances. He was reviewed regularly and appeared to have stabilised. Unlike Dr Oyewopo, who recognised the seriousness of the deceased's secondary decline in the early hours of 21 May 2015, the physicians dealing with the deceased were unprepared for his sudden arrest in the early hours of 22 May 2015. The deceased's underlying co-morbidities interfered with his system's ability to again compensate for further insult and he died. The case of the deceased needs to be used as a learning exercise as to the potential for a fatal outcome with lingual laryngeal dystonic reactions.

RECOMMENDATION

I RECOMMEND THOSE CARING FOR PATIENTS TREATED WITH ANTIPSYCHOTIC MEDICATION BE TRAINED TO RECORD IN THE NOTES WHETHER ANY NOTED BREATHING DIFFICULTY RELATES TO INSPIRATION OR EXPIRATION. THIS MAY PROVIDE A DIAGNOSTIC TOOL IN RECOGNISING THE POTENTIAL FOR LARYNGEAL DYSTONIA AND PROMPT MEDICATION AND INTENSIVE BREATHING SUPPORT PRIOR TO ARREST.

E F Vicker **Deputy State Coroner** 16 July 2018