QUEENSLAND COURTS

CORONERS COURT OF QUEENSLAND

FINDINGS OF INVESTIGATION

CITATION:	**Non-inquest findings into the death of RR-W**
TITLE OF COURT:	Coroners Court
JURISDICTION:	BRISBANE
DATE:	20/09/2017
FILE NO(s):	2015/4627
FINDINGS OF:	Ainslie Kirkegaard, Coronial Registrar
CATCHWORDS:	CORONERS: elderly patient; SIADH (syndrome of inappropriate anti-diuretic hormone); impact of citaprolam and duloxetine on pre-existing hyponatraemia; management of severe hyponatraemia; hypertonic saline infusion; inter-hospital transfer processes; clinical communication; clinical documentation; recognition and response to clinical deterioration; use of early warning & response observations tools (EWARS)

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Background

RR-W was an 83 year old woman who died at a metropolitan public hospital on 19 November 2015.

RR-W's death was reported to the coroner because she was considered to have died from complications of the antidepressant drug, citalopram.

Mrs R-W's medical history

Review of RR-W's hospital records shows she had a past medical history including atrial fibrillation, chronic obstructive pulmonary disease (bronchiectasis) with asthma and anxiety, falls and frailty.

She had been admitted to a small metropolitan private hospital in 2013 under physician Dr A and rehabilitation physician, Dr R. During this admission she was investigated for hyponatraemia which was found to be due to syndrome of inappropriate anti-diuretic hormone (SIADH).

She was admitted to the same private hospital on Friday, 13 November 2015 under the care of a different physician, Dr H. She had initially presented to a nearby metropolitan public hospital emergency department on 4 November 2015 after a fall at home. She then represented to the metropolitan public hospital on 11 November with non-cardiac chest pain. She was noted on those presentations to have a chronic hyponatraemia (sodium 122-124).

RR-W was noted to be not coping well at home. She lived on her own in a villa in a small complex. Her daughter, who was her primary carer, was overseas at the time.

Urine cultures grew Escherichia coli and she was commenced on antibiotic therapy. Pathology results showed mild hyponatraemia (122mmol/l; normal range 135-145).

The metropolitan public hospital emergency resident referred RR-W to Dr H for admission to the small metropolitan private hospital. Dr H says she asked for a CT head to be done at the public hospital to exclude any reversible causes or mass lesion. This was done and the emergency resident phoned Dr H later that morning to advise the CT head was normal. Dr H then accepted RR-W for admission. She had not previously treated RR-W.

On admission to the private hospital and following review by Dr H the plan was to withhold her diltiazem, cease the antibiotic pending repeat urine microscopy, commence intravenous normal saline 1000ml over 24 hours to treat the hyponatraemia, commence citaprolam 10mg the following morning for depression and for blood tests the following morning. RR-W was noted to be very distressed about becoming unable to cope at home.

She received her first dose of citaprolam at 2:00pm on Saturday 14 November 2015.

The citaprolam was ceased on 15 November after one dose given Dr H's documented concern it "may worsen the hyponatraemia". She was then commenced on duloxetine (30mg) which she received on 16, 17 and 18 November.

Dr H reviewed RR-W at 10:50am on 18 November noting a sodium level of 113mmol/l. She ordered intravenous therapy with normal saline 1 litre over 24 hours, ceasing the duloxetine and haloperidol. RR-W was on fourth hourly neurological observations and hourly urine measures. She was receiving supplemental oxygen via nasal prongs which she at times removed. Once reapplied, her oxygen saturations were maintained between 90-97%.

At 2:00pm, nursing staff documented that RR-W was drowsy throughout the morning and

became difficult to rouse after her shower. Her GSC was 12 and this was escalated to Dr H who was present on the ward at the time. Neurological observations were continued fourth hourly. RR-W maintained a GCS 12.

At 4:20pm, nursing staff documented having contacted Dr H regarding RR-W's hourly urine output measures. Dr H was noted to be happy with the urine output and indicated she was waiting for blood results following a blood test scheduled for 6:00pm. These results returned a sodium level of 111, so Dr H arranged for RR-W to be transferred back to the nearby public hospital emergency department for further monitoring and treatment.

RR-W arrived in the public hospital emergency department at 8:37pm. At triage, the nursing assessment was that her GCS was 10, she was pale, warm to touch and her blood pressure 110 systolic. She was triaged as priority Code 2. When reviewed by a Junior House Officer at 8:50pm, she was considered to have severe hyponatraemia (now 114) to be discussed with the consultant and for her sodium level to be corrected "as per protocol" with hypertonic sodium chloride solution intravenously – this was ordered to be given as 100ml 3% sodium chloride over 10 minutes followed by 566ml 3% sodium chloride at 24ml/hr and her sodium level to be rechecked at one hour after starting initial bolus. She was to remain in the emergency department overnight with repeat sodium at the end of treatment.

The first complete set of observations recorded RR-W was GCS 9 with sluggish pupils and no response to arm and leg movements but responding to pain.

The records indicate Dr H told the emergency consultant she would be contacting the family that night.

At 10:53pm, RR-W's care was handed over to the Night Registrar who documented at 2:53am that Dr H had spoken with the family and advised to continue the sodium chloride infusion.

Her condition deteriorated in the early hours of 19 November with deterioration of her GCS to 4. On review her oxygen saturation had dropped but improved when she was commenced on high flow oxygen.

The following morning, at 7:54am, the Registrar noted RR-W's condition had been discussed with Dr H, she continued to have a GCS 4 and her blood pressure was 114 systolic. The Registrar ordered CT imaging of the head, chest, abdomen and pelvis to investigate possible malignancy, referral to the Medical Registrar to consider admission under the medical team and for the hypertonic saline infusion to continue. The Registrar's notes indicate Dr H was going to speak with the family.

The Emergency Department Registrar completed an Acute Resuscitation Plan having regard to the private hospital Critical Care Plan and discussion with Dr H who advised RR-W was not for resuscitation but for medical treatment. Dr H is noted to have again advised CT imaging of the head, chest, abdomen and pelvis and for the hypertonic saline infusion to continue. It is documented that Dr H also advised that depending on the results, palliation may need to be discussed with the family.

RR-W was referred to the Night Medical Registrar at around this time who after a brief review noted she had deteriorated. That morning her GCS was assessed as ranging from 6-7. She was hypothermic, had an increased respiratory rate and a raised jugular vein pressure. It was thought her deterioration was caused by hyponatraemia (secondary to the syndrome of inappropriate antidiuretic hormone secretion, complication of medication or malignancy) or from correction of hyponatraemia. Her sodium levels had increased from 115mmol/l at 11:45pm the previous evening to 121mmol/l at 8:45am that morning.

She was discussed at medical handover and reviewed at 8:00am by the Director of Medicine and a medical consultant. The medical consultant then spoke with RR-W's son who agreed to a trial of non-invasive ventilation with BiPAP, which commenced at 9:30am. It was decided she should remain in the emergency department as family members were on their way to the hospital to discuss ceiling of care.

After discussion with the family, RR-W was transferred to the Palliative Care team for palliation in accordance with her known wishes. She was admitted to an inpatient ward at midday. Her condition continued to deteriorate and she died at 4:15pm that afternoon.

Preliminary independent clinical review outcomes

Given concerns about the death having been the result of a medication complication, RR-W's clinical management was reviewed by an independent doctor from the Department of Health Clinical Forensic Medicine Unit.

The reviewing doctor noted RR-W's blood results on admission to the small metropolitan private hospital showed her to have low sodium and low osmolality. I note this presentation can be caused by conditions including the syndrome of inappropriate antidiuretic hormone secretion (SIADH), psychogenic polydipsia (drinking too much water), underactive thyroid or adrenal insufficiency. SIADH may have multiple causes, including lung malignancy or lung infection.

The reviewing doctor was concerned that this state (low sodium, low osmolality) existed prior to the prescription of citaprolam, noting this would be a relative contraindication to the antidepressant tablet; so saying the complication of hyponatraemia is relatively rare given how often these drugs are used, though it is more common in the elderly and more common again in those with fluid balance disturbance as in this case.

The reviewing doctor noted that while this complication was recognised for the drug initially prescribed (citalopram), it was not recognised by the prescribing physician that the substitute drug duloxetine actually has the same risk.

The reviewing doctor advised that the transfer of RR-W to the metropolitan public hospital emergency department to facilitate the administration of hypertonic saline was appropriate as complications can arise from such solution (osmotic demyelination). However, the reviewing doctor was concerned about how proactively RR-W was managed in the emergency department overnight. The team of doctors arriving in the morning have provided substantial documentation and their decision making appears clear. RR-W was very unwell at the time of their review and further supportive intervention would have been necessary. Family discussion is well documented and decision making clear and reasonable at this time.

Acknowledging the benefit of hindsight, the reviewing doctor identified three main issues warranting further coronial investigation, namely:

1. the prescription of a drug that may exacerbate existing hyponatraemia (while not causing the initial hyponatraemia this is likely to have caused the severe drop);
2. the documented decline in RR-W's level of consciousness and oxygen saturations with apparent minimal action or associated documentation; and
3. the absence of an earlier management plan around the hyponatraemia (particularly fluid restriction and re-testing)

Having regard to this advice, the then Deputy Registrar declined to accept the proposed cause of death certificate.

Autopsy findings

An external examination and full internal autopsy were performed at the John Tonge Centre on 30 November 2015. The brain was examined by a specialist neuropathologist. The final autopsy report issued on 19 May 2016.

The autopsy revealed brain swelling with bilateral uncal herniation. Having regard to the clinical history of decreasing level of consciousness in the setting of low sodium, the pathologist considered the brain swelling to be consistent with encephalopathy secondary to hyponatraemia, which he considered caused the death. Autopsy did not identify any anatomical cause for the hyponatraemia leading the pathologist to conclude this was likely an adverse effect of citaprolam. There was also moderate coronary atherosclerosis which the pathologist considered potentially contributory to death.

Having considered the autopsy findings, the reviewing doctor observed positive findings that may impact on sodium levels including a scar in the pituitary (thought its location, size and significance are not described in the pathologist's report) and evidence of albeit mild infection in areas of bronchiectasis.

Response from Dr H & the small metropolitan private hospital

Dr H was given an opportunity to respond to the issues identified by the reviewing doctor.

Having considered the autopsy report, Dr H acknowledged that one 10 mg dose of citalopram may have worsened RR-W's pre-existing hyponatraemia. However, she believed it was also possible RR-W experienced severe and unexpected hyponatraemia due to the administration of duloxetine 30 mg in three doses, which did not respond to usual management of hyponatraemia including fluid restriction, slow administration of intravenous normal saline and cessation of duloxetine. She noted this to be a rare and unexpected side effect of duloxetine.

Admission to the small metropolitan private hospital

Dr H advised that RR-W's previous hyponatraemia (attributed to SIADH during a previous admission to the private hospital in 2013) was not known to her as the notes from that admission were not available at the time she admitted RR-W on 13 November 2015 (due to a change in record handling at the hospital). Consequently she did not know that the sodium level of 122 recorded at the metropolitan public hospital that morning was a chronic condition related to SIADH and not an intercurrent one related to the presenting complaint.

At the time of RR-W's admission in November 2015, the private hospital had medical records stored both onsite and at an off-site storage facility. The hospital introduced scanned records in September 2015 - this was implemented by scanning charts only on discharge from that date; there was no back scanning of earlier charts.

The small metropolitan private hospital has since clarified that the process in place at the time of RR-W's admission via the public hospital emergency department during business hours was for the patient to be registered in the hospital's patient management system by the administrative team. In doing so, this team identified whether there were any other volumes of charts that need to go to the ward with the patient and notified the Medical Records team in various ways to arrange for the medical record to be sent to the relevant clinical department.

RR-W's previous records from 2013 were located at an off-site storage facility at the time of her admission. Transaction records from the storage facility indicate that her chart was tracked out from that facility and delivered to the hospital on 19 November 2015.

The hospital's subsequent review of RR-W's admission identified there was no formal or consistent process in place to communicate emergency admissions to the medical records team, resulting in a delay in receiving the additional medical records. I am advised that as at August 2017 the hospital had implemented a formal process to review all medical record requests relating to emergency admissions to it. Additionally, the hospital's Current Clinical Alert form has been updated to indicate what paper records are in existence for the patient. This mechanism was not in place at the time of RR-W's admission.

Review on Saturday 14 November 2015

RR-W's pathology results were not available to Dr H when she reviewed her at 8:40am on Saturday 14 November 2015. At this time, RR-W was afebrile, her breathing was normal and her blood pressure had improved. She was sitting out of bed reading the paper at that time. Dr H explained her plan was for wait for the blood test results, commence the citaprolam 10mg, cease diltiazem and remove the intravenous cannula at RR-W's request.

Review on Sunday 15 November 2015

Dr H reviewed RR-W at 9:25am on Sunday 15 November 2015. Her condition was unchanged and she remained very anxious and depressed. Repeat urine microscopy showed an E.coli urinary tract infection. Dr H says she noted the blood test results from the previous day, in particular the sodium level of 127. Her plan at that stage was to cease the citaprolam, commence trimethoprim and commence duloxetine 30mg from 8:00am the following morning. She says RR-W had not been given her daily citaprolam at the time of this review.

Review on Monday 16 November 2015

Dr H saw RR-W at 1:00pm on Monday 16 November. She recalls her complaining of nausea. RR-W told her she had seen a psychiatrist in the past and had previously taken antidepressant medication but could not recall what it was. Dr H noted her to be anxious and expressing paranoid thoughts. Her plan at that time was to her stat doses of gentamicin (for the urinary tract infection) and esomeprazole, give maxalon for the nausea, commence haloperidol 0.5mg for agitation, recommence intravenous normal saline 100ml over 24 hours and withhold clexane and aspirin (because of possible active dyspepsia). Dr H says she was not told that nursing staff initiated nebulised saline and supplemental oxygen via nasal prongs that night.

Dr H says she ordered bloods tests for the following day but there is no notation to this effect in her entry in the medical record regarding her review of Mrs H that day and nor I have not been provided with a pathology request form prepared by her on that day.

Review on Tuesday 17 November 2015

Dr H says that despite her having ordered bloods to be taken on Tuesday 17 November 2015, no results ever appeared. There is a pathology notation in the records indicating bloods were collected at 6:30am.

She saw RR-W at 2:15pm that afternoon noting she was over-sedated and dry. She did not record a GCS at that time but recalled RR-W was opening her eyes in response to voice, responding verbally to simple questions and obeying commands, this equating (retrospectively) to GCS 13. Her plan was to change the haloperidol to a night oral dose only (as it may have caused over-sedation), change the maxolon to "as needed", given 4% dextrose 1/5th normal saline 1000ml over 12 hours than normal saline 1000ml over 12 hours (to replete her fluid status more rapidly and support her oral intake which was poor) and order bloods to be collected the following morning.

She acknowledges a nursing entry at 8:00pm that evening that she had been asked to be informed of observations outside the normal range.

The hospital subsequently investigated what happened with the pathology results and clarified that the issue was not a missing blood sample from 17 November but rather an incorrect entry about blood collection on 17 November in the wrong patient's notes.

The pathology entry in RR-W's notes at 6:30am on 17 November appears to read "Blood collected INR" – the hospital suggests it is unlikely she would have an INR collected as she was not prescribed anticoagulants. For this reason the hospital considered this is not a lost specimen but rather an entry by a phlebotomist in the wrong patient's notes. Another patient had an INR collected at that time on that day which was received by the hospital from the pathology laboratory. However, the blood collection was not recorded in her notes until 8:30am that morning after that patient had another specimen ordered. The hospital suggests this may have been a clerical error by the phlebotomist who realised her error after the collection from the other patient at 8:30am and recorded the earlier collection in that patient's notes but did not correct her entry in RR-W's notes.

I have been provided with a copy of the pathology request form for the specimen collected from RR-W on 18 November – it was completed on 17 November and there is no time on the form but from the clinical notes, Dr H attended RR-W in the afternoon. The hospital suggests it is possible RR-W just missed the afternoon phlebotomy round. The usual practice at that time was for pathology requests not marked "urgent" to be held over to the next day if they missed the afternoon phlebotomy round.

Review on Wednesday 18 November 2015

Dr H notes a nursing entry at 7:00am that RR-W was muttering constantly and her oxygen saturation had dropped to 76% on room air. Observations taken at 7:20am did not record a respiratory rate though her oxygen saturation was 90% on 4L supplemental oxygen via nasal prongs. Her blood pressure was 140/70, heart rate ~60 beats per minute, temperature ~35 degrees Celsius and conscious state 'alert'.

The hospital has since interviewed the Registered Nurse caring for RR-W at that time. She says RR-W had removed the nasal prongs and when she reapplied them, the oxygen saturations went to 90% straight away. The nurse was counselled about this being a significant decrease in saturations which should have been plotted on the early warning and response observation tool (Q-ADDS) which would have prompted the activation of a Medical Emergency team call. The nurse has since been asked to complete a Reflective Practice Tool pertaining to the signs and symptoms of hyponatraemia, escalation of clinical deterioration to Visiting Medical Officers and documentation of observations on the Q-ADDS.

I am advised that the Chief Executive Officer & Chief Medical Officer of the hospital group has since issued an alert to all staff and Visiting Medical Officers advising that a Medical Emergency Team call must be activated if a patient meets MET call criteria. This is being monitored through the Clinical Safety Systems Audit and the Patient Bedside Safety Audit. The inpatient areas also conduct twice daily "Buzz Meeting" where clinical risks including deteriorating patients are highlighted to all members of the treating team so that appropriate morning and escalation can occur.

Dr H observed that no observations were recorded on the Q-ADDS form for RR-W between 7:20am and 6:00pm that day, 18 November.

Dr H recalls receiving a phone call from the pathology department at around 10:50am notifying her that RR-W's sodium level was 113. She attended to review RR-W noting her eyes opened

to voice, she was confused but obeyed commands, this equating to a GCS 12. She was normotensive and there was no respiratory distress. Dr H says she tried but was unable to access the hospital electronic record system. RR-W's old paper chart had not reached the ward from storage yet.

Dr H's plan was to give intravenous normal saline over 24 hours, give fully thickened fluid and vitamised food diet only when alert, cease both the duloxetine and haloperidol, commence fourth hourly neurological and general observations, insert an indwelling urinary catheter for hourly urine measures, repeat urine microscopy and biochemistry (the latter at 6:00pm), send urine for osmolality and sodium levels, phone RR-W's son and make both the registered nurse and nursing team leader aware of the plan.

Dr H does not provide any information about what contact, if any, she had with RR-W's son thereafter.

I note that neurological observations were taken at 2:00pm and it was documented in the progress notes that RR-W's saturations were 88% on room air. Dr H was noted to be present on the ward and nursing staff made her aware of her condition at this time.

An indwelling catheter was inserted and hourly measures were monitored. The nursing notes indicate that Dr H was aware of the urine output with the plan to await the blood results at 6:00pm. Observations were undertaken at this time.

The urine sodium level returned at 42 (the lower limit of normal) and the urinary osmolality was inappropriately high at 536, which Dr H felt was in keeping with SIADH.

Dr H was contacted by the nurse at 4:30pm about RR-W's urine output which was between 10ml-35ml/hour. Dr H explained the low urine output was due to low oral intake of water and in keeping with the low normal urine sodium level. She says she elected to continue the current management plan because she feared increasing the rate of intravenous normal saline would cause a more rapid salt load and would precipitate central pontine demyelinosis.

A repeat blood test was taken at 6:05pm, returning a sodium level of 111 (reported at 8:15pm). The level had fallen further without any new precipitant and despite treatment. Dr H suggests at this point, the only treatment she could see that would improve RR-W's condition was intravenous hypertonic saline 3%, a treatment that could not be given on the general medical ward at the small metropolitan private hospital due to the need for close monitoring. This is why she arranged for RR-W to be transferred to the metropolitan public hospital emergency department, something she says she made clear to the emergency department team leader by phone as the reason for the transfer.

Dr H does not provide any information about any contact she had from or with the metropolitan public hospital emergency department team thereafter.

The small metropolitan private hospital has since advised that in response to the failure to record observations on the Q-ADDS form, nursing staff have been informed to document all observations on the Q-ADDS form. A process change has been implemented to ensure all acute patients have a minimum of 4th hourly observations conducted unless otherwise documented by the admitting consultant. The importance of this has been reinforced with staff during recent training.

Interaction with the Metropolitan public hospital clinicians on Thursday 19 November 2015

Dr H recalls receiving a phone call from the Consultant Physician in General Medicine at the metropolitan public hospital during her morning clinic. She says he asked her what she thought he should do for RR-W. She says she was surprised to receive this call as she assumed that treatment with hypertonic saline 3% would have been commenced on transfer to the emergency department. She discussed RR-W's condition with the Consultant Physician and says she reiterated that her intention in transferring RR-W was so she received hypertonic saline.

Meeting with RR-W's son on Thursday 19 November 2015

Dr H recalls RR-W's requested a meeting with her to discuss his mother's care. She met him later that morning. He asked why she had transferred his mother to the public hospital emergency department when she deteriorated instead of providing her with palliative care at the private hospital. She says she explained the transfer was to give RR-W the best chance at life.

Rationale for choice of antidepressant

Dr H explained that when she first reviewed RR-W, she had expressed suicidal ideation and was in significant distress. Consequently treatment of her mental state was a priority in conjunction with treatment of her physical condition.

She has used citaprolam in elderly patients with good effect and in her experience, any adverse reaction (including hyponatraemia) has resolved on cessation of the drug.

She ceased citaprolam on Sunday 15 November with the expectation that the sodium level would continue to become normal. However, she remained concerned about RR-W's mental state and changed the antidepressant medication to duloxetine.

Correction by slow administration of intravenous normal saline was recommenced on Monday 16 November when RR-W complained of nausea, by which time she had ordered citaprolam had been ceased in favour of duloxetine. Dr H would normally have expected this action to correct the sodium level.

Dr H does not consider one 10mg dose of citaprolam on Saturday 14 November to have likely caused the precipitant fall in RR-W's sodium to 113 as recorded in the pathology results on Wednesday 18 November. Rather, she has identified duloxetine as the more likely cause of the fall in sodium level. She suggests this was a rare and catastrophic side effect of an effective, usually well tolerated and widely described anti-depressant medication.

Management of hypoxia

Dr H attributes RR-W's hypoxia, which coincided with her decrease in level of consciousness on Wednesday 18 November, to the side effect of hyponatraemia rather than as a separate respiratory complication.

She notes the fall in oxygen saturation to 76% on room air at 7:00am that morning was not recorded in the observation chart and did not trigger a phone call from the nurse to her.

As soon as she became aware of the change in RR-W's condition that day, she implemented a detailed management plan. She reviewed RR-W and ordered repeat testing of the sodium level that evening.

Management plan for hyponatraemia

Dr H maintains she was mindful that hyponatraemia is a risk for elderly unwell patients and for this reason, in treating her depression and anxiety, she prescribed the lowest dose of a drug which is usually well tolerated, and monitored her sodium levels.

She says she considered SIADH as a differential diagnosis for the hyponatraemia as this is a common cause in elderly patients with acute general medical illnesses. In fact, unbeknownst to Dr H, this had been diagnosed during RR-W's 2013 admission.

She says her management plan was influenced by her thinking that RR-W's dehydration was likely contributing to her hyponatraemia – the urinary sodium level of 42 (where a level less than 40 would indicate dehydration) suggests this was not without basis. She notes the CT brain scan performed prior to her admission to the private hospital found no mass lesion or haemorrhage. Otherwise, RR-W was not on a diuretic, her thyroid function was normal, there were no definite signs of hypocortisolism, a malignancy solid organ tumour was not evidence and there was no acute suppuratives lung condition.

Retesting of serum sodium

Dr H was under the impression that blood was collected from Mrs R-W on Tuesday 17 November, and the result was not made available to her. She says that had the result indicated a fall in sodium she would have expected the pathology department to have notified her (as in fact happened the following day in relation to blood collected that day). She suggests that depending on the result, re-testing of the sodium between the level of 127 on the morning of Saturday 14 November and the level of 113 on the morning of Wednesday 18 November may have led to earlier cessation of duloxetine.

The hospital has since clarified that RR-W did not have blood collected on the morning of 17 November – Dr H completed a pathology request when she reviewed RR-W early that afternoon and the specimen was not collected until the following morning.

Fluid restriction

Dr H explained she did not specifically order a particular fluid restriction because she and the nurses could see and were recording that RR-W's oral intake was poor. There was no suggestion clinically that she was taking in oral fluid that would have exceeded any recommended oral fluid restriction. Dr H did not believe that normal or excessive oral fluid intake was contributory to RR-W's hyponatraemia.

The metropolitan public hospital clinical review outcomes

I have received a statement from the Senior Medical Officer who was responsible for RR-W's care when she was transferred back to the public hospital emergency department on the night of Wednesday 18 November 2015.

The Senior Medical Officer recalled receiving a telephone call from Dr H requesting assistance with RR-W. He was advised she had become acutely unwell with an altered level of consciousness over the preceding hours in the setting of severe hyponatraemia (113) and GCS 10.

The Senior Medical Officer recalls Dr H telling him RR-W had a current Acute Resuscitation Plan in place and that she had had prior discussions with the family regarding limits of care. RR-W was not for intensive care admission and in the event of decline, was not for emergency resuscitation efforts. Dr H requested a trial of hypertonic saline to see if correcting the low

sodium improved RR-W's level of consciousness (as this treatment was not available at the private hospital) and if it did not improve RR-W's condition, she would be accepted back to the private hospital for palliative care.

The Senior Medical Officer recalls the hyponatraemia was thought to be multifactorial – a chronic hyponatraemia possibly worsened by the addition of citaprolam two days earlier. He reviewed RR-W immediately on her transfer to the emergency department and read the private hospital notes and the documented current Acute Resuscitation Plan that accompanied her.

The Senior Medical Officer says he discussed the management plan with both the evening and later the Night Registrars. The plan was to cautiously correct her hyponatraemia according to a recognised Queensland Health protocol (available on Queensland Health website) with an initial bolus and then infusion of hypertonic saline, with the sodium level to be rechecked after the bolus.

He says her initial observations were GCS 10, blood pressure 110 systolic, pulse 80 and oxygen saturation 98%.

He recalls RR-W did not improve with treatment despite her sodium level increasing to 121. He says she had an acute deterioration at 7:30am with GCS falling to 4 and her oxygen saturation dropping to 73% (which responded to high flow oxygen). He says she was referred back to the private hospital for palliation but was not accepted for admission and instead referred to the inpatient medical team and subsequently to the palliative care team.

The Senior Medical Officer recalls that the Night Registrar contacted Dr H on two occasions overnight to confirm that RR-W was not for intensive care or advanced life support. He says these calls were not for specific management advice but were to reconfirm the wishes of the patient and her family. Further he says Dr H did not give any specific instructions regarding the management of the hyponatraemia.

The Senior Medical Officer acknowledges his documentation in the medical record was minimal but noted the more thorough and accurate notes of the Evening and Night Registrars which referenced his involvement and agreement with the management plan.

The Senior Medical Officer advised there was no early referral to the inpatient medical team as there was never an intent to have RR-W admitted to the metropolitan public hospital. Rather, the plan was to have her hyponatraemia corrected and then return her to the small metropolitan private hospital.

The metropolitan public hospital undertook a clinical incident review of the care provided to RR-W over 18-19 November 2015.

The clinical review team:

- acknowledged the management of hyponatraemia is a complex and emergency task – while emergency department staff had previously managed patients with hyponatraemia, RR-W had severe hyponatraemia rarely seen at the metropolitan public hospital emergency department;
- considered RR-W was correctly triaged as a priority code 2 – she was assessed by a medical officer within the recommended 10 minute wait time;
- noted the expectation that patients with hyponatraemia are managed by a Senior Registrar with Consultant or Senior Medical Officer supervision - RR-W was initially assessed and managed by a junior doctor who escalated her care to the consultant emergency physician on shift. Her care than then handed over to a Senior Registrar on the night shift with the same consultant on call that night. Her condition was discussed frequently throughout the night with Dr H;

- was satisfied the emergency consultant and Registrar involved in RR-W's care had enough education, training and experience to manage patients with hyponatraemia, and while the junior doctor had minimal experience in this regard, the emergency consultant was involved in the treatment plan;
- noted severe hyponatraemia has a high mortality rate and is difficult to manage especially when patients have an advance health directive in place limiting treatment;
- noted concerns about communication within the emergency department team working in the afternoon and night shifts. Specifically, the oncoming night shift nurse received inadequate handover from the off-going afternoon shift nurse and was not given sufficient information regarding the reason for RR-W's presentation. This appears to have occurred because the SBAR handover tool was not utilised during handover. More significantly, RR-W's condition was not escalated to the nursing team leader either by nursing or medical staff. This was considered to be an unusual occurrence in this emergency department. Medical and nursing staff involved in RR-W's care were unable to recall why this usually regular communication with the nursing team leader did not occur;
- considered that given the complications that can arise from treating severe hyponatraemia, it was appropriate for RR-W to remain within the emergency department for ongoing monitoring;
- noted the metropolitan public hospital did not have a protocol/procedure for managing hyponatraemia; instead the Medical Officer followed the procedure then currently endorsed for use at another metropolitan public hospital (Hypertonic Saline – sodium chloride – 3% solution Intravenous Therapy for Severe Hyponatraemia – ADULTS LNH 3158). While this procedure was available and used as a guide to determine the initial management plan for RR-W's hyponatraemia, there was minimal documentation around her ongoing management plan, especially around the retesting of sodium levels;
- noted the Emergency Department Queensland Adults Deterioration Detection System (Q-ADDS) chart was commenced by nursing staff:
    - at 9:00pm the Q-ADDS score met E due to RR-W only responding to pain, this requiring the initiation of an emergency department response – the emergency consultant was notified at this time;
    - the next set of observations was performed at 10:15pm at which time the Q-ADDS score had improved to 4 as RR-W was responding to voice;
    - the observations performed at 11:27pm achieved a Q-ADDS score of E – the night Registrar was notified of RR-W's decreased GCS;
    - at 1:13am, the total Q-ADDS score remained at E;
    - the allocated nurse discussed the decreased GCS with the Night Registrar at 1:45am, questioning what the family actually wanted and querying the treatment plan noting there was "no real plan" except for treating the hyponatraemia;
    - at 3:10am the Registrar was notified that RR-W's GCS was 6 – the Registrar spoke with Dr H who advised to continue with the hyponatraemia plan. The nurse also questioned the Acute Resuscitation Plan as it was a copy of that from the small metropolitan private hospital;
    - the next set of observations was performed at 7:00am with the Q-ADDS score remaining at E;
    - documentation at 7:45am from the morning shift nurse indicates the Medical Officer was notified of RR-W's GCS 4 and decrease in oxygen saturation – the Medical Officer advised she was not for further/active treatment.

- was concerned that existing procedures for the measurement and documentation of observations using Q-ADDS and escalation of care for the deteriorating patient were not followed appropriately when escalating RR-W's total Q-ADDS scores as the Registrar was notified instead of the Consultant, and that observations were not consistently records on a frequent basis – it was identified that nursing staff were confused about the actual treatment/management plan for RR-W and through discussions with the Registrar, were advised she was for conservative management. On this basis, nursing staff felt she

did not warrant escalation to the Consultant;
- found there was minimal documentation in the medical record regarding the plan to manage the hyponatraemia or action taken in response to RR-W's clinical deterioration;
- due to the lack of documentation, was unable to determine the degree of involvement by Dr H and the emergency consultant in RR-W's care;
- noted lack of documentation regarding escalation to the emergency Consultant overnight and no entries made by the Consultant – the review team was told the Consultant did review RR-W but considered the lack of documentation of this review made it difficult to support this claim;
- acknowledged the omission of documentation by a Consultant has previously been identified as a concern within the Emergency Department resulting in ongoing work with the Senior Medical Officer group to improve documentation;
- noted documentation suggesting discussions with Dr H on at least three occasions overnight indicating Dr H had consulted on RR-W's management plan, liaised and updated the family about her condition and advised the night Registrar regarding her resuscitation status;
- noted it is common practice for the small metropolitan private hospital patients to be transferred to the metropolitan public hospital emergency department for acute interventions or management overnight and then transfer back in the morning often resulting in these patients having to stay in the emergency department until the private hospital is ready to accept the patient back; and
- considered concerns about the degree of Dr H's involvement in the management plan and liaison with the family given she was not credentialed to practise at the metropolitan public hospital – as such her involvement should have been only to provide advice and information about RR-W's history. Staff involved in her care reported that this frequent consultation with Dr H created confusion among the team and made it difficult to determine a clear and consistent management plan.

The clinical review team concluded:
- it was appropriate for RR-W to remain in the Emergency Department for management of hyponatraemia;
- she should have been referred sooner to the Medical Registrar;
- despite the lack of documentation, Dr H requested that RR-W be treated overnight in the emergency department as there was no hypertonic saline available at the private hospital and that RR-W be transferred back there in the morning for further medical management or palliation depending on her response to the hypertonic saline;
- the metropolitan public hospital emergency department is not an appropriate location to monitor the small metropolitan private hospital patients overnight – it recommended the development of a process to ensure these patients are managed in the same way as all other presenting patients and referred for medical or surgical admission;
- although the Acute Resuscitation Plan was not completed until the morning of 19 November 2015, a copy of RR-W's advance health directive was available within the medical record and referred to by the emergency Consultant; and
- it was not appropriate for the small metropolitan private hospital consultants to set limits of care, treatment/management plan and liaise with the family for patients admitted to the metropolitan public hospital.

The clinical review culminated in the following formal recommendations:
1. To develop and implement mandatory criteria for escalation to a Senior Medical Officer and higher within the Emergency Department

2. To review the inter-hospital transfer process from the small metropolitan private hospital with a process to be endorsed by both facilities – I have since been advised that the small metropolitan private hospital and the metropolitan public hospital conduct monthly liaison committee meetings to discuss operational service related issues such as inter-

hospital transfer issues. The two facilities worked together in the development of a public hospital protocol for the transfer of the private hospital's patients to the public hospital emergency department. I have sighted this protocol and note it incorporates a clear expectation that patients from the small metropolitan private hospital who require further care at a higher clinical services capability level facility should be transferred to a larger private hospital. In relation to those patients requiring emergent transfer to the public hospital, the protocol clearly sets out how the transfer process is to be managed and who has clinical responsibility for the patient once transfer has occurred.

3. To develop a local hyponatraemia procedure

4. To provide education and training to nursing staff about use of the Emergency Q-ADDS tools

5. Case discussion with the Emergency Department for clinical education.

Findings required by the *Coroners Act 2003*, s. 45

Identity of the deceased: [*deidentified for publication*]

How she died:

RR-W died from a rare but recognised complication of antidepressant therapy initially with citaprolam and then duloxetine. Due to a recent change in the management of hospital records at the small metropolitan private hospital, the physician who prescribed these medications, Dr H, was not aware of RR-W's previous admission during which she was investigated for hyponatraemia which was found to be due to syndrome of inappropriate anti-diuretic hormone (SIADH). Consequently Dr H did not recognise the sodium level of 122 recorded at the metropolitan public hospital on admission was a chronic condition related to SIADH.

Dr H has appropriately acknowledged the combined impact of the citaprolam and duloxetine on RR-W's condition. Not appreciating that the hyponatraemia may have been chronic, Dr H was expecting this to correct once she ceased the citaprolam, replaced it with duloxetine and treated RR-W with slow administration of intravenous normal saline. Unfortunately, the hyponatraemia continued due to the unanticipated effect of the duloxetine. Dr H has also appropriately acknowledged that had bloods been tested sooner than 18 November 2015, this may have led to earlier cessation of duloxetine. While the evidence supports a finding that Dr H did not anticipate the duloxetine to affect RR-W's sodium level, I am satisfied she is now very aware of this potential complication.

It is evident that as soon as Dr H was made aware of the worsening hyponatraemia on 18 November 2015 she put a plan in place involving urgent repeat blood tests which confirmed an ongoing process, and she then appropriately arranged for RR-W to be transferred to the metropolitan public hospital emergency department to receive intravenous hypertonic saline.

Aspects of the nursing care provided to RR-W were less than optimal but I am satisfied these deficiencies were not significantly outcome changing for her. The small metropolitan private hospital has recognised these issues and taken appropriate steps to remedy them with its nursing staff.

While I am satisfied RR-W was appropriately treated according to a recognised Queensland Health clinical protocol, the metropolitan public hospital emergency department was not the optimal location in which to monitor her overnight and she would have been better managed as an inpatient, whether at the metropolitan public hospital or elsewhere. Unfortunately, her situation was complicated by poor communication within the team allocated to her in the emergency department about the definitive care plan, and the fact that a non-metropolitan public hospital consultant was the liaison point with the family until the morning of 19 November 2015. I can not help but observe that earlier direct communication by the metropolitan public hospital staff with RR-W's son would have better informed her management. I am satisfied that the metropolitan public hospital and the small metropolitan private hospital have worked together to clarify the process by which the small metropolitan private hospital patients are now managed when they are transferred to the metropolitan public hospital and consider the current process will prevent a recurrence of the confusion that arose in relation to RR-W's management in the metropolitan public hospital emergency department over 18-19 November 2015.

Place of death: A metropolitan public hospital

Date of death: 19 November 2015

Cause of death:

1(a) Hyponatraemic Encephalopathy
1(b) Adverse effects of citaprolam and duloxetine administration
2 Coronary Atherosclerosis

I close the investigation.

Ainslie Kirkegaard
Coronial Registrar
CORONERS COURT OF QUEENSLAND
20 September 2017