MAGISTRATES COURT *of* TASMANIA

CORONIAL DIVISION

Record of Investigation into Death (Without Inquest)

Coroners Act 1995
Coroners Rules 2006
Rule 11

I, Olivia McTaggart, Coroner, having investigated the death of Stewart Williams

Find, pursuant to Section 28(1) of the *Coroners Act* 1995, that

a) The identity of the deceased is Stewart Williams;

b) Mr Williams died in the circumstances set out in this finding;

c) The cause of death was hypoxic/ischaemic brain damage - in turn, caused by blood loss and airway obstruction due to facial fractures resulting from blunt force injury (assault) to the face delivered by another person on 24 February 2019. Inadequate medical treatment contributed to his death; and

d) Mr Williams died on 2 March 2019 at Hobart, Tasmania.

In making the above findings, I have had regard to the evidence gained in the comprehensive investigation into Mr Williams' death. The evidence in this investigation is set out in Annexure 'A' to this finding.

Background

Dr Stewart Williams was born on 9 May 1964 in Devon, United Kingdom and was 54 years of age at the time of his death. He was divorced and lived in South Hobart. Dr Williams worked at the University of Tasmania as a senior lecturer in human geography and environmental planning and held a PhD in this field.

He had been married twice and had three children. Dr Williams and his former partner were separated but shared child care and support for the two younger children.

At times, Dr Williams took on boarders at his residence. On 21 February 2019, three days before Dr Williams was assaulted, Kamruzzaman Shipon and Prince Siddique had commenced renting a room from Dr Williams. Mr Siddique stated that Dr Williams had been very kind to him in the time that he encountered him.

Circumstances surrounding death

Circumstances of Assault

In the early hours of 24 February 2019, Dr Williams was in Mobius nightclub in Despard Street, Hobart with Mr Siddique. Dr Williams was intoxicated.

Around 4.45am, they were both walking up the stairs towards the entrance of the nightclub. As they were on the landing platform halfway up the stairs, one Beau Wayne Kelly and a friend were walking down the stairs. There was a brief verbal altercation between Dr Williams and Mr Kelly which culminated in Mr Kelly saying words similar to *"You want me to fuck you up?"* He then punched Dr Williams once to the face, knocking him unconscious. He fell into a semi-seated position against the wall. Mr Siddique spoke with a crowd controller who rendered first aid. Dr Williams was able to be roused quickly and, at 4.48am, a witness called an ambulance which arrived and transported him to the Royal Hobart Hospital (RHH).

The blow caused orbital fractures (facial bones surrounding the eye). Although it was not recognised by treating doctors until it was too late, he also sustained a posterior epistaxis (bleeding at the rear of the nasal cavity) which caused unstemmed blood loss ultimately resulting in haemorrhagic shock and death in hospital six days later.

It is likely that, if the source of the bleeding had been detected and treated in a timely manner, as it should have been, Mr Williams would have survived. The following section deals with the course of his medical treatment and the deficits which significantly contributed to his death.

Course of medical treatment

An ambulance was called to the scene at 4.48am and was dispatched at 4.52am. Dr Williams arrived at the Department of Emergency Medicine (ED) of the RHH at 5.19am and was taken from the ambulance stretcher at 5.30am.

Ambulance paramedics reported that Dr Williams had a brief loss of consciousness and that there was active epistaxis (bleeding from the nose). Otherwise, they noted that Dr Williams' vital signs were normal and he was lucid and coherent, but affected by alcohol.

At 6.11am Dr Williams was seen by the ED Registrar. At that time, anterior nasal packs (to stem bleeding from the front of the nose) were inserted and a CT scan ordered.

The ED nursing assessment documentation recorded at 7.12am described active epistaxis, a blood pressure of 140/80 mmHg and heart rate of 89 bpm. The ED triage nurse assessment

notes described Dr Williams as "dizzy", his haemoglobin level at 148g/L and continuing epistaxis.

At about this time, an ED nurse attending Dr Williams noted that he spat up blood into an emesis bag despite first aid. This fact was only recorded in a later discharge letter which described that 500mls of blood, a significant quantity, was measured in the emesis bag. There was no contemporaneous chart record of this. The lack of documentation on the fluid balance chart meant that Dr Williams' ongoing internal bleeding and loss of blood went undetected.

As will be further discussed, the experts in epistaxis - the Oral Maxillary-Facial Unit (OMFU) team - should have been called and consulted at this time because of the significant blood loss. However, this did not occur.

The ED Registrar applied co-phenylcaine (local anaesthetic) and phenylephrine 0.5% (vasoconstrictor to decrease blood flow) intranasally. The anterior nose was then packed with tranexamic acid to promote coagulation. This packing occurred at some time before 7.33am. This treatment, in itself was a reasonable approach at that time.

At 7.33am a CT scan was conducted of Dr Williams' head and facial bones. No intracranial haemorrhage was detected, and motion artifact (patient movement during image acquisition) limited the accuracy of the assessment of the complex fracture of the facial bones. It was clearly apparent, however, that blood was still filling Dr Williams' sinuses. This again was a signal for specialist intervention, being a situation likely beyond the usual experience of an ED Registrar for epistaxis. This is because Dr Williams was continuing to bleed despite anterior packing, vasoconstriction and promotion of coagulation.

Upon the evidence in the coronial investigation, Dr Williams' continued bleeding at this time suggested a posterior source. An angiography (radiography of blood and lymph vessels) should have been added as an addendum to the CT scan to determine more accurately which vessels were involved, if any, and an assessment of the best approach to stop the bleeding.

Further, a balloon tamponade (a temporary inflatable device to apply pressure and stop bleeding) should have been applied. This would have involved blocking any posterior bleeding and could well have rectified the issue. If it did not, and the bleeding had then continued, the clinical team should have treated the situation as an emergency, with specialist advice sought. This did not occur.

At 7.45am, external pressure was applied to the anterior of Dr Williams' nose for 50 minutes. Subsequently, his nose was still actively bleeding, but the blood had slowed. His care was then handed over to the morning ED staff.

At 8.32am, Dr Williams was transferred to a high visibility area in the ED, with five-minute observations. He continued to spit up blood.

At 8.56am, his blood pressure had fallen to a dangerously low 76/28 mmHg with a heart rate of 115 bpm. Dr Williams' haemoglobin level had fallen to 129 g/L.

Within four hours of being a patient in ED, Dr Williams entered into haemorrhagic shock as evidenced by the above observations and his clinical course involving continuing blood loss. The treating doctors involved in Dr Williams' care did not appreciate the severity or risks associated with his continuing bleeding. At this point, an investigative plan should have been devised with senior clinicians which may have prevented the death of Dr Williams. However, there was no investigative plan, and senior clinicians were not, by this stage, involved in the decision-making in respect of further urgent treatment.

Dr Williams was not under the care of any particular medical team at that time (none had accepted care) and he was not adequately monitored. Despite already being within the ED, the Medical Emergency Team (MET) was not called to Dr Williams for several hours.

At 9.05am, nursing staff noted that Dr Williams' had ongoing bleeding, a patent airway, spontaneous breathing, pale and cool skin and that he was sweating. Nursing staff also noted that his airway was at risk due to his bleeding and that he was tachycardic (abnormally fast heart rate). Intravenous access was established through two cannulae and blood was transfused (one unit of O negative packed red blood cells) via a fluid warmer. Tranexamic acid was administered intravenously.

Dr Williams' vital signs improved at this time but by 10.10am his blood pressure had again dropped, indicating that the treatment administered was inadequate.

At 10.24am, the OMFU registrar reviewed Dr Williams. Epistaxis was continuing despite anterior nasal packing. A large clot was removed from the oropharynx and nasopharynx (in the pharynx behind the mouth and nose). The OMFU registrar's notes indicated that his gagging had decreased and that they had attempted control of the epistaxis with pressure. They also indicated that after discussion with the OMFU consultant, they were content to admit Dr Williams under the OMFU team.

The assessment by the OMFU team was limited and delayed. There was no plan to manage continued bleeding, and at 11.24am, the epistaxis had recommenced. Dr Williams was

coughing up blood clots and was complaining of blood running down the back of his throat. The ED registrar was made aware of this and discussed it with a member of OMFU. An antibiotic was administered to Dr Williams. At this time, his anterior nasal packs were removed. It was unclear who removed the packs and the rationale for this. It appears that the only appropriate reason for removal of the packs could have been to insert posterior packs. However, this did not occur.

At 11.44am, a MET call was made due to Dr Williams' ongoing bleeding, hypotension, tachycardia and desaturation. His haemoglobin had fallen to a concerning 100 g/L and his blood pressure was dangerously low at 64/42 mmHg.

At 11.52am, concerns were raised by staff about the missing nasal packs. A chest x-ray was performed, with the comment on the x-ray being *"previously lost nasal packs not in chest"*. However, posterior nasal packs were not put in place for over one hour despite the MET presence. A further two units of pack red blood cells (cross-matched) were administered at 12.00pm and 12.25pm respectively.

At 12.17pm, the MET was stood down. This decision was premature as Dr Williams' condition had not stabilised.

At 12.27pm, Dr Williams' condition again started to deteriorate with increased tachycardia, continued hypotension, oxygen desaturation and increased agitation. The MET was called again and OMFU was present. Anterior and posterior nasal packing were performed with "Rapid Rhinos". Dr Williams, who was conscious, said that he thought the blood flow down the back of his throat had ceased. This indicated that the posterior packing had slowed blood loss.

Despite the two MET calls, there was still no definitive plan to determine the cause of the bleeding and arrest the bleeding. By this stage, over four hours had elapsed during which steps should have been taken to properly assess the cause of Dr Williams' continuing blood loss and the potentially life-threatening nature of his condition.

I cannot determine upon interpretation of the records whether a third MET call occurred, possibly at 1.08pm. If it did, there was no change in Dr Williams' management following this call and he remained critically unstable.

MET protocols required that a second MET call within a 24-hour period must trigger immediate contact with an ICU Senior Registrar/Consultant and the home unit (OMFU) consultant for review. This protocol may not have been well-publicised at the time. Although there is an indication on the evidence that a Senior ICU Registrar was advised, it is unclear

when that occurred and the nature of that contact. The OMFU consultant was not contacted, possibly because the treatment focus at that stage was upon fluid replacement rather than surgical intervention.

At about 1.45pm, before his ultimate transfer to the Intensive Care Unit (ICU), Mr Williams passed 2000ml of blood rectally.

At approximately 2.00pm, Dr Williams was transferred to the ICU. He was placed on oxygen via a Hudson mask and subsequently switched to high flow oxygen therapy. Whilst he was initially stable, his condition began to deteriorate.

At 2.25pm the ICU Senior Registrar contacted the ICU consultant to inform him of Dr Williams' instability and requested the presence of the consultant. Fluid resuscitation was commenced at this time. It appears that the gravity of Dr Williams' condition was then appreciated.

At 2.45pm, the ICU consultant arrived and requested activation of the Massive Transfusion Protocol. Management plans for larger venous access and airway management were made.

Dr Williams' blood pressure dropped to 78/50 mmHg, his respiratory rate was 27 breaths per minute and he experienced oxygen desaturation. He was ashen in colour with peripheral circulatory shutdown. He was noted to be more confused, drowsy and complained of blood clots in the oropharynx. Copious amounts of recent blood were detected in his airway, which was suctioned and scooped out manually.

At 2.57pm, Dr Williams lost consciousness and a decision was made for emergency intubation of his airways. His heart rate fell to 45 bpm and his blood pressure was unreadable indicating a loss of cardiac output. CPR was commenced. There was a Code Blue called (patient in cardiac arrest). During resuscitation, the endotracheal tube became dislodged and Mr Williams went into cardiac arrest.

Between 3.13pm and 4.50pm, Dr Williams underwent a prolonged resuscitation of 97 minutes. During resuscitation, at 3.17pm, Dr Williams received blood products that included six units of packed cells, four units of fresh frozen plasma (used to treat excessive bleeding) 10 units of cryoprecipitate (helps control bleeding) and one unit of platelets (helps body to form clots to stop bleeding).

At 3.27pm, during recurrent CPR, Dr Williams' endotracheal tube was found to be dislodged and was replaced.

At 3.54pm a return of spontaneous circulation was achieved and Dr Williams was connected to the ventilator to assist with his breathing.

For the following three days until 27 February 2019, Dr Williams did not show recovery and remained fully sedated, intubated and ventilated in ICU. All sedation ceased on 27 February 2019.

On 28 February 2019 an MRI was performed to assess Dr Williams' degree of brain injury caused by his hypovolaemic (severe loss of blood or body fluid) and hypoxic (reduced supply of oxygen) cardiac arrest requiring a long period of resuscitation. As a result, it was assessed that he was in a critical neurological condition with global hypoxic ischaemic injury from which he could not recover. In consultation with his family members, active treatment was withdrawn and replaced with palliative treatment.

On 1 March 2019 Dr Williams' endotracheal tube was removed and he was commenced on a morphine infusion.

At 1.50am on 2 March 2019 Dr Williams was pronounced deceased.

I am satisfied that he died as a result of his massive brain injury, occurring because of a lack of blood and oxygen in the setting of his prolonged resuscitation.

Comments upon Mr Williams' medical treatment

It will be apparent from the chronology set out above that the severity of his condition should have been recognised and treated effectively at an earlier time and before he had lost a critical quantity of blood. If his bleeding had been controlled, Dr Williams would not have entered into cardiac arrest.

In the investigation, I sought and received an independent opinion from Professor Peter Cameron, experienced specialist in Emergency Medicine and Academic Director of The Alfred Emergency and Trauma Centre.

I also sought an independent opinion from Dr Anthony Bell, coronial medical consultant who was formerly the Director of the Department of Critical Care Medicine. The opinions expressed by Dr Bell in his report regarding treatment deficits closely coincided with those expressed by Professor Cameron in his report.

Additionally, I have received the Tasmanian Health Service *Final RCA Report*, which has also been of considerable assistance in explaining the deficits in treating Dr Williams.

As a result of receiving these expert reports, I make the following comments relating to Dr Williams' medical treatment.

I find that triage at the RHH was adequate, and the initial, routine measures of first aid and assessment were performed appropriately. However, there were ample opportunities subsequently to properly assess and assertively treat Dr Williams' condition. Such opportunities were not taken.

In his report, Professor Cameron set out the essence of the treatment deficits in respect of Dr Williams as follows:

> *"This patient was clearly in hypovolaemic shock after 2 hours in the ED. He had lost a large volume of blood and was continuing to bleed. He was inadequately monitored, had poor vascular access and was poorly resuscitated, given the obvious large volume blood loss, evident from soon after his arrival. Given that he had ongoing bleeding, a plan should have been made to investigate this further and senior clinicians should have been involved in this decision making, when it became apparent that he was not responding to simple measures. This should have been around 9.30am at the latest, four hours after arrival and when he was clearly in haemorrhagic shock."*

The failure to appreciate the severity of Dr Williams' condition should be viewed in light of a number of matters. Firstly, deaths from epistaxis are quite rare, with most occurring as a result of complications from pre-existing conditions. Dr Williams did not have any relevant pre-existing conditions.

Further, bleeding from this type of injury is nearly always controlled by pressure or packing and bleeding requiring emergency surgical or radiological intervention is very rare.

Moreover, Dr Williams' state of intoxication may have been a complicating factor, although he was cooperative with treatment. He also had a history of intravenous drug use and Professor Cameron speculated that there may have been some tendency on the part of doctors to ascribe his sweating and tachycardia to drug withdrawal. Further, his intravenous drug use made vascular access more difficult.

I do not, however, consider that these factors should reasonably have prevented proper assessment of the significance and level of deterioration of his condition and the serious medical issues involved.

A summary of the main inadequacies in treatment, based upon the expert opinion, can be summarised as follows;

(a) No medical team (namely, OMFU) had full responsibility or accountability for Dr Williams' care whilst he was in the ED, as should have occurred in a timely manner;

(b) Consultants in OMFU and/or ICU, with the experience to treat the presenting issue, were not notified of Dr Williams' condition until it was too late;

(c) The relevant consultants were not called immediately after the second (and third) MET calls as should have occurred by protocol;

(d) There was a failure to recognise in ED by 9.30am that Dr Williams had entered into hypovolemic shock and required urgent treatment and resuscitation;

(e) The MET team left Dr Williams without ensuring that his condition had safely stabilised;

(f) No definitive plan was formulated to determine the cause, to manage the continued bleeding and to arrest the bleeding;

(g) There was a lack of awareness by ED staff of the volume of blood loss, partly due to inadequate and incorrect recording on the fluid balance chart;

(h) There were inadequate preparations for a significant bleed by cross-matching blood, appropriate transfusion and available vascular access; and

(i) There was confusion regarding the deliberate removal of the initial anterior nasal packs and failure to perform posterior nasal packing with a balloon tamponade until 12.27pm.

It is highly likely that, had prompt and effective treatment occurred, Dr Williams would have survived. Earlier posterior packing should have occurred. If this was not successful, then angiography should have taken place with further options considered by a skilled consultant – including arterial embolisation, ligation and packing - before the occurrence of critical blood loss.

I cannot, of course, find with absolute certainty that Dr Williams would have survived with competent and timely treatment as there is always the possibility of the condition not being able to be treated despite all reasonable measures. However, in this case, I can find on the balance of probabilities that he should have survived his injuries.

Action taken by the Royal Hobart Hospital subsequent to Dr Williams' death

The RCA panel, in assessing root cause of the failures in respect of Dr Williams, placed emphasis upon particular systemic issues – these included; (a) lack of staff awareness of the need for the relevant consultants to review a patient who has been subject of two MET calls in 24 hours; (b) lack of documentation of output on the fluid balance chart; (c) a lack of clarity regarding the process for triggering the *Trauma Team Response Protocol* for patients already in ED that meet the criteria for trauma call or trauma alert criteria and (d) lack of clarity regarding the process that the majority of patients with isolated facial fractures are admitted under the OMFU team led to no early call and review by a Surgical Registrar and/or an anaesthetist/intensivist.

The RCA panel made five primary recommendations responsive to what it considered to be the primary causes of the failure to treat Dr Williams adequately. These are summarised below;

1. That RHH ED initiate measures to improve fluid balance documentation, including a review of current processes, raising awareness amongst ED nursing staff and auditing fluid balance charts in the ED.

2. That the *RHH Medical Emergency Response (MET and Code Blue) THS Protocol* be revised to give prominence to the instruction for escalation to consultant level of any patient who has triggered two MET calls criteria within 24 hours.

3. That an education package be included in the Tier 2 Mandatory Education, Training and Assessment protocol for all relevant THS South clinical staff to educate them in the escalation process for any patient who triggers two MET call criteria within 24 hours.

4. That actions be taken to raise awareness of clinical staff within ICU and ED regarding the importance and clinical accountability for Bedside Clinical Handover requirements to be performed in accordance with THS protocol; and for documentation requirements to detail a deteriorating patient's management/monitoring, evaluation and follow-up plan in accordance with THS protocols.

5. That there be a review of the *Trauma Team Responses Protocol* to provide clarity regarding when a trauma call can be activated for patients in the ED, particularly relating to a patient whose injury is significant enough to require a blood transfusion within four hours of presentation while in ED.

I acknowledge that the recommendations made by the RCA panel were implemented promptly.

In his report, Professor Cameron commented that the following matters should occur:

1. That RHH review its management protocols for patients suffering facial trauma with ongoing bleeding including:

    (a) Inclusion of senior clinicians where ongoing bleeding is evident;

    (b) Focusing on a definitive plan to investigate further bleeding; and

    (c) Focusing on airway assessment and cardiovascular stability in the initial evaluation of epistaxis, considering airway intervention, fluid resuscitation and emergent otolaryngological consultation (as experts in epistaxis).

2. That RHH reviews its resuscitation protocols for ongoing traumatic haemorrhage.

I do not consider that it is necessary to make recommendations over and above those that have been already implemented by the RHH in accordance with the RCA report. However, it would be appropriate for the RHH, having been provided with Professor Cameron's report, to consider whether reviews of the areas to which he refers are necessary in order to prevent a similar outcome to that occurring in this case.

Criminal proceedings in respect of the assault upon Mr Williams

Mr Beau Wayne Kelly appeared in the Hobart Supreme Court in 2020 and pleaded guilty to one count of manslaughter contrary to Section 159 of the *Criminal Code* 1924. He was sentenced on 9 April 2020 to a term of five years imprisonment with a non-parole period of two and a half years.

In sentencing, His Honour Justice Brett made the following comments:

> *"There was no reason for you to punch this man. You have not asserted that your actions were provoked by him. Even if he did say something to cause offence or anger during the verbal exchange, this could not possibly have justified your actions. The only conclusion that I can reach is that your conduct resulted from drunken bravado. You had been drinking heavily for several hours and must have been extremely intoxicated when you committed the crime.*

> *… I accept that you did not realise how badly you had injured him. I accept also that, even in your drunken state, you did not consciously think about the potential consequences of your actions, before you punched him. However, you deliberately committed an unlawful assault, and that act caused Mr Williams' death. You are, therefore, responsible for that consequence… I suspect that your capacity for such insight was inhibited by the effect of the alcohol you had consumed, but this, of course, will not mitigate your moral culpability for the crime."*

Conclusion

Unfortunately, Dr Williams was the victim of trauma deliberately inflicted by Mr Beau Kelly. This trauma was a substantial cause of Dr Williams' death, as reflected by the criminal proceedings against him for manslaughter. However, it is unlikely that Dr Williams would have died had his bleeding been controlled as it should have been whilst he was a patient at the hospital.

The deficits in his treatment were not attributable to any one individual and there is no suggestion that the treating staff were not attempting to do their best in the circumstances. I acknowledge that the RHH acted promptly upon the recommendations of the *RCA Report* in order to improve its systems and procedures in the relevant areas.

I am grateful to investigating officer, Detective Senior Constable Andrew Peterson for his very thorough investigation.

I convey my sincere condolences to the family and loved ones of Dr Stewart Williams.

Dated: 17 November 2023 at Hobart in the State of Tasmania.

Olivia McTaggart

Coroner

MAGISTRATES COURT TASMANIA

MAGISTRATES COURT *of* TASMANIA

CORONIAL DIVISION

ANNEXURE 'A'

Evidence in the investigation into the death of Stewart Williams

- Police Report of Death for the Coroner;
- Royal Hobart Hospital Death Report to the Coroner;
- Affidavits confirming identity and life extinct;
- Opinion of the forensic pathologist regarding cause of death;
- Toxicology report of Forensic Science Service Australia;
- Tasmanian Health Service and Grosvenor Street Medical Practice records for Dr Williams;
- Statutory declaration of George Williams, son of Dr Williams;
- Statutory declarations of seven witnesses at the venue where the incident occurred;
- Supreme Court of Tasmania Comments on Passing Sentence;
- CCTV footage of the venue and Despard Street where the incident occurred;
- Signed photograph of the CCTV footage;
- Search conducted by Tasmanian Police of 3 Robbins Place;
- Police Interview with Beau Kelly;
- Mobile phone data of Beau Kelly;
- Various court documents including offence report, brief and facts for the prosecutor, prior convictions for Beau Kelly and victim impact statement;
- An affidavit from the ED registered triage nurse;
- Affidavits from two ED registered nurses regarding Dr Williams' treatment in the 'Mountain' (lower acuity) area of the ED;
- Affidavit from the ED Registrar regarding his transfer to 'River' (higher acuity) area of the ED;

- Affidavits from the ED Staff Specialist and two Registrars regarding his care in the 'River' (increased clinical surveillance) area of the ED;
- Affidavits from three ED registered nurses regarding his care in the resuscitation area of the ED;
- Affidavit of the ED registrar involved in the first MET call;
- Affidavit of Resident Medical Officer involved in the first MET call;
- Affidavit of Consultant Oral and Maxillofacial Surgeon;
- Affidavit of Senior Registrar, Intensive Care Medicine;
- Affidavit of the on-call Intensive Care Specialist;
- Affidavit of the on-call consultant to the ICU, Staff Specialist;
- Affidavit of Senior Resident Medical Officer, Anesthetics and ICU;
- Affidavit of Registrar, Emergency Surgical Unit;
- Affidavit of Intensive Care Registrar;
- Affidavit of Clinical Nurse Consultant- trauma;
- Affidavit of Registered Nurse, Department of Critical Care Medicine;
- Tasmanian Health Service Death Review Committee Minutes from 27 May 2019;
- Tasmanian Health Service Root Cause Analysis report and update from the RHH regarding the status of the Root Cause Analysis recommendations and their implementation;
- Letter from Dr Emma Huckerby regarding staffing levels and occupancy from 10.00pm Saturday 23 February 2019 to 10.00pm Monday 25 February 2019;
- Letter from Dr Huckerby regarding the second MET call in relation to Dr Williams;
- Opinion of Peter Cameron, Professor of Emergency Medicine at Monash University;
- Review of Dr Williams' medical care by Dr Anthony Bell, coronial medical consultant; and
- Review of medical records conducted by the Coronial Nurse.