CORONERS ACT, 2003

SOUTH *AUSTRALIA*

FINDING OF INQUEST

An Inquest taken on behalf of our Sovereign Lady the Queen at Adelaide in the State of South Australia, on the 30th and 31st days of March, the 22nd day of June and the 28th day of October 2010, by the Coroner's Court of the said State, constituted of Anthony Ernest Schapel, Deputy State Coroner, into the death of James William Wallace.

The said Court finds that James William Wallace aged 39 years, late of 9 First Avenue, Semaphore Park, South Australia died at the Royal Adelaide Hospital, North Terrace, Adelaide, South Australia on the 23rd day of February 2008 as a result of rhabdomyolysis complicating cocaine toxicity against a background of atherosclerotic coronary artery disease. The said Court finds that the circumstances of his death were as follows:

1. Introduction and reason for Inquest

1.1. James William Wallace, aged 39 years, died at the Royal Adelaide Hospital (RAH) on Saturday 23 February 2008. In an incident that had occurred in the early hours of Thursday morning 21 February 2008, Mr Wallace had suffered a cardiorespiratory arrest as a result of which he had sustained severe brain damage. He was ultimately declared brain dead and his life support was terminated. The incident on the Thursday morning had involved bizarre behaviour on Mr Wallace's part and a number of police officers and paramedics of the South Australian Ambulance Service (SAAS) had attended. I discuss that incident in due course.

1.2. A post mortem examination of Mr Wallace's body that involved a full autopsy was conducted by Professor Roger Byard, a forensic pathologist at Forensic Science South

Australia. In his post-mortem report[1] Professor Byard expresses Mr Wallace's cause of death as rhabdomyolysis complicating cocaine toxicity against a background of atherosclerotic coronary artery disease. In the course of these findings I will discuss the deceased's cause of death in some detail, but for current purposes Professor Byard's opinion as to Mr Wallace's cause of death can be described in lay terms as muscle meltdown (one of the toxic effects of cocaine) contributed to by undiagnosed heart disease that was identified for the first time during the course of the autopsy.

1.3. Mr Wallace's cocaine toxicity had manifested itself in the first instance in disorderly conduct and strange behaviour that presented as both a danger to himself as well as to the public. There was a great deal of evidence as to Mr Wallace's behaviour. This was provided by various eyewitnesses who gave statements to the investigating police. A convenient summary of this evidence is contained within the comprehensive report of the investigating officer, Detective Sergeant Cameron Georg of the Major Crime Investigation Branch[2]. I here set out Detective Sergeant Georg's summary:

> 'At about 12.45 a.m. on Thursday 21 February 2008, James William Wallace (the deceased) left his home address of 9 First Avenue Semaphore Park driving his red and white Ford Bronco FI00 utility. Whilst driving out of his garage, his vehicle collided with the roller door support causing damage to the garage and the side of his vehicle.
>
> He then drove to the home address of his girlfriend in nearby Third Avenue Semaphore Park. There, he drove in an erratic manner, accelerating and then breaking harshly on a number of occasions. He then sped off in the direction of Military Road.
>
> A short time later he was observed on Bower Road Ethelton, driving in a dangerous manner, swerving from one side of the road to the other at a speed of about 80km/h. A short time later, his vehicle collided with a traffic island and road signage on Military Road Semaphore South before running a red light at the intersection of Semaphore Road and Bower Road Semaphore. The deceased then reversed back through the intersection and sped off along Military Road continuing to swerve from one side of the road to the other. He then executed another u-turn on Military Road and drove directly at another motorist coming from the opposite direction, causing the motorist to stop. He then drove up on the footpath before executing another u-turn and driving erratically north along Military Road towards the Police Academy.
>
> A short time later the deceased drove his vehicle through the perimeter fencing of the Fort Largs Police Academy at Taperoo. He then proceeded to drive through the complex, colliding with numerous fixed objects, causing damage to his vehicle, academy buildings and fences.

[1] Exhibit C58
[2] Exhibit C56

> A short time later he crashed his vehicle over an embankment on the foreshore on Lady Gowrie Drive at Largs North. He then got out of his vehicle and stood in the middle of the roadway. He appeared as if in pain and was shouting uncontrollably. He confronted several other motorists in what appears to be an attempt to get into their cars. One of these vehicles may have struck the deceased slightly as the driver attempted to drive off. The deceased fell to the ground and was then assisted by the motorist to the side of the road. The deceased continued to shout and cry out and appeared to be suffering from a psychotic episode. Nearby residents were awoken by the crash and commotion and telephoned for the police and an ambulance.' [3]

I adopt that summary for the purposes of these findings and indicate that in my opinion it is an accurate account of what transpired.

1.4. Naturally Mr Wallace's behaviour attracted police attention. Paramedics from SAAS also attended at the scene at Lady Gowrie Drive. After the arrival of the authorities Mr Wallace continued much in the same vein as before. Together, the police officers and the paramedics attempted to control Mr Wallace with a view to conveying him by ambulance to hospital. To this end paramedics administered Mr Wallace intravenously with 2mg of midazolam, a sedative, while police physically restrained him. Within a very short period of time after the administration of the sedative Mr Wallace experienced a cardiorespiratory arrest. Resuscitative measures were put in place as a result of which circulation was eventually restored. Mr Wallace was at first conveyed to the Queen Elizabeth Hospital (QEH) and from there to the RAH Intensive Care Unit where he would die on 23 February 2008. The cardiorespiratory arrest and the 'downtime' experienced before his circulation could be restored had unfortunately led to a severe hypoxic brain injury which could not be reversed. All this begged the question as to what had caused the cardiorespiratory arrest in the first instance. Hence the autopsy. The cause of Mr Wallace's cardiac arrest was a live issue during the course of this Inquest. Among the issues that required consideration in this regard were the effects of cocaine toxicity upon Mr Wallace's body, the contribution made by his underlying heart disease and whether the administration of the sedative, midazolam, had in any way contributed to the cardiorespiratory arrest.

1.5. Although both the intervention of police and SAAS had involved Mr Wallace's liberty being restrained, police did not at any time purport to exercise their powers of arrest or detention in respect of Mr Wallace. The reason for imposing restraint upon Mr Wallace's liberty was primarily to secure his transport by ambulance to a hospital

[3] Exhibit C56, page 2

where he could be examined and treated for whatever acute illness, be it physical or psychological, that Mr Wallace was apparently experiencing. To the extent that the definition of 'death in custody' as it appears in Section 3 of the Coroners Act 2003 might encompass a death of a person where there is reason to believe that the cause of death arose, or may have arisen, while the person was in defacto custody, as this arguably was, Mr Wallace's death was investigated as a death in custody. A death in custody requires a mandatory Inquest into the death. In any event, it was necessary and desirable that an Inquest be held into the cause and circumstances of Mr Wallace's death, particularly having regard to the fact that one aspect of the cause of Mr Wallace's death, namely the cardiorespiratory arrest, had occurred while Mr Wallace was in the hands of the authorities and while he was being physically restrained. As well, Mr Wallace's father, Mr William Wallace, who in the Inquest represented the late Mr Wallace's family, has expressed some deep concern about the circumstances in which his son came to be restrained by police and SAAS personnel and, in particular, the circumstances of, and contribution to his death made by, the administration of midazolam to his son.

1.6. It was for all of the above reasons that an Inquest was held into this matter.

2. Mr Wallace's antecedents

2.1. Mr Wallace's physical medical history was unremarkable. As already mentioned, there is no evidence that any pre-existing coronary artery disease or other heart disease had been diagnosed in Mr Wallace's lifetime. However, at the post-mortem examination Professor Byard found marked focal coronary artery atherosclerosis with approximately 75% stenosis of the right coronary artery. In addition, there was also fibrosis of the heart muscle in keeping with previous ischaemic damage. The degree of stenosis of the right coronary artery is of some significance when considering the fact that the cardiac arrest occurred in the course of, or following, major exertion on Mr Wallace's part. The fibrosis that was consistent with previous ischaemic damage of the heart also meant that sometime in the past Mr Wallace had experienced a heart attack. This heart attack may have been asymptomatic at the time because there is no evidence that Mr Wallace had been diagnosed with any heart disease, be it acute or chronic.

2.2. Mr Wallace was said to be a regular cannabis user. Evidence located by police at his home following his death as well as in his car supported the existence of such a habit. He was also known to be a user of cocaine. Ms Sally Hammond had been Mr Wallace's partner since approximately December 2006, although she had known him for many years prior to that. Ms Hammond gave a statement to police[4] in which she asserts that Mr Wallace was a user of both cannabis and cocaine. He ingested cocaine by way of nasal inhalation through a straw. She describes the effect that cocaine ingestion had on his behaviour. Ms Hammond relates a recent incident in which Mr Wallace had exhibited some apparently paranoid behaviour involving a belief that his house was being invaded and she suggested that it was known that Mr Wallace had experienced similar episodes in the past while under the influence of cocaine. Mr Wallace's former partner Nicola Wallace confirmed Mr Wallace's cannabis and cocaine usage and his suffering paranoid delusions about intruders while under the influence of cocaine[5]. In fact, records from the QEH reveal that in 2000 Mr Wallace had been detained under the Mental Health Act 1993 following a psychotic episode at his home address where he was said to have been armed with a firearm and was guarding his house against non-existent intruders. The Acute Crisis Intervention Service (ACIS) assessment at the time involved a diagnosis of several weeks of increased paranoid ideation in the context of heavy cocaine use and concluded that he was prone to violent outbursts whilst taking cocaine and would be hyper vigilant and paranoid. The principal diagnosis included cocaine induced psychosis that was supported by a urine drug screen which gave a positive result for the presence of cocaine.

3. The toxicology results and their interpretation

3.1. I received in evidence the report of Amanda Thompson who is a forensic scientist and toxicologist employed by FSSA[6]. It states that a blood sample taken from Mr Wallace on his admission to hospital following the incident in question was found to contain a small concentration of methylamphetamine, approximately 0.04mg of cocaine per litre, 4.2mg of benzoylecgonine per litre and a sub-therapeutic concentration of midazolam.

[4] Exhibit C15a
[5] Statement of Nicola Wallace - Exhibits C34 and C34a
[6] Exhibits C5 and C5a

3.2. As to the interpretation of these levels and their possible effect on Mr Wallace I received evidence from Professor Jason White. Two reports of Professor White were tendered in evidence[7]. Professor White also gave oral evidence in the inquest. Professor White is the Professor of Addiction Studies and Head of the Discipline of Pharmacology at the University of Adelaide. As well, he is the Director of the Pharmacotherapy Research Unit for the Drug and Alcohol Services of South Australia. Professor White has an Honours Bachelor of Science Degree in Psychology from the University of Adelaide as well as a PhD in Psychology from that same University. He received post-doctoral training in pharmacology in the United States. He has given expert evidence not only in this Court but in other Courts in South Australia on many occasions.

3.3. Before discussing Professor White's evidence as to the potential effects of cocaine ingestion, there is one matter regarding the toxicological analysis that requires discussion. It is true that the blood sample that was to be analysed by and reported on by Ms Thompson, in terms of its potential evidentiary value, was not maintained in ideal conditions prior to its analysis. In his first report and also in his evidence Professor White discusses this issue and its possible effect upon the reliability of the toxicological results. I do not need to go into the details of the sub-optimal maintenance of the sample prior to its analysis because Professor White, in the event, was of the very firm opinion that it essentially made little difference to the way in which the toxicological results were to be interpreted. The sample revealed both the presence of cocaine and a substance known as benzoylecgonine which is a substance that cocaine breaks down into in the bloodstream. That latter substance can only emanate from cocaine. The presence of benzoylecgonine in Mr Wallace's blood sample is only attributable to the breakdown of the cocaine that Mr Wallace had ingested. Whilst the less than ideal circumstances in which the sample had been maintained may have had some deleterious consequences as far as accurate analysis of the amount of cocaine is concerned, Professor White told me in evidence that the same issue does not apply to the presence and level of benzoylecgonine that was detected within the sample. Professor White's evidence is that the concentration of benzoylecgonine in Mr Wallace's blood sample was an extremely high one and indicated that Mr Wallace originally had a very high cocaine concentration in his blood in the period in which he was observed to drive and act in the manner

[7] Exhibits C57 and C56b

described. Such a level of cocaine could account for that behaviour. I am satisfied that the toxicological result is accurate insofar as the level of benzoylecgonine indicates that Mr Wallace had ingested a significant quantity of cocaine that was greater than what might be considered to be a usual so-called recreational quantity.

3.4. In the opinion of Professor White, Mr Wallace's behaviour when driving his vehicle and during the following period can be explained by his consumption of cocaine with the small quantity of methylamphetamine. The methylamphetamine may have added slightly to the effects of the cocaine but the major effect on Mr Wallace's behaviour was likely to have emanated from the cocaine. Both drugs have the capacity to produce a psychotic reaction that is characterised by hallucinations, delusions and paranoia. A person affected is likely also to appear very agitated with rambling, incoherent speech and aggression. Individuals differ in their susceptibility to this type of reaction and their behaviour is influenced also by the amount of drug consumed. Professor White noted Mr Wallace's previous alleged experience with cocaine and a level of paranoid behaviour attributed thereto. In Professor White's view this indicated that Mr Wallace had previously consumed sufficient doses of the drug to induce psychotic symptoms and it could mean that Mr Wallace was particularly susceptible to the psychotic effects of the drug. In my view Mr Wallace's behaviour during the morning in question is explicable on the basis of cocaine consumption and the mind altering and behavioural effects of that substance. I so find.

4. The Lady Gowrie Drive incident

4.1. It is necessary to say something more of Mr Wallace's behaviour at the Lady Gowrie Drive scene in order to place the actions of police and paramedics into their proper context. As Mr Wallace's vehicle passed the junction of Kybunga Terrace, it struck the raised median strip in the middle of the road and travelled along the median strip for a distance of about 20 metres before returning to the southbound carriage of Lady Gowrie Drive. It then continued on for a further 60 metres before crossing over the centre line. It then mounted the western footpath and collided with the foreshore fencing. The vehicle came to rest on the downward incline of the embankment that abutted the beach. Mr Wallace was then seen to attempt to reverse the vehicle from its resting position but the rear drive wheels had little or no traction on the embankment. During this time the vehicle continued to rev loudly with its rear wheels spinning but with the vehicle itself not moving. Mr Wallace then alighted

from the vehicle and ran diagonally north across Lady Gowrie Drive to about the middle of the road where he was heard to be yelling out for help. A number of nearby residents made observations of what was taking place and it is pertinent to observe that a number of telephone calls were made by the public to police regarding Mr Wallace's behaviour. Although some of Mr Wallace's behaviour could be viewed as consistent with his having sustained an injury in the accident, much of it objectively must also have been highly consistent with the appearance of psychosis. He was screaming and yelling and calling out for help and his behaviour in endeavouring to get into a passing motorist's car was outwardly bizarre. He was yelling odd things to passing motorists such as 'the ghosts are coming, someone's trying to hit me'[8]. At one point Mr Wallace leaned over a vehicle and started punching the windscreen. As the vehicle drove off Mr Wallace continued to punch the rear passenger side of the vehicle. He was also heard at other points to be saying 'put a bullet in my head'[9]. This same utterance was to be heard by police when they attended the scene.

4.2. The first officers to arrive on the scene were Constable Tanya Leonard and Probationary Constable Andrew Murdock. Following the incident, Constables Leonard and Murdock both gave detailed statements about the incident to senior police officers[10]. As well, their debriefing process consisted of separate interrogations by Detective Sergeant Georg on 28 February 2008[11]. In addition, both officers provided further written statements in October 2008[12]. All of those statements were tendered to the Inquest.

4.3. Constables Leonard and Murdock arrived at the scene at approximately 1:20am. Both officers observed Mr Wallace's vehicle which was positioned precariously on the embankment situated between the western side of the carriageway and the beach. The position of this vehicle of itself must have outwardly suggested that its driver was subject to an impairment of one form or another. A number of bystanders were at the scene and it appeared from their demeanour that they were keen on police intervention in the light of what had taken place, and was still taking place. Mr Wallace was lying on the southbound carriageway of Lady Gowrie Drive. He was rocking side to side and kicking his legs around, keeping his arms over his head as

[8] Exhibit C21a, Statement of Rebecca Debroughe
[9] Exhibit C25a and C25b, Statements of Brian Neve
[10] Exhibits C38a and C39a
[11] Exhibits C38b and C39b
[12] Exhibits C38c and C39c

though protecting his head. Police were concerned that Mr Wallace might be struck by traffic travelling along the road and so the scene was illuminated with police vehicle headlights and the flashing red and blue lights. Constable Murdock went to Mr Wallace and noticed immediately that he was very agitated and it was then that Mr Wallace asked Constable Murdock to shoot him. Attempts by Constable Murdock to determine whether or not Mr Wallace was injured were met with resistance during which Mr Wallace was constantly verbalising in an incomprehensible way for the most part, but which could be understood to include more requests for police to shoot him. Constable Murdock also had concerns about the possibility that Mr Wallace had suffered a physical injury and was further concerned that his behaviour of thrashing about might make that injury worse, as well as putting Constable Murdock's own personal safety at risk. He was also concerned to prevent Mr Wallace from injuring any other person in close proximity. Constable Murdock states in his final statement[13] that it was going through his mind during the ensuing incident that police could not just simply leave Mr Wallace there without some kind of intervention. Constable Leonard describes Mr Wallace's behaviour as unable to be controlled. His body was thrashing around violently on the ground and he was pulling his arms to his chest as if he was either in pain or was attempting to prevent police from trying to control his arms. To Constable Leonard it appeared that Mr Wallace was 'suffering some type of mental breakdown'[14]. Constable Leonard points out in her final statement[15] that police had some information that Mr Wallace had been involved in an impact with another vehicle and so what was unknown to police was the extent of any possible injuries that he may have suffered. She makes it plain that she also thought at the time that Mr Wallace may have been suffering from a mental impairment as evidenced by his behaviour which involved yelling and screaming.

4.4. Constables Leonard and Murdock were joined at the scene by Senior Constable Sean McMahon and Probationary Constable Rachel Poolman. It appears that a SAAS crew comprising Intensive Care Paramedic Stella Sharman and Paramedic Nicole Powell arrived at the scene around this time. Officers McMahon[16] and Poolman[17] were both subjected to the same debriefing process as the other officers. McMahon and Poolman confirm in their various statements that Mr Wallace was extremely agitated

[13] Exhibit C38c
[14] Exhibit C39a, page 3
[15] Exhibit C39c
[16] Exhibits C40a and C40b
[17] Exhibits C41a and C41b

and was shouting out. He was heard to say words to the effect 'I know you all want to kill me'[18]. The two male officers, Constables Murdock and Senior Constable McMahon, endeavoured to verbally reassure Mr Wallace but Mr Wallace continued to yell at them. He thrashed about violently on the ground and Senior Constable McMahon became concerned about his own safety as Mr Wallace was reaching out at him. Constable Murdock held Mr Wallace's legs while Senior Constable McMahon attempted to hold him on the ground by placing his left hand on his right shoulder and his right hand on his right arm. Senior Constable McMahon describes Mr Wallace as 'completely irrational'.

4.5. At about this point the two paramedics intervened. A pillow was placed under Mr Wallace's head in order to protect his head and face from the bitumen surface of the road. Mr Wallace resisted efforts at that stage to place handcuffs on his wrists but this was eventually able to be achieved. A flexicuff was also applied to both ankles.

5. Midazolam is administered

5.1. Having regard to Mr Wallace's unusual presentation, the two paramedics decided to convey him to hospital. In her first witness statement Ms Sharman[19] states that her initial assessment of Mr Wallace was that he was very delusional and paranoid[20]. This impression was based on her own observations as well as on information about his behaviour that was supplied by police. Ms Sharman observed that Mr Wallace's speech was erratic and that he was apparently speaking to his father who was not there. Attempts by Ms Sharman to examine Mr Wallace physically for any injuries was rendered difficult by his uncooperative and aggressive nature and the need for him to be restrained. Nevertheless, Ms Sharman observed no obvious sign of injury and Mr Wallace appeared to have good strength and movement in his limbs. Ms Sharman states that whenever the officers tried to roll him over, Mr Wallace would 'thrash about'[21] and her concern was that someone was going to get hurt. That included the possibility in her mind that Mr Wallace himself might break a limb or damage his head on the road surface. Ms Sharman, being the senior of the two paramedics, decided that Mr Wallace needed to be sedated in order to calm him down for the purposes of conveying him to hospital. Ms Powell, the other paramedic, drew

[18] Exhibit C40a, page 3
[19] Exhibit C9a
[20] Exhibit C9a, page 3
[21] Exhibit C9a, page 4

up the midazolam and was then instructed to examine Mr Wallace's vehicle to see if there were any substances there to explain Mr Wallace's behaviour. In the event, some cannabis and a cannabis pipe were located. There was no suggestion that this in itself would have contraindicated the administration of the sedative. None of the officers, or the paramedics, was to know that Mr Wallace's behaviour was explained by cocaine psychosis and even if they had known this, it would not necessarily have precluded the administration of midazolam.

5.2. Ms Sharman administered 2mg of midazolam via an intravenous cannula that she was able to insert into Mr Wallace's left lower arm. Her statement reveals that when she did this she instructed the police officer who was holding the right side of Mr Wallace not to apply too much pressure to that side of the patient's chest. Mr Wallace continued to struggle while the cannulation was taking place. According to Ms Sharman he was also continuously talking and yelling throughout the cannulation. He was not pausing and appeared not to have any difficulty with his breathing at that time. The police officers confirm that Mr Wallace continued to struggle during the cannulation process.

5.3. Ms Sharman then administered the 2mg midazolam through the cannula. The manner in which the midazolam was administered has been the subject of critical comment that I shall deal with presently. In her first statement Ms Sharman describes the process as follows:

> 'I administered 2mgs of Midazolam and I directed the SAPOL officer who was holding the left arm to decrease that grip she had on the bicep area of left arm. About 20 seconds later the patient went quiet.' [22]

One available interpretation of that description is that Ms Sharman may have administered the midazolam in one uninterrupted and rapid administration over a very short period of time, sometimes referred to as bolus administration. However, as was pointed out to me during the course of the evidence by Professor Byard, if Ms Sharman had intended to administer the midazolam by way of a rapid bolus, it is surprising that it would have been administered through a cannula and not merely intramuscularly with an ordinary needle. Be that as it may, a supplementary affidavit was taken from Ms Sharman[23] in order to clarify the manner in which she had

[22] Exhibit C9a, page 6
[23] Exhibit C61

administered the midazolam. In that affidavit she describes the administration as follows:

> 'I injected the Midazolam as a "slow push" in accordance with how I had been taught and what I understood to be standard procedure. By "slow push" I mean that I slowly depressed the plunger with my thumb until 2mg of the fluid had been dispensed and then withdrew the syringe. I am not able to say precisely how long the administration of the 2mg took. I have read portions of the transcript and do not believe it took as long as 1 minute but it was not given as a "bolus" in the manner in which this word is used in general medical language.' [24]

5.4. I raise the manner in which the midazolam was administered because Professor White passed comment about the need to avoid administering a sedative drug in that quantity rapidly, which might then involve respiratory complications.

5.5. At the time with which this Inquest is concerned there were certain guidelines promulgated within SAAS concerning the use of midazolam in the context of acute behavioural emergencies. The guideline[25] as it then existed is attached to the statement of Dr Hugh Grantham who is the Medical Director of SAAS. Dr Grantham explains in his statement that in contexts such as these, SAAS has had considerable experience in using midazolam in prescribed doses without adverse effects. Intensive Care Paramedics were permitted to administer midazolam without any prior medical consultation with a doctor. The then guideline described the required administration process as follows:

> 'In adults, consider administration of midazolam in 1mg **increments** IV to a maximum total dose of 3mg.' (emphasis added)

Professor White's second report[26] in effect amounts to a critique of Ms Sharman's method of administration as described in her supplementary affidavit. Professor White[27] explains that the dose of 2mg is the recommended range for the purposes of sedation and that the effects of the drug are observed within 2 minutes after commencing administration. He suggests that the rate of administration of a dose of 2mg is based on a low infusion rate. One recommended rate of infusion is 30 seconds per 1mg. Anther more conservative recommendation suggests that the infusion rate should allow for at least 2 minutes for a 2.5mg dose to be given to a healthy adult. In any event, the total dose administered should be adjusted to take account of the

[24] Exhibit C61, pages 1-2
[25] Exhibit C36b
[26] Exhibit C56b
[27] Exhibit C57, page 3

ongoing response of the patient. It should also have regard to the required 'end-point', namely the safe sedation of the patient, which should be readily observable to the clinician. If the desired end-point is reached, the infusion is ceased irrespective of whether the whole of the planned dose has been administered. I took all this to mean that the clinician determines the dose by reference to the observed effects of the drug as it is gradually administered. Professor White suggests that monitoring for adverse effects should be ongoing, particularly in relation to its effect on the heart and respiration. There is also a need to bear in mind that the effects of the drug may continue to increase for 2 minutes or more after an infusion has ceased. For that reason monitoring of the patient's response should continue for at least that period of time before any supplementary dosage is given. It seems to me that all of this is encompassed in the word 'increments' as utilised within the above SAAS guideline. Professor White regarded the manner of infusion as described by Ms Sharman in her supplementary affidavit as not conforming to the usual recommendations regarding the use of the drug for sedation and could not therefore be considered appropriate. He acknowledged that the 2mg dose was consistent with usual recommendations, but suggests that the rate of infusion was greater than is recommended insofar as it left inadequate time to observe the effects of the drug on the patient, and therefore to adjust the dose according to these effects. I infer from Ms Sharman's description that although the 2mg of the drug was administered not in bolus form but by way of a slow push, it was administered continuously without any interruption to check for Mr Wallace's ongoing response to the drug. I return to the question as to whether or not the administration of the midazolam contributed to Mr Wallace's cardiac arrest.

6. Events following the administration of midazolam

6.1. It appears that the agitated behaviour of Mr Wallace ceased quite rapidly following the administration of the midazolam. In his first statement Constable Murdock suggested that Mr Wallace became completely motionless within 5 seconds of the administration of the sedative[28]. Mr Wallace was still on the ground at that stage and was still handcuffed. According to Murdock, Mr Wallace was then placed on the ambulance gurney and it was at that time that Murdock and Constable Poolman removed the handcuffs. Mr Wallace was then wheeled into the ambulance and at that

[28] Exhibit C38a, page 4

point an ambulance officer said 'I think he has gone into cardiac arrest'[29]. Both ambulance officers then went into the ambulance and commenced resuscitative efforts.

6.2. Constable Leonard, in her original statement[30], states that she watched while the ambulance officer inserted the needle into Mr Wallace's left arm 'at which time he seemed to immediately calm down'[31], although he continued to mumble incomprehensibly. The ambulance officer then injected the sedative through the needle and within 10 seconds Mr Wallace calmed down completely and stopped struggling. Constable Leonard says that she found this sudden reaction to be concerning and so she squeezed Mr Wallace's left earlobe in order to get a response. There was no response. According to Constable Leonard, Mr Wallace was then lifted onto the gurney and was wheeled into the ambulance. Constable Leonard then went to check Mr Wallace's vehicle and was thus momentarily distracted from the events at the ambulance. However, she then heard an ambulance officer state 'he's arrested', meaning of course that he had gone into cardiac arrest. According to Constable Leonard's account is consistent with Mr Wallace having already been placed in the ambulance at the time his cardiac arrest was detected.

6.3. Senior Constable McMahon states that after the sedative was administered Mr Wallace immediately calmed, such that they could release him and roll him on his side. Mr Wallace was then placed on the gurney and wheeled to the rear of the ambulance, throughout which he remained calm. According to Senior Constable McMahon, at that point Constable Leonard asked the ambulance officer whether Mr Wallace was alright and Ms Sharman checked him and replied that he was breathing. According to Senior Constable McMahon, Mr Wallace's handcuffs were removed and Mr Wallace was then moved into the ambulance whereupon one of the paramedics yelled out 'he's arrested … I don't have a pulse'.

6.4. Probationary Constable Poolman, in her original statement[32] suggests that it was about 10 seconds after the drug was administered that Mr Wallace stopped struggling. He was then lifted onto the gurney. Constable Poolman describes Constable Leonard pinching his earlobe with a lack of response. Mr Wallace was pushed to the rear of

[29] Exhibit C38a, page 5
[30] Exhibit C39a
[31] Exhibit C39a, page 4
[32] Exhibit C41a

the ambulance where the handcuffs were removed and he was then placed into the rear of the ambulance. One of the ambulance officers then announced that he had gone into cardiac arrest.

6.5. On an analysis of the evidence of the four officers there appeared to be a rapid response by Mr Wallace to the administration of the sedative.

6.6. Ms Sharman's description of these events was that Mr Wallace went quiet about 20 seconds after she administered the midazolam. Ms Sharman suggests that at that point one of the female police officers asked her whether the sedative was supposed to work that quickly, at which stage Ms Sharman checked Mr Wallace for a pulse. To Sharman it appeared that there was a very faint pulse but she noted that he was by then not breathing, that he had instantly stopped talking and that he was no longer responsive. Ms Sharman believed that she detected a carotid pulse and then directed the officers to move Mr Wallace onto the gurney. The gurney was then moved to the rear of the ambulance and Ms Sharman applied a bag and mask and commenced ventilation whilst the officers removed the handcuffs. CPR was then commenced.

6.7. According to the paramedic Ms Powell[33], at the time of the administration of the sedative she was in the vicinity of Mr Wallace's vehicle checking to see if there was any substances in the vehicle. She then returned to the position of the ambulance. By then Mr Wallace had been placed on a gurney which was at the rear of the ambulance. From Mr Wallace's appearance Ms Powell states that she could tell that he was in cardiac arrest[34]. By then Ms Sharman had begun preparations to resuscitate the patient. She had obtained a bag and mask for that purpose. They decided it was best to load him into the ambulance where CPR was commenced.

6.8. An analysis of the evidence of Sharman and Powell, Mr Wallace was in difficulty in terms of his respiration, circulation or both at a time before he was placed on the gurney.

6.9. It is not surprising that in a dramatic incident such as this, involving as it did the rapid collapse of a patient, that there would be differences in the perceptions of various participants in the incident. The officers seemed to hold the belief that the cardiorespiratory arrest not was detected until after Mr Wallace had been placed on

[33] Exhibit C10a
[34] Exhibit C10a, page 4

the gurney and then into the ambulance. On the other hand Ms Sharman, who was the person who actually detected the cardiorespiratory arrest, suggests that the arrest occurred prior to the placement of Mr Wallace onto the gurney. When all things are considered, it is objectively more likely that the person who actually detected the cardiorespiratory arrest, namely the qualified Intensive Care Paramedic, Ms Sharman, would be accurate in this regard.

7. Professor Byard's evidence as to the cause of Mr Wallace's death

7.1. I have already alluded to the fact that Mr Wallace's autopsy was conducted by Professor Byard, a forensic pathologist. In his evidence Professor Byard explained that one might well regard Mr Wallace's hypoxic brain damage, which had resulted from Mr Wallace's cardiac arrest, as the cause of death. However, in Professor Byard's view to say that this was the cause of Mr Wallace's death would be an oversimplification and would ignore that fact that there were available explanations for the cardiac arrest. Thus, Mr Wallace's cause of death was more appropriately to be considered in terms of what had caused the cardiac arrest. In Professor Byard's opinion, the cardiac arrest is explained by rhabdomyolysis complicating cocaine toxicity, together with Mr Wallace's pre-existing but undiagnosed ischaemic heart disease. Therefore, the cause of death is more correctly rhabdomyolysis complicating cocaine toxicity against a background of atherosclerotic coronary artery disease. It will be noted in that recitation of Professor Byard's cause of death that the possible effects of, and contribution made by, the midazolam administration is not mentioned.

7.2. I turn to the question of whether Professor Byard's stated cause of death is one that I should adopt and find to be the case. I direct myself that the standard of proof in this regard is the balance of probabilities.

7.3. The principal anatomical finding at autopsy was the rhabdomyolysis. Rhabdomyolysis is typically known as muscle meltdown. Professor Byard explained that when muscles become overheated they start to breakdown with the result that the muscle cells release their contents, following which the kidneys become significantly impaired. In addition, high levels of potassium that might be generated could stimulate arrhythmias in the heart. In Mr Wallace's case Professor Byard identified muscle meltdown, particularly on the left side of his neck where the muscle could be seen by the naked eye to be swollen and full of fluid. On microscopic examination it

could be seen that the muscle was actually breaking down. There were also casts in the kidney tubules and these casts consisted of myoglobin which is the protein inside muscle cells[35]. As to the possible causes of rhabdomyolysis, Professor Byard explained that it can be brought on by overheating such as that experienced by athletes exercising in very high temperatures. It also occurs in states of excited delirium in which people experience high bodily temperatures. On admission to hospital Mr Wallace recorded a temperature of 40.5°C which is high. In Professor Byard's opinion, in Mr Wallace's case the rhabdomyolysis was the product of his ingestion of cocaine and amphetamines. Professor Byard explained that quite apart from the psychiatric manifestations of cocaine toxicity such as agitation, paranoia and aggression, cocaine toxicity and the resulting excited delirium can manifest itself in sweating, high temperatures and then rhabdomyolysis. The excited delirium is a behavioural manifestation or psychiatric state brought about by the cocaine toxicity[36]. Mr Wallace's behaviour just prior to his collapse was in keeping with excited delirium[37]. Professor Byard was of the opinion that in Mr Wallace's case his rhabdomyolysis was quite profound and indeed was to an extent that Professor Byard had not previously seen described. He had not seen described in any of the literature muscle necrosis which had involved a whole muscle having necrosed[38]. In Professor Byard's opinion the degree of rhabdomyolysis was in and of itself potentially fatal and that his death could have been caused by that alone regardless of any pre-existing heart condition[39].

7.4. I add at this point that a suggestion that was allegedly or apparently made by one of the clinicians in the hospital to which Mr Wallace was taken to the effect that it appeared that Mr Wallace may have been strangled, was explained in Professor Byard's opinion by the existence of the rhabdomyolysis in the neck area of the deceased. There was no other evidence of any kind of strangulation and it is of significance that rhabdomyolysis was only diagnosed at autopsy and not clinically within the hospital.

[35] Transcript, page 114
[36] Transcript, pages 115-116
[37] Transcript, page 116
[38] Transcript, page 121
[39] Transcript, page 122

7.5. Professor Byard explained that cocaine and methylamphetamine are classically the two drugs that are associated with hyperthermia and rhabdomyolysis and this is naturally in keeping with the toxicology analysis that I have already referred to.

7.6. In Professor Byard's opinion the approximate 75% stenosis of Mr Wallace's right coronary artery was also an important finding as was the fibrosis that was consistent with previous ischaemic heart damage. Although there was no evidence of an acute heart attack, Mr Wallace was at high risk of a cardiac arrest irrespective of the circumstances. Firstly, there was the significantly narrowed right coronary artery and secondly, there was the area of scarring. There were a number of factors that would, in the circumstances, have increased Mr Wallace's heart rate at around the time of his collapse, such as his agitation and movement. Added to that was the high level of cocaine and amphetamine, a combination that stimulates adrenaline such that there would be an increase in heart rate and blood pressure adding further stress to the heart.

7.7. It was for those reasons that Professor Byard recited Mr Wallace's cause of death as rhabdomyolysis complicating cocaine toxicity against a background of atherosclerotic coronary artery disease. Professor Byard rejected the notion that Mr Wallace's physical restraint had made any contribution to the mechanism of his cardiac arrest insofar as it may have compromised his ability to breathe. Professor Byard did not believe there to have been in Mr Wallace's case any evidence of what is termed 'positional asphyxia' or 'restraint asphyxia' which is a condition that at times has occurred in situations involving physical restraint where the restraint might compromise a person's ability to breathe. In Mr Wallace's case Professor Byard highlighted the fact that, to the point of Mr Wallace's collapse, he was continuously verbalising in an agitated way, even during the cannulation process, and did not appear to have any difficulty with his breathing. According to the Intensive Care Paramedic, Ms Sharman, Mr Wallace was continuously talking and yelling throughout the cannulation; he did so without pause and he appeared not to be having any difficulty with his breathing[40]. Moreover, Ms Sharman specifically directed the officer who was holding Mr Wallace's upper body in the shoulder area not to apply

[40] Exhibit C9a, page 5

undue pressure to the right side of his chest and the officer appeared to acknowledge that. In short, Professor Byard said:

> 'I can't see any evidence in the history or in my autopsy findings to implicate restraint asphyxia.' [41]

7.8. There was no evidence of significant trauma to Mr Wallace. There were multifocal superficial abrasions and bruises, but there is no suggestion that they are anything other than in keeping with them having resulted from Mr Wallace's own behaviour and his resistance to restraint. A suggestion that there had been a possible clinical diagnosis of a split liver or other traumatic liver damage revealed on a CT scan prior to Mr Wallace's death proved to be unfounded at autopsy. There was no liver injury either external or internal identified by Professor Byard.

8. The possible contribution of midazolam to Mr Wallace's cause of death

8.1. Professor White and Professor Byard had slightly differing views about the possible contribution of the midazolam administration. Professor White left open the possibility that it played a role. Professor Byard did not believe it had played any role.

8.2. Professor White did not seek to bring into question the post-mortem finding that cocaine consumption and the resulting rhabdomyolysis was the most significant factor in Mr Wallace's death. However, Professor White did express certain views about the possible contribution of the sedative midazolam. In his first report[42] Professor White suggests that the effect of midazolam is to decrease the agitation and psychotic symptoms produced by cocaine and that it has been recommended and used for this purpose. Therefore, there is nothing to suggest that the use of midazolam in itself, and even if the SAAS Intensive Care Paramedic had been made aware that Mr Wallace's symptoms were being caused by cocaine ingestion, was contraindicated. However, in someone who was also at risk of cardiac arrest because of the effects of cocaine, and particularly if the risk was high as a result of atherosclerotic coronary artery disease as well, Professor White suggested that the effect of midazolam on respiration could theoretically increase the risk of cardiac arrest due to the decreased oxygen supply from depressed respiration. In his evidence Professor White suggested that with appropriate administration, the risk of cardiac arrest would not be regarded

[41] Transcript, page 127
[42] Exhibit C57

as very significant, even with a cocaine toxicity and overdose, but the risk is increased where there is coronary artery disease already in existence. While Professor White agreed that it was possible that the midazolam did not play a significant role in Mr Wallace's cardiac arrest and death, the observation that Mr Wallace's condition worsened immediately after administration of midazolam could not be wholly ignored. In this regard Professor White suggested that there may be some significance in the fact that Mr Wallace's condition was noticed to worsen following midazolam administration but suggested that because the matter was outside his specific field of expertise, how much of that worsening contributed to his eventual death he could not say. He said:

> 'The fact that that occurred very rapidly suggests some contributing role for midazolam but as I indicated I wouldn't like to pass judgment on the relative importance.' [43]

8.3. In the event, in his evidence Professor White expressed the view that the relative contribution of each factor, including midazolam administration, could not be stated with certainty. He did say that he would defer to the opinion of a forensic pathologist as to cause of death and would not venture an alternative hypothesis to Professor Byard's conclusion that rhabdomyolysis was the principal cause of death.

8.4. For his part, Professor Byard suggested that midazolam, on a statistical basis at least, carried a very small risk in terms of respiratory depression. He suggested that midazolam was not a cardiac toxic drug in itself. However, Professor Byard acknowledged that the temporal association of Mr Wallace collapsing soon after the injection naturally made one wonder whether midazolam may have been involved after all. Nevertheless, when the entire circumstances were properly considered, Professor Byard expressed the view that the sedative played no role[44]. Firstly, even though the temporal association was worrying, he did not think it proved any association with his death in fact. This was because Mr Wallace had experienced both a muscle meltdown and also had coronary artery disease and any of those two things at any time could have caused his cardiac arrest. The extent of muscle meltdown, it will be remembered, was in Professor Byard's opinion possibly fatal in and of itself. So was the pre-existing heart disease. Those two factors in combination, together with Mr Wallace's exertion and agitation, very much suggests that Mr Wallace's sudden collapse is to be explained on the basis of those factors alone.

---

[43] Transcript, page 92
[44] Transcript, page 132

8.5. The other matter that Professor Byard thought was significant was that very soon after Mr Wallace's collapse he was administered with oxygen. The resuscitative efforts had taken place rapidly. Professor Byard suggested that the oxygen therapy ought to have ameliorated the adverse effects of respiratory depression caused by the midazolam. Professor Byard suggested that if respiratory depression had been caused by the midazolam, it ought to have preceded any cardiac arrest that may have resulted from the respiratory depression. Such respiratory depression in his view probably would have been noticed prior to any cardiac arrest. The fact that the cardiac arrest was detected as one of the first manifestations of Mr Wallace's collapse was more in keeping with the effects of rhabdomyolysis and the effects of cocaine than the effects of any sedative or respiratory depressive drug[45].

9. Conclusion regarding the cause of Mr Wallace's death

9.1. In my opinion the cause of Mr Wallace's death was as described by Professor Byard, namely rhabdomyolysis complicating cocaine toxicity against a background of atherosclerotic coronary artery disease. I accept the evidence of Professor Byard on that account. Professor White's evidence did not differ in that regard. Professor White's evidence left open a possible contribution by way of the administration of midazolam whereas Professor Byard discounted the possibility. The evidence in my view is overwhelming that the cocaine ingestion and consequent rhabdomyolysis was the principal factor in the causation of Mr Wallace's death. That in and of itself could have killed him, quite apart from the pre-existing ischaemic heart disease which I find also rendered him susceptible to the possibility of a cardiorespiratory arrest.

9.2. In my view the possible contribution of midazolam and its extent if any in considering Mr Wallace's cause of death is quite unclear. Whereas the temporal connection between the administration of midazolam and Mr Wallace's collapse is a stand-out feature of the circumstances of his collapse, it is simply impossible to say that on a balance of probabilities it had made any contribution at all. It remains a theoretical possibility, but one which cannot be elevated to a degree of probability.

9.3. Accordingly, I do not propose to record in Mr Wallace's cause of death any reference to the possible effects of midazolam.

[45] Transcript, page 135

10. General conclusions

10.1. During the course of the Inquest, Mr William Wallace, the late James Wallace's father, drew my attention to a number of inconsistencies and discrepancies between the statements of the various participants in this incident. True it is that there are some differences between the accounts of the police officers as between themselves and between those accounts and the accounts of the two ambulance officers. The major difference is that already described, namely the timing and position of Mr Wallace at the time his cardiac arrest was detected. To my mind that difference does not require any resolution having regard to its lack of materiality. As to any other discrepancies that might exist, I do not need to cite them and I need say no more about the issue except that they are the kinds of differences that one would expect when several witnesses have been asked independently to describe the same event, especially an event with such dramatic and unfortunate consequences. They are not the kind of discrepancy that would necessarily cause one to doubt the veracity of the evidence of an individual witness.

10.2. Mr Wallace urged me to find that the actions of the police in restraining Mr Wallace were inappropriate, as were the actions of the ambulance officers in administering a sedative drug. Mr Wallace went so far as to suggest that once the police had successfully handcuffed his son, the matter regarding his son's behaviour could have been satisfactorily resolved by simply arresting him and taking him into custody. The suggestion is that if that course had been adopted, his son might still be alive today. I have very carefully taken all of Mr Wallace's submissions into account and have asked myself on many occasions, both during and since the Inquest, whether there were indeed alternative measures open to police and SAAS, measures that might have altered the outcome.

10.3. In considering Mr Wallace's submissions it has to be kept in mind that even if an alternative course of action was open to police and the SAAS paramedics, it does not follow that the actions actually taken by them were therefore unreasonable. Granted the police could have arrested Mr Wallace for disorderly behaviour, but the statements and records of interview of the four police officers make it very plain that the last thing on their mind was arrest, even having regard to the fact that his behaviour was erratic, unlawful and such as to possibly place the public at risk and that for those reasons his arrest could have been justified. The statements taken from

all police officers make it clear that their motive in restraining Mr Wallace was purely and simply to better effect his transportation to hospital for his own welfare. I have no reason whatsoever to doubt that was the case. It was not simply a case of police encountering a man whose behaviour was disorderly. His behaviour was very much suggestive of the fact that Mr Wallace was experiencing an adverse medical episode, be it from the ingestion of drugs or by way of some naturally occurring mental illness. The officers perceived a necessity to get Mr Wallace to a hospital and in my opinion it cannot be said that this was an unreasonable view of the matter. On the contrary, some might say it was the only view of the matter. And their concern was proven to be well placed when it is remembered that the fact of the matter was that Mr Wallace was profoundly physically ill from the effects of cocaine. One only has to contemplate what the outcome and the sequelae may have been if police had arrested Mr Wallace and he had then died in the cells. The officers concerned would have exposed themselves to criticism for an egregious failure to obtain medical assistance when it was obvious that that was what the prisoner required.

10.4. It seems to me also that having regard to Mr Wallace's behaviour, the officers would have been lawfully justified in exercising their powers under Section 23(1) of the Mental Health Act 1993 (MHA) as it then was. The exercise of these powers did not in any way depend upon whether or not police had detected or suspected the commission of any offence. The powers under the MHA as they existed at the time were designed purely to enable police to apprehend a person whom they had reasonable cause to believe had a mental illness and that the conduct of that person was, or had recently been, such as to cause danger to himself or to others. In my view it would have been open to any one of the four officers to form such a conclusion and then to have apprehended Mr Wallace, using only such force as was reasonably necessary for the purpose, in order to take him to a medical practitioner for examination. In practice that of course would have meant a hospital. The provisions of Section 23 of the MHA were such that a member of the police force and an ambulance officer might assist each other in the exercise of the powers under that section[46]. The actions of both the police and of SAAS personnel in restraining Mr Wallace's liberty would have been lawfully justified on that basis alone.

[46] Section 23(8) of the Mental Health Act 1993

10.5. In short, in my opinion it cannot be said that the police officers either acted unlawfully or inappropriately. I am perfectly convinced that their motivation was at all times benign and one in which they had uppermost in their minds the welfare of Mr Wallace, his protection and that of the general public.

10.6. As far as the actions of the SAAS paramedics are concerned, the more recent statement of Dr Hugh Grantham, Medical Director of SAAS[47], suggests that the paramedics were entitled to rely on the common law doctrine of necessity to justify the administration of a sedative drug. If any analogy can be drawn, it might be said that the administration of a sedative drug is akin to the physical force that might be required to place a badly injured, but perhaps unwilling individual, into an ambulance before being taken to hospital. It has not been necessary for me to pass any comment about the application of the doctrine of necessity as a defence to what might otherwise be an assault insofar as it might be said to justify the administration of a drug. I say so for a number of reasons. Firstly, I am of the firm view that the two paramedics in this instance acted utterly in good faith and in what they believed to be the best interests of Mr Wallace. The administration of a sedative was genuinely thought to have been necessary in order to secure his conveyance to hospital. It is difficult to see how it can be said that such a conclusion was unreasonable. Moreover, none of the circumstances as they existed indicated that its administration was clinically contraindicated. Secondly, and in any event, Section 23 of the MHA as it then existed empowered an ambulance officer when summonsed by a police officer, to convey a person to, say, a hospital and to 'use such force as is reasonably necessary for the purpose'[48]. Arguably, such force could have included the administration of a sedative drug where such was reasonably necessary for the purpose of conveying a person who had been apprehended by the police under Section 23 of the MHA to a place where medical treatment could be effected. Again, I do not need to decide this having regard to the fact that the new Mental Health Act 2009, which came into effect on 1 July 2010, specifically empowers an authorised officer, which includes an ambulance officer, to restrain a person by means of the administration of a drug when it is reasonably required in the circumstances[49].

10.7. As I have indicated above, in any event the evidence that midazolam played a role in Mr Wallace's cardiac arrest is unclear. It may not have played any role at all. It

[47] Exhibit C62
[48] Section 23(6)(b) of the Mental Health Act 1993
[49] Section 56(3)(d) of the Mental Health Act 2009

certainly cannot be said that it has been demonstrated on a balance of probabilities that midazolam contributed to Mr Wallace's death.

10.8. When considering the appropriateness or otherwise of the actions of police and the SAAS personnel in this case, it is worthwhile observing that no bystander who witnessed the incident or part of the incident makes any adverse comment about the behaviour of police or paramedics. The common theme among the witness statements of bystanders is that Mr Wallace's behaviour required professional intervention[50]. The objective viewpoints of bystanders are exemplified as follows. The statement of Mr Brian Neve suggests that Mr Wallace's behaviour was uncontrolled and irrational and that the police had to use much of their strength to hold him down as Mr Wallace was using a great deal of force to try and push them away. He states:

> 'I did not see police use excessive force but they had to try hard to hold him down for the ambulance officers to treat him.' [51]

In a second statement[52] Mr Neve indicates that he believed the officer who was mainly responsible for Mr Wallace's restraint knew what he was doing and did not appear to be doing anything inappropriate and was not using excessive force. A Mr Ray Evans, suggests in his statement that from what he could tell the police:

> '…were doing a good job and trying to keep the male on the ground and holding him still' [53]

The witness Barbra Anne Murphy[54] suggests in her statement that her impression was that both the police and ambulance personnel were endeavouring to 'help' Mr Wallace.

10.9. There can be no suggestion that once Mr Wallace's cardiorespiratory arrest was detected that the resuscitative measures taken by SAAS personnel were inadequate. Unfortunately they were unsuccessful and Mr Wallace suffered a severe hypoxic brain injury from which he eventually succumbed. There is also no certainty that the lethal effects of the rhabdomyolysis could have been reversed[55].

---

[50] Statements of Julie Anne Dean, Exhibit C19a, Rebecca Debroughe, Exhibit C21a, Aaron Tulic, Exhibit C22a, Walter John Rantanen, Exhibit C27a, Therese Rantanen, Exhibit C28a, Naomi Jan Fewings, Exhibit C29a, Pat Alys Brealey, Exhibit C30a, Errol Dean Ford, Exhibit C32a and Ines Patrizia Raimondo, Exhibit C33a,
[51] Exhibit C25a
[52] Exhibit C25b
[53] Exhibit C26a
[54] Exhibit C31a
[55] Transcript, pages 87 and 122

10.10 As to the suggestion that Mr Wallace's behaviour may have been connected with a drug deal that had gone awry or that there had been some kind of sinister component to his condition that might have involved the unlawful activity of another individual, such was beyond the scope of this inquiry. However, there is nothing in the evidence to suggest that this was anything other than a case of a man who had voluntarily ingested an overdose of an illicit substance in respect of which he had an existing habit and died as a result.

11. Recommendations

11.1. Pursuant to section 25(2) of the Coroner's Act 2003 I am empowered to make recommendations that in the opinion of the Court might prevent, or reduce the likelihood of, a recurrence of an event similar to the event that was the subject of the Inquest.

11.2. Attached to the statement of Dr Hugh Grantham is a revised guideline concerning the administration of midazolam as a sedative. The principal difference from the guideline that was in existence at the time with which this Inquest is concerned, is that the revised guideline[56] contains the following:

> 'Do not administer midazolam to patients who are prone, rear handcuffed, under restraint using the physical weight of another agency member across the torso to control them or who are not yet safely apprehended to allow clinical examination. Only one drug should be selected and administered.'

As with the pre-existing guideline, the revised guideline does not say anything about the rate of administration. I addition, I notice that the revised guideline drops the reference to the need to administer it incrementally. However, other documentation that was apparently promulgated within SAAS in the aftermath of this incident does make reference to this subject[57]. This appears to be a reflection of the kinds of issues identified by Professor White, namely that midazolam should be administered in such a manner so as to enable the person administering it to gauge in a gradual way the reaction of the patient. I would **recommend** that the Medical Director of the SAAS consider issuing a guideline that describes a method of administration in accordance with the evidence given by Professor Jason White, in particular that set out in paragraph 5.5 herein.

[56] Exhibit C36d
[57] Exhibits C36e and C36f

11.3. I also had the benefit of receiving into evidence the affidavit of Madeline Elizabeth Glynn[58] who is an Assistant Commissioner with SAPOL. In that document Assistant Commissioner Glynn describes a number of measures and initiatives that are designed to enhance training with respect to, and to minimise risk involved in, restraint by police officers. The document includes reference to positional asphyxia and excited delirium and describes the training directed to officers in respect of identification of the same and preventative measures that might be taken to ensure the safety of an arrested person who is handcuffed, and if the person is in a state of drug induced excited delirium. The measures and initiatives are to be commended.

11.4. In all of the circumstances I do not see any necessity to make any other recommendation in respect of this matter other than to suggest that these findings be distributed to all police officers so that they may be better informed about excited delirium and the possible effects of substances such as cocaine on a person's behaviour.

[58] Exhibit C55

Key Words: Police Involvement; Death in Custody; Drug Overdose

In witness whereof the said Coroner has hereunto set and subscribed his hand and Seal the 28th day of October, 2010.

Deputy State Coroner

Inquest Number 10/2010 (0240/2008)