Coroners Act, 1996
[Section 26(1)]

Western  ***Australia***

RECORD OF INVESTIGATION INTO DEATH

Ref No: 26/14

I, Evelyn Felicia Vicker, Deputy State Coroner, having investigated the death of ***Gillian HANSON****, with an Inquest held at Bunbury Coroners Court, Stephen Street, Bunbury, on 7-9 July 2014 find the identity of the deceased was* ***Gillian HANSON*** *and that death occurred on 20 June 2010 at Bunbury Regional Hospital, in the following circumstances;*

Counsel Appearing :

Ms I Burra-Robinson assisted the Deputy State Coroner

Mr N van Hattem (instructed by the State Solicitors Office) appeared for the WA Country Health Service South West
Ms B Burke (instructed by Australian Nursing Federation) appeared for nurses Angelika Duyn and Christine O'Brien

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INTRODUCTION

Gillian Hanson (the deceased) was an involuntary patient at the Bunbury Regional Hospital Psychiatric Unit on 20 June 2010, when she was located on her ward room floor following a cardiac arrest. She could not be revived.

The deceased was 62 years of age.

The provisions of the *Coroners Act 1996* require the circumstances of any death of an involuntary patient under the *Mental Health Act 1996* be investigated by way of public inquest. (section 3 & 22 (1)(a)) The Coroner inquiring into the death must comment upon the quality of the supervision, treatment and care received by that person while an involuntary patient. (section 25 (3)).

BACKGROUND

The deceased was born on 7 July 1948 in United Kingdom. She migrated to Australia when still a child and later married and had two daughters. She was the widow of a Vietnam Veteran and was supported by a war widow's pension following his death.

The deceased was first referred to a psychiatrist in 1979 in her early 30's when living in Victoria following a period of acute stress. She was diagnosed with chronic schizophrenia with affective symptoms and from that time

required ongoing psychiatric care, with intermittent admissions to hospital when suffering extreme expressions of her illness. She was initially treated with the antipsychotic, stelazine.

At the time of her death the deceased was living in Dunsborough and caring for one of her daughters, who suffered a mild intellectual disability. The deceased's other daughter was a school teacher and required to move to different locations due her employment.

MEDICAL

The deceased appears to have had 2 or 3 general medical practices dealing with her general medical needs in the community. One was Dr Buckeridge of the Cape Naturalist Medical Centre. She also attended the Busselton Medical Centre. She is recorded as suffering hypertension, asthma, stress incontinence, obesity, impaired glucose intolerance and gastro-oesophageal reflux. She did not drink alcohol or use illicit substances, but was a smoker.

In May 1998 the deceased had suffered a life-threatening asthma attack for which she received emergency treatment at Busselton Hospital. She was advised to give up smoking.[1]

[1] Ex 2

Dr Kemp, Consultant Psychiatrist at Bunbury Regional Hospital (BRH), had known of the deceased since at least 2001 when he was treating her then husband for traumatic stress disorder following his experiences in Vietnam. At that stage the deceased's husband was concerned the deceased was noncompliant with her medications and so supervised all her requirements. Dr Kemp stated the deceased's husband died in approximately 2006 and following that time there was evidence the deceased was noncompliant with her medication.

Dr Buckeridge (GP) had seen the deceased since November 2005 and he mainly treated her for her mental health issues whilst in the community in conjunction with her community mental health team and for minor medical ailments. Dr Buckeridge was treating the deceased for hypertension, depression and her psychosis.

In June 2007 Dr Buckeridge noted the deceased recorded a low sodium level (hyponatremia) of 130 (134-146). Dr Buckeridge stopped one of her hypertension medications because he knew it could exacerbate hyponatremia.

Later blood tests indicated the deceased's sodium level had returned to the lower range of normal. It was 134 at the last bloods taken by Dr Buckeridge on 4 May 2009.

In 2008 the deceased was admitted to the Bunbury Regional Hospital Psychiatric Unit with acute relapse of her chronic schizophrenia, exacerbated by noncompliance with her medication. She was referred from Busselton Hospital by Dr Large. Her treating psychiatrist believed her admission had been precipitated by conflict with the daughter with whom she lived.

While at BRH the deceased came under the care of Dr John Kemp, Consultant Psychiatrist. She was diagnosed as suffering chronic obstructive airway disease (COAD). Dr Kemp discharged her from hospital on the antipsychotic risperidone, orally.

In the community the deceased was an outpatient of the South West Mental Health Service (SWMHS) and received approximately 6 monthly reviews by the psychiatrist Dr Lanquillion. The community mental health nurse (CMHN) most involved with the deceased was Stephen Hooyberg who effectively became the deceased's case manager in May 2008.

In May 2009 the deceased was again admitted to BRH Psychiatric Unit following noncompliance with her medication. On her subsequent discharge from BRH Dr Kemp placed her on the anti-depressant escitalopram, a selective serotonin reuptake inhibitor (SSRI), as well as daily oral risperidone, and a fortnightly depot injection of

risperidone. The depot injections were to be administered by her CMHN and ensured review and compliance with her medications.

In March 2010 the deceased went for her usual community psychiatric review and was seen by Dr Paul Corrigan, Consultant Psychiatrist. On his review Dr Corrigan noted the deceased's history, and that since her release from BRH in June 2009, she had been treated voluntarily with risperidone, long acting injections 37.5mg fortnightly, risperidone tablets 0.5mg twice daily and escitalopram tablets of 10mg daily. He was aware Dr Buckeridge was the deceased's treating GP.

Dr Corrigan found the deceased's mental health appeared to be stable, although he believed she minimised past symptoms and was guarded and suspicious in the interview situation. The deceased denied any delusions, but admitted to some auditory hallucinations, which she said did not distress her. Dr Corrigan found she had limited insight into her illness but was willing to continue with treatment. He noted she was compliant and expressed concern about having her regime altered. It was clear she had a rapport with her CMHN, Mr Hooyberg, and trusted him to monitor her effectively in the community.[2]

[2] Ex 1, tab 7

Following introduction of the regular depot medication in May 2009 the deceased appears to have remained stable, until a review by a CMHN on 16 June 2010. On that date her usual CMHN, Mr Hooyberg, was on leave and she was visited by another CMHN, David Gough.

Mr Gough visited the deceased to provide her depot medication, however, she became angry and refused to allow him into the house. Her refusal was discussed at a team meeting. Mr Gough contacted her GP, Dr Buckeridge and asked Dr Buckeridge to speak with the deceased in an attempt to persuade her she should continue with her depot medication.

In response to CMNH Gough's request Dr Buckeridge telephoned the deceased and offered to give the depot injection himself instead. The deceased refused the injection from Dr Buckeridge, or anyone else, and said she did not need injections any more.

In evidence, Dr Buckeridge stated that on his telephoning her the deceased was very dismissive and just wanted to be left alone. She gave the impression over the telephone of being very paranoid and stated she did not believe it was Dr Buckeridge on the telephone. She knew she was talking to a doctor, but actually stated she didn't believe it was Dr Buckeridge. Dr Buckeridge believed that indicated she

was quite paranoid and psychotic.[3] Given Dr Buckeridge usually had reasonable rapport with the deceased he considered it likely she had lapsed into one of her previous states of psychosis.

Following Dr Buckeridge's inability to persuade the deceased to accept her depot medication from either him or the CMHNs, the SWMHS received telephone calls from the deceased's daughter, with whom the deceased was living, indicating she had concerns about her mother's mental health.

As a result Dr Corrigan, Mr Gough, and two police officers attended at the deceased's home address late in the afternoon of 18 June 2010 to review the deceased. Dr Corrigan was concerned the deceased would require an involuntary admission to BRH which was why they attended with police.

Dr Corrigan described that visit as being relatively unproductive in obtaining the compliance of the deceased with their suggestions as to how she may be medicated. She refused any of the options provided for taking her medication and said she felt she didn't need to continue with the injections. Dr Corrigan was concerned with her presentation and, as it was Friday afternoon, believed the preferable option, in view of her behaviour towards her

[3] t 8.7.14, p134

daughter, was to provide the deceased with an involuntary hospital admission.[4]

Dr Corrigan's concerns were purely with respect to the deceased's mental health and her apparent paranoia and lack of insight. He said there was nothing in her presentation which indicated the deceased was medically compromised, other than her psychiatric problems.

Dr Corrigan contacted the psychiatric liaison nurse at BRH to arrange for the deceased's admission that day, and he completed the standard referral transfer to mental health inpatient services for her acute problems. Dr Corrigan described the deceased as being emotionally upset and not happy with their presence but not physically agitated in any way, and her physical demeanour being more or less normal.

BUNBURY REGIONAL HOSPITAL (BRH)

The deceased was admitted to the inpatient psychiatric unit at 5:10pm on Friday 18 June 2010, again under the care of consultant psychiatrist, Dr Kemp. She was admitted to the ward at 6:30pm and refused a physical examination. The deceased was admitted directly to the ward, rather than going via the emergency department for assessment having been recently assessed by Dr Corrigan. As a result she did

[4] t 7.7.14, p32

not require a physical medical examination in the emergency department nor receive one on her arrival in the psychiatric unit.

Dr Praveena Arora was the psychiatric medical officer on duty on 18 June 2010 and on her attempt to assess the deceased on her admission to the ward she found the deceased to be very uncooperative. The deceased declined to answer direct questions and minimised her symptoms. She denied any psychotic symptoms and was very guarded and suspicious of her whole situation. As a result of her non-cooperation a physical examination was not performed due to a concern it would stress her unnecessarily.

Dr Arora described the deceased as quite angry at being in the hospital and as verbally abusive and paranoid. She described how the deceased refused all treatment and refused to talk to the doctor or respond to any questions when Dr Arora tried to question her about her medical history.[5] Her refusal was so complete that when the doctor asked her to poke out her tongue to enable the doctor to assess dehydration, she refused. Dr Arora thought the deceased's presentation was consistent with her underlying diagnosis of paranoid schizophrenia.

Dr Arora agreed it was usual to give all patients entering a psychiatric unit a thorough physical medical examination to

[5] t 8.7.14, p102

rule out any clinical problems which may be exacerbating mental health issues. She stated patients who were non-compliant would usually settle within a day or two, especially if a rapport was established with the treating nurses, who would then persuade a patient to comply. Consequently, it was believed on the evening of her admission it would be better to leave the deceased for a while, hoping she would become more compliant and cooperative with treating staff over the weekend.

Dr Arora stated there was nothing about the deceased's presentation to make her think the deceased required urgent medical review.

The deceased was reviewed by Dr Kemp at 9:15am on 19 June 2010. Dr Kemp performed a basic mental state examination and found the deceased was still not cooperative. His examination was based on observations and interactions with the deceased.

The deceased denied auditory hallucinations and stated she had been cured of her psychiatric illness and no longer required medications. Dr Kemp found her to have marked paranoid ideation and she did not believe Dr Kemp was her treating psychiatrist.

The deceased had a good grasp of where she was. She was orientated to time, person and place but appeared to be

suffering from chronic schizophrenia with worsening symptoms and lacked insight as to the need for her medication. Dr Kemp asked for simple blood and urine tests to be performed which may reveal other sources for her deterioration, but the deceased was uncooperative and refused to provide any assistance. He found her behaviour and speech to be disjointed and disorganised. The blood finger prick test was the only physical examination the deceased permitted at the cajoling of the nurses.

The deceased was prescribed her usual mediations of risperidone, escitalopram, diltiazem, irbesartan, glucosamine and multivitamins. An acceptable finger glucose reading of 5.3 was obtained and her urine analysis was normal.

Dr Kemp admitted the deceased under the *Mental Health Act 1996* so he could treat her with the recommencement of her antipsychotic depot medication. The intention was to perform a physical examination with full blood and urine tests once she became more settled. Dr Kemp arranged for the deceased's risperidone depot injection to be given to her later that day.

Through the rest of 19 June 2010 the deceased was cared for by Registered Mental Health Nurse Christine O'Brien (RMHN O'Brien) who noted the deceased to be drinking

water excessively. At 7:30pm the deceased vomited a large amount of water and appeared breathless and upset.

RMHN O'Brien found the deceased sitting in her room at approximately 8 o'clock and discussed a shower. The deceased refused.

An hour later RMHN O'Brien re-entered the room to take the deceased's observations and noted the deceased was attempting to change her clothes and was unsteady on her feet. The deceased had again vomited watery fluid and been incontinent of urine and faeces while sitting on the chair in the bedroom.

The deceased's recorded observations were temperature 36.5°C, pulse 80bpm, respiration rapid, but a formal rate was not taken, and her oxygen saturation was 98%. As a result of her incontinence the deceased was persuaded to take a shower. She was assisted in the shower by Nurses Duyn and O'Brien, who noted her to be leaning to the right side, but without reported weakness.[6] She was resistant to the nurses' attempts to help her.

RMHN O'Brien telephoned the on call medical officer, Dr Arora, and was advised to *"give the deceased maxolon if needed to settle her vomiting and to call on the medical registrar if she continues to vomit or become unwell".* The

[6] t 7.7.14 p88

permissibility of maxolon was written on the front of the medication chart, but was not given to the deceased because, when RMHN O'Brien went to check on her, she found the deceased to be asleep and decided it was preferable she be allowed to rest.[7]

In evidence, Dr Arora did not recall being told the deceased had been drinking a large amount of water prior to vomiting,[8] but agreed if the nurse's contemporaneous note[9] indicated that was a concern it was likely she had been told. Dr Arora believed had she registered the situation with the deceased's excessive water drinking she would have asked the nurses to attempt to restrict the drinking because it may be a symptom of her mental illness. Dr Arora explained people with schizophrenia can drink a lot of water and become water intoxicated.

Registered Nurse Duyn (RN Duyn) continued with the observations later that night. At midnight on 19 June 2010 RN Duyn noticed the deceased to be sitting on her bed and she appeared quite calm. RN Duyn described how the deceased appeared to be moving her hands in front of her, but not in any way which alarmed the nurse. The nurse did not enter the room due to the deceased's previous resistance to interaction and she did not wish to upset her because she

[7] t 7.7.14, p90
[8] t 8.7.14, p106
[9] Ex 1, tab 11

appeared settled.[10] Both nurses had experience with the deceased from prior admissions.

The deceased was on routine hourly check, as were the other patients in the ward, and RN Duyn again went to assess the deceased at approximately 1:00am. On this occasion the nurse noticed the deceased was not on her bed, however, the light was on in the bathroom with the bathroom door closed. RN Duyn went into the room to check on the deceased and found her lying on the floor between the head of the bed and the bedside table with her head leaning against the side of the table.[11] RN Duyn went to assist the deceased because she thought she had fallen out of bed and asked the deceased to look at her but received no response.

RN Duyn touched the deceased. She felt warm but due to her position RN Duyn was not able to check the deceased's pulse. RN Duyn attempted to move the deceased and thought she heard a breath so she called for assistance and moved the deceased onto the mattress on the floor to assess her. RN Duyn checked the deceased's mouth and on hearing a faint groan placed her in the recovery position on her right hand side. RN Duyn went to collect a crash cart whilst someone else called a code blue. Full resuscitation was commenced on the deceased but was not successful and she died at approximately 1:37am on 20 June 2010.

[10] Ex 1, tab 12, t 7.7.14, p18
[11] Ex 1, Tab 12

A blood sample was taken from the deceased at the time of her arrest and before the commencement of IV fluid resuscitation.

The report on that analysis indicates that blood taken at 1:15am on 20 June 2010 recorded a sodium level of 114mmol/L, described by Dr Corrigan as very low[12] and a level at which he would expect symptoms quite quickly. Dr Corrigan mentioned he believed cerebral oedema could become quite severe at those levels.

POST MORTEM EXAMINATION[13]

A post mortem examination was carried out by Dr Clive Cooke, Chief Forensic Pathologist at PathWest, on 22 June 2010.

At that post mortem examination Dr Cooke noted two main findings;

1. Congestion and increased expansion of the lungs, with increased mucus in the airways raising the possibility of bronchial asthma and;
2. Early arteriosclerosis of the arteries with mild narrowing.

[12] t 7.7.14, p49
[13] Ex 1, Tab 13

Dr Cooke clarified those findings by way of microscopy which showed widespread aspiration of vomit into the small airways in the lungs, with increased mucus raising the likelihood of terminal asthma.[14] The heart muscle showed focal scarring as may occur with coronary arteriosclerosis.[15]

The additional investigations Dr Cooke had requested were microbiology, neuropathology and toxicology. The deceased's blood sugar level was not raised indicating her diabetes was not an issue and overall, from the post mortem findings alone, Dr Cooke considered there was not enough information upon which to make a definitive determination of the cause of death. It was left as unascertained.

In evidence Dr Cooke expanded upon his post mortem investigations. Essentially the microbiology showed mixed bacteria but did not identify any specific infection which could be related to her death and the tests for viral infections were negative.

Toxicology with respect to the deceased reflected the deceased's known illnesses and resuscitation attempts. All the deceased's prescribed medications were at recognised therapeutic levels, and her escitalopram was reflected in the toxicology by a therapeutic level of citalopram.[16]

[14] t 8.7.14 p118-119
[15] t 8.7.14, p120
[16] t 8.7.14 p120

Neuropathology showed no significant abnormalities with respect to the deceased's brain at the time of death and Dr Cooke noted Dr Fabian had not commented upon any swelling of the brain (cerebral oedema), but thought the likely explanation was that oedema was a very common change seen in most post mortem brains and no longer seemed as significant as a phenomenon. He stated he also had not noticed any brain swelling at the post mortem examination and he believed it was something pathologists were used to seeing all the time, and was essentially a normal post mortem brain.[17]

There were two areas with respect to the histology of the deceased's tissues which were relevant to an overall consideration of the deceased's likely cause of death. Dr Cooke referred to the deceased's lung tissues and her heart tissues.

With respect to the deceased's heart Dr Cooke indicated that although the deceased had some early coronary artery disease by way of atherosclerosis, he considered that to be mild and fairly reasonable for a middle aged woman with the history of the deceased. Dr Cooke was certainly not of the view the deceased's mild coronary artery disease, with microscopic scarring in the heart muscle, would be enough to explain the deceased dying as a result of a heart attack or

[17] t 8.7.14 p122-123

coronary artery disease based on the degree of coronary artery narrowing.

In the event the deceased had suffered a heart attack or cardiac arrest, there would have needed to be a reason for that to happen rather than the mild degree of disease observable at microscopy. Dr Cooke did not believe it to be a cardiac event on the basis of coronary artery disease.

Taking into account the deceased's toxicology and mild cardiac disease, Dr Cooke agreed it can be a very rare side effect of citalopram (SSRI medication) to affect the electrical conduction of the heart, by prolonging the QT interval of the electrocardiogram causing an abnormal heart rhythm, which can be fatal. He considered it to be exceptionally rare where the levels of citalopram appeared to be therapeutic rather than an overdose. Long QT syndrome was not identifiable at post mortem and was not something which could be tested post mortem from the histology or other post mortem investigations.[18]

With respect to the finding of congestion and increased expansion of the lungs, Dr Cooke observed the deceased had been a heavy smoker and diagnosed with COAD which was common in smokers. Dr Cooke pointed out asthma, from which the deceased had been known to suffer severe attacks much earlier in her life, is a form of airway disease.

[18] t 8.7.14, p120-121

Dr Cooke stated while his gross findings at post mortem examination may have been indicative of bronchial asthma, the microscopic findings were not supportive of asthma being a triggering event for the deceased's death. Dr Cooke said the typical reaction seen in terms of cellular reaction was not present and therefore did not support a fatal asthma attack. Rather it was more likely the deceased aspirated vomit which caused an asthma reaction as a terminal event, rather than a triggering event.

Dr Cooke put it this way *"I think the main final event for her has been aspiration of vomit and then there may have been some mucus accumulate in the airways as a consequence of that in the very final event. And so, yes, you know, in the last few seconds or perhaps minute or so before death, there may have been, but it's very much a terminal event"*[19]

Dr Cooke was referring to a significant difficulty with breathing for the deceased leading up to her death as a result of an asthma attack, and pointing out he believed she would have only had difficulty breathing at the time of death, and that would have been as a result of the aspiration of vomit prior to that difficulty and as a consequence of that.

Dr Cooke was reasonably satisfied asthma had not been a trigger for the deceased's sudden arrest but rather an

[19] t 8.7.14 p118-119

outcome as a result of whatever it was that had triggered her death.

Having discounted findings which Dr Cooke believed supported a final event, but didn't cause it, he then discussed the very low sodium level recorded in the deceased at the time of her collapse. Dr Cooke agreed a sodium level of 114 was profoundly low.[20]

Dr Cooke viewed the deceased's hyponatremia at the time of her collapse as a significant finding and did not find any other explanation for the deceased's death to be satisfactory.[21]

Dr Cooke indicated his reservation in not confirming hyponatremia on the post mortem investigation report was the hesitation he had with the lack of a specific finding of brain swelling, usually associated with hyponatremia and resulting in seizure.

Dr Cooke was not alone in talking about sodium levels of 114 being extremely low. Dr Corrigan, in evidence, stated that once sodium levels dropped below 120mmol/L one would expect to see symptoms of a low sodium level which he categorised as a deteriorating level of consciousness, confusion, and then progressively becoming worse. He

[20] t 8.7.14 p121
[21] t 8.7.14, p123

included unconsciousness and seizures as a very serious sign and agreed that 114mmol/L was *"very low"*[22]

Dr Kemp discussed low levels of sodium (hyponatremia) and some symptoms one would expect to see including nausea, dizziness and light headedness. Dr Kemp agreed with Dr Cooke that the sodium level of 114mmol/L taken from the deceased at the time of her collapse, before the administration of IV fluids, was likely to reflect a true reading.[23]

Dr Arora, who since 2010 has worked in other facilities as a psychiatric registrar, was clear a sodium level of 114 *"is really quite low, actually"*.[24] Specifically, Dr Arora, who has been used to prescribing antipsychotic and antidepressant type medications in the elderly, continued with this comment;

"*– and she's on escitalopram, which is an SSRI and that can cause hyponatremia, but on top of that if she was drinking a lot of water she would have passed a lot of sodium and plus she vomited, so it would have dropped her sodium levels further. So, yes, that might have contributed too."*[25]

[22] t 7.7.14 p49
[23] t 7.7.14, p65
[24] t 8.7.14 p109
[25] t 8.7.14 p109

Dr Arora was then asked if the excessive water drinking was a symptom of hyponatremia or a cause, and her response was it could be both.

"And usually it's more common in the elderly, so – like, where I work now, it's an elderly unit, so we're very cautious about starting SSRIs in people who are 65 plus. However, like, you know, I wasn't worried about the SSRIs when she came into hospital because I assumed that she was on them for a while. And when you're on them for a while then the body kind of adjusts the sodium levels, so I didn't think – but I think the vomiting and the drinking water might have contributed and she may have had a seizure".

Dr Buckeridge, one of the deceased's treating GP's, but the one apparently most involved with her mental health issues, while indicating he was not aware of a connection between SSRI medication and hyponatremia, was certainly of the view a sodium level of 114mmol/L was hyponatremia at a very serious level. He indicated "*I've never seen that or heard of that in my life*".[26]

While Dr Buckeridge agreed a level of 114mmol/L sodium was extremely low, he believed in the deceased's case, it was more related to factors other than her SSRI medication. Dr Buckeridge was familiar with hypertensive medications causing hyponatremia. He had been very alert to a difficulty

[26] t 8.7.14 p129

for the deceased with hyponatremia in the past and had discontinued one of her medications when he was concerned about her sodium levels. Following the discontinuance of one of her hypertensive medications the deceased's sodium levels had returned to the low normal range.

Dr Buckeridge, with his psychiatric patients, did do routine blood tests which would normally cover electrolyte levels. In addition, he would certainly include electrolyte levels in any blood test referred where the patient was talking about signs he considered may indicate a level of hyponatremia, such as confusion, nausea and light headedness.

The overall impression from the post mortem evidence as a whole indicates the deceased died as the result of hyponatremia, causing a seizure, during which the deceased aspirated vomit and died. If there was a seizure it was unwitnessed, but would account for the post mortem findings and the known hyponatremia at the time of her collapse.

The reasons for the hyponatremia would appear to be a combination of factors, specific to the deceased, at that particular point in time. Her levels of confusion and irritability in the days leading up to her admission to BRH Psychiatric Unit were symptoms she usually exhibited during one of her periods of approaching serious

unwellness. They are also symptoms which may relate to a low level of hyponatremia.

In addition, all practitioners advised that excessive drinking of water can be a factor of a psychosis, and may also be a symptom of, and cause for, hyponatremia. The fact the deceased was drinking excessive amounts of water and vomiting, whether related to her psychosis or not, could also account for lower levels of sodium in her system at that time, if her kidneys were not able to excrete the water rapidly enough. It therefore seems likely there were a number of contributing factors to the deceased's hyponatremia at that point in time, possibly including her SSRI medication, which is known to contribute to hyponatremia.[27]

CONCLUSION AS TO THE DEATH OF THE DECEASED

I am satisfied the deceased was a 62 year old lady who had suffered with mental health issues for much of her life. Generally, she had been able to manage her condition with the assistance of treatment and medication.

Following the death of her husband the deceased had some difficulty remaining compliant with her treatment regime and exhibited periods of non-compliance with her

[27] Ex 7 – A Bundle of Literature provided by Dr Corrigan to the Court including two articles discussing the need to be aware of the possibility of hyponatremia when administering SSRI medications.

medication. These frequently resulted in her deterioration and the need for intervention by her CMHS.

At the time of her death the deceased was living independently and caring for one of her daughters, who also suffered medical issues.

During periods in 2008 and 2009 the deceased's functionality had deteriorated which was reflected in difficulty interacting with her daughter. This prompted intervention by her CMHS to the extent of admissions to BRH Psychiatric Unit where she became known to Dr Kemp, Consultant Psychiatrist.

Dr Kemp instituted depot medication for the deceased in an effort to ensure her compliance with her medication. Her depot injections were administered fortnightly by her CMHN.

In June 2010 the deceased's regular CMHN was on leave and his place was taken by another CMHN with whom the deceased was not familiar.

One of the symptoms of the deceased's illness was a level of suspicion and paranoia. When her regular CMHN did not attend to provide her depot injection the deceased became hostile and suspicious. She assumed those attempting to assist her were not her regular clinicians. This extended to

her disbelieving her general practitioner, Dr Buckeridge, when he telephoned her in an attempt to persuade her to take her regular medication.

It was as a combination of the deceased's refusal to take essential medication, the telephone call from her daughter, concerned about her mother's increasing level of unwellness, and in the knowledge noncompliance caused the deceased to become excessively unwell, her CMHS psychiatrist visited the deceased at home. He believed it may be necessary for him to have her admitted to BRH as an involuntary patient for the purposes of administering her medication to enable her to stabilise.

The deceased was openly hostile when Dr Corrigan attempted to persuade her to take her medication and it became necessary she be admitted to BRH as an involuntary patient. Having been assessed by Dr Corrigan the deceased went straight into the BRH Psychiatric Unit. She bypassed the emergency department due to her significant lack of cooperation with those trying to help her.

As a result of the deceased bypassing the emergency department she also bypassed the usual physical medical examination which would have involved regular blood tests and physical review.

It was a Friday evening and some tests would probably not have provided results until Monday. It was hoped the deceased could be settled over the weekend and so provide investigative results in a similar time frame.

Consequently, the deceased was admitted directly to the ward without the usual physical investigations. All those dealing with the deceased were hoping as she became more settled, so she would be more compliant with their attempts to assist her. Dr Arora attempted to review the deceased but was met with a serious lack of cooperation. There was general concern the deceased would become excessively distressed and therefore extremely agitated and unwell with any continued attempts to assess her on admission. She was subjected to usual ward observations on the ward rounds.

The deceased was observed by the nurses and attempts made to have her comply with simple daily living activities, such as showering. The deceased continued to be agitated and the nurses generally left her alone after enquiring as to her welfare. They attempted not to be excessively intrusive as it definitely distressed her.

On the morning of 19 June 2010 the deceased was assessed by Dr Kemp, Consultant Psychiatrist. The deceased remained uncooperative and Dr Kemp confirmed her involuntary status under the *Mental Health Act 1996* which

allowed enforced compliance with the reinstitution of the deceased's medications. One of the interventions which was undertaken against the deceased's will was the administration of her depot medications later that day.

On the evening of 19 June 2010 the deceased was noted to be agitated, drinking excessive amounts of water and vomiting. These are known signs of psychosis.

Due to the deceased's continued excessive drinking of water and vomiting contact was made with Dr Arora by telephone at approximately 10pm. Over the telephone Dr Arora suggested the deceased be provided with an anti-nausea medication and advised the nurses if the symptoms persisted they should contact the emergency department for medical review.

When RMHN O'Brien went to provide the deceased with maxolon she observed the deceased to be asleep. Because the concern had been with the deceased's inability to sleep, the nurse allowed the deceased to sleep rather than administer medication.

At approximately midnight the deceased was observed in her bedroom sitting on her bed, apparently talking to herself, but otherwise appearing calm.

At the next check approximately 50 minutes, later the same nurse found the deceased collapsed and unresponsive. A medical emergency was immediately called but despite appropriate and aggressive resuscitation the deceased could not be revived.

She died in the early hours of 20 June 2010.

On consideration of the post mortem evidence and all the relevant input from Dr Cooke, and all the other doctors called during the course of the inquest I am satisfied the deceased died as a result of hyponatremia, a low sodium level in her blood, at the time of her death. The mechanism of her death is likely to have been by way of seizure, and the cause for the deceased's hyponatremia was likely a combination of the deceased's psychosis and SSRI medication, as exhibited by her excessive fluid intake and vomiting.

I find death arose by way of Natural Causes.

COMMENTS ON THE DECEASED'S SUPERVISION, TREATMENT AND CARE WHILE IN BUNBURY REGIONAL HOSPITAL PSYCHIATRIC UNIT AS AN INVOLUNTARY PATIENT

While the presence of hyponatremia at the time of the deceased's collapse is apparent from the bloods collected at

that time, its cause in this case can be attributed to a range of factors relevant to the deceased.

It was very clear on the whole of the evidence that although hyponatremia is a recognised side effect of some SSRIs, and other medications including some anti-psychotics, it was a relatively rare, although recognised, side effect.[28] Certainly the symptoms of mild hyponatremia would seem to be similar to those exhibited by the deceased when she became unwell in the normal course of events. The deceased had been on escitalopram for a considerable amount of time and her sodium levels, while low, had certainly not fallen outside the normal range before as far as we know.

On an earlier occasion, before the prescribing of escitalopram, when her general practitioner had been concerned as to the deceased's sodium level, he had withdrawn one of her hypertensive medications. This had resulted in a return of her sodium levels to the lower normal range.

There is no doubt the deceased's supervision, treatment and care whilst a voluntary patient in the community was good. She had a CMHN with whom she was comfortable and generally responded to intervention, although, as her GP commented, she could be difficult when becoming unwell.

[28] t 7.7.14, p68

I accept she would have been a difficult patient but she was managed in the community appropriately and, on the whole, functioned relatively well.

On the occasions the deceased deteriorated she was appropriately assessed and admitted to BRH for stabilisation. Usually, this had been as a voluntary patient.

On this occasion I am unclear as to whether the deceased had been compliant with her oral medication up until her refusal of her depot injection on 16 June 2010. From the reports to her clinicians it would seem she probably had not, or was simply becoming unwell. She deteriorated to the extent she refused to believe clinicians were attempting to assist her on 16 June 2010. On that date her normal CMHN did not attend for her depot medication and she was already hostile and suspicious.

The deceased was appropriately assessed in the community by her CMHS and it was decided she should be admitted as an involuntary patient. That appears to be an entirely appropriate outcome for both the deceased and her daughter, who was effectively in her care. It was a Friday afternoon. She was treated very conservatively in an attempt to not further unsettle her.

I do not have an issue with the reasons for not taking bloods from the deceased on her admission to BRH on the

evening of Friday 18 June 2010 in view of her state of mind and the likelihood results would not have been received promptly.

Once the deceased was in BRH as an involuntary patient on 19 June 2010 she was provided with her depot medication, risperidone, and continued with her escitalopram. Apparently both these medications can affect sodium levels although it is more recognised with the SSRI escitalopram.

Over the remainder of the day the deceased remained hostile and uncooperative and later was noted to be drinking excessive amounts of water which resulted in vomiting. These are recognised symptoms of psychosis and known contributors to hyponatremia. A doctor was contacted and anti-nausea medication authorised but not given because the deceased appeared to have settled.

There is no issue with the supervision, treatment and care the deceased received while an involuntary inpatient at BRH Psychiatric Unit.

Both Dr Corrigan and Dr Kemp were of the view it would be prudent to regularly check electrolyte levels in all patients taking SSRI medication. They believed it was done routinely in any event, and would generally be done on a patient's admission to a psychiatric facility.

Dr Buckeridge, as the deceased's GP, considered electrolyte levels would routinely be done as part of an annual review. He did not consider they would need to be done at other times, unless a patient was showing symptoms associated with low sodium levels, such as light headedness, nausea, irritability or confusion.

On all of the evidence in this case it seems most likely the deceased died as a result of a seizure triggered by hyponatremia, possibly contributed to by her SSRI medication in conjunction with her water intake and vomiting. There needs to be an elevated awareness amongst clinicians for the potential for SSRI medications, and indeed some anti-depressant medications, to affect sodium levels.

SSRI medications have generally revolutionised care of depressed patients in recent times. They are becoming widely used as their benefits over some of the older anti-depressants becomes evident. It would be a tragedy if the benefits of medicating patients liable to depressive episodes with SSRI medications were negated by a failure to appreciate a need to monitor sodium levels in appropriate circumstances.

It may be in future, now we appear to have a death related to hyponatremia and SSRI medication, rather than just the fact of hyponatremia; blood tests should be taken as routine

for all new admissions to psychiatric units, however, that is a matter for the chief psychiatrist.

The fact SSRI medication is known to cause hyponatremia may be a reason to consider blood tests for patients with the symptoms the deceased was exhibiting and thought to be solely due to her unwellness.

RECOMMENDATION

I RECOMMEND THAT FOR PATIENTS TREATED WITH SSRI MEDICATION, ESPECIALLY THOSE OUTLINED IN THE LITERATURE AS BEING MORE SUSCEPTIBLE TO HYPONATREMIA, THAT IS FEMALE, MORE ELDERLY, AND WITH OTHER COMORBIDITIES WHICH MAY AFFECT THEIR KIDNEY FUNCTION, THERE BE AN ELEVATED AWARENESS OF THE NEED TO MONITOR FOR HYPONATREMIA.

E F Vicker

DEPUTY STATE CORONER

17 October 2014