I, Olivia McTaggart, Coroner, having investigated the death of Tyler John Broomhall

Find, pursuant to Section 28(1) of the _Coroners Act 1995_, that:

(a) The identity of the deceased is Tyler John Broomhall;

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

(c) Mr Broomhall died as a result of intra-cerebral haemorrhage following methylenediazepam use;

(d) Mr Broomhall died on 20 April 2013 at the Royal Hobart Hospital, Hobart in Tasmania; and

(e) Mr Broomhall was born in Devonport, Tasmania on 15 February 1990; he was aged 23 years and was employed as a factory hand at the date of his death.

In making the above findings I have had regard to the evidence gained in the comprehensive investigation into Mr Broomhall’s death. The evidence comprises an opinion of the forensic pathologist who conducted the autopsy, relevant police and witness affidavits, medical records and reports, and forensic evidence.

Mr Broomhall was a fit, young man who played Australian Rules football with the Wilmot Football Club. He worked as a casual factory hand at Harvest Moon, a vegetable processing farm at Forth. He was a habitual cannabis user. It is unclear on the evidence as to the extent that he used other illicit drugs.

Mr Broomhall received a minor head injury at work about 4 to 6 weeks before his death, which he did not report. In the weeks leading up to his hospitalisation and death it would appear that this injury caused Mr Broomhall to become forgetful, emotional, and lethargic and to lose motivation. He was described as “not being himself.” He did not seek medical attention for these symptoms. It would appear that
his use of cannabis, and possibly methylamphetamine, exacerbated the effects of the head injury, giving him severe headaches.

On 13 April 2013, Mr Broomhall played football and received a heavy knock during the first half of the game. Post mortem examination indicated that this caused a rib fracture.

Mr Broomhall's mother and girlfriend stated in their affidavits for the investigation that they were told by one of Mr Broomhall’s friends that Mr Broomhall had used the drug “ice” (being methylamphetamine) at half time to assist with keeping him on the ground. There were no eye witnesses to Mr Broomhall using this substance. However, from the result of blood tests taken on his presenting to hospital, I am able to find that he did use the substance. I am unable to say if this use was a one-off event or if he had been taking the drug for any extended period before the football match.

At 5.05pm the same day, being 13 April 2013, Mr Broomhall was transported by ambulance to the emergency department of the North West Regional Hospital (NWRH). His chest trauma suffered in the football game was noted by medical staff. His history of vomiting and coughing up blood-streaked sputum was also documented. It was recorded that he had consumed methylamphetamine and that he was confused and agitated.

Serial electrocardiograms (ECG) were conducted upon Mr Broomhall. The doctors were of the opinion that the results showed ECG changes consistent with acute myocardial injury (heart muscle injury). Mr Broomhall was therefore treated with aspirin, being a standard treatment for a myocardial infarction. Over a period of two hours he returned to his normal self and insisted on discharge.

At 10.30pm Mr Broomhall was admitted again, on this occasion to the Mersey Community Hospital, (MCH), upon request of medical staff at the NWRH. Due to concerns about myocardial injury he was treated enoxaparin (an anti-coagulant). The admission was for monitoring and management of amphetamine induced myocardial injury.

At 3.00am on 14 April 2013 Mr Broomhall was noted to be hyper-vigilant, slightly agitated and to have dilated pupils.

At 5.00am Mr Broomhall was noted to be confused and disorientated, with his condition deteriorating. At 6.15am he was reviewed by Dr David Selvanayagam. Intracranial pathology was suspected and a CT scan of the brain was therefore requested.

Later that morning, a CT scan was conducted which showed a large frontal lobe haemorrhage. He was therefore transported to the Royal Hobart Hospital at 3.15pm that afternoon. At the Royal Hobart Hospital Mr Broomhall underwent further CT
scans and was subsequently taken to surgery. A left frontal craniotomy and evacuation of the hematoma was performed. Over the following days his condition did not improve, and he remained in hospital.

On 20 April 2013 Mr Broomhall suffered further intracranial hypertension despite full medical management. Therefore a left sided decompressive hemicraniectomy was performed. However, his condition deteriorated and, unfortunately, he passed away.

On 22 April 2013 Dr Christopher Lawrence performed an autopsy upon Mr Broomhall at the Royal Hobart Hospital.

In his report Dr Lawrence stated:

“Autopsy reveals an intracranial hemorrhage in the frontal area which appears to be a primary intracranial hemorrhage rather than extension of a cerebral contusion. The heart shows mild enlargement and contraction band necrosis consistent with methylamphetamine cardiac damage.

Possible explanations for the cerebral hemorrhage include:

1. An earlier injury complicated by hypertension due to methylamphetamine. The neurosurgeon Mr Jens Peter Wilke thought that there was some older hemorrhage present at the craniotomy on 15 April 2013. However, no haemosiderin was detected in the specimen sent to Anatomical Pathology to confirm this observation.
2. Combined effects of an injury sustained during the football game and the effects of methylamphetamine. While there was a left rib fracture there was no evidence of head trauma.
3. Hypertensive hemorrhage due to the effects of methylamphetamine.

His presentation at NW Regional Hospital and later at the Mersey include cardiac features as well as neurological features which I think fit best for cardiac features of methylamphetamine toxicity. Methylamphetamine was identified on drug screen and on toxicology analysis of ante-mortem blood. I think that methylamphetamine is the major factor in the intracranial hemorrhage.

This case highlights the danger of recreational use of methylamphetamine in sport.”

Dr Lawrence cited the cause of death as intra-cerebral hemorrhage following methylamphetamine use. I accept his opinion.

It was during the course of the coronial investigation that it was discovered that Mr Broomhall had sustained the head injury, referred to earlier, while at work 4 to 6 weeks prior to his hospitalisation on 13 April 2013. This involved him hitting his head on a piece of machinery while cleaning underneath it. He did not report this as a work injury despite being urged to by a workmate who witnessed the incident. Although he had a visible lump on his head as a result of the knock, it did not break
the skin and he considered it too minor to report. The circumstances of the injury were investigated by Workplace Standards. As a result of the evidence in the investigation I am satisfied that the injury was minor and the piece of machinery concerned did not present any particular risk to an employee operating the machinery. I note that the employer fully cooperated in the investigation and has now taken steps to cover the sharp edge in question.

A drug squad investigation has not been able to determine the source of the methylamphetamine used by Mr Broomhall.

**Comments and Recommendations**

**Late diagnosis of head injury**

I note that investigations initially led to Mr Broomhall being treated for a cardiac problem caused or exacerbated by drug use. There was a 6 hour delay in him being correctly diagnosed with an intracranial haemorrhage.

Dr Anthony Bell, coronial medical consultant, has reviewed Mr Broomhall’s medical care and provided an opinion at my request. In his report, Dr Bell notes that Mr Broomhall’s ECG changes had not progressed as would be expected with myocardial injury. Thus the diagnosis should have been reviewed before the administration of the anti-coagulant medication. The non-specific ECG changes without progression actually led to treatment of an acute myocardial infarction which was not present. Dr Bell stated that the treatment was with anti-coagulation and aspirin both of which are contra-indicated in cerebral haemorrhage due to increased risk of bleeding. Thus, the poor interpretation of the ECGs led to inappropriate treatment. However, he further notes that the diagnosis of a cerebral haemorrhage was not possible by clinical examination and there was no specific reason to perform a CT scan of Mr Broomhall's brain at initial presentation.

The diagnosis would have been especially difficult as Mr Broomhall appeared to have minimal signs, if any, of cerebral haemorrhage and gave no history of his earlier head injury. Therefore, given the difficulty of the diagnosis of intracranial pathology, I am not able to say that the standard of care was inadequate.

Director of emergency at North West Regional Hospital, Dr Marielle Ruigrok, has provided a helpful report for the coronial investigation regarding the hospital’s review of the delay in diagnosis. She noted that, upon analysis, an emergency physician rather than an anaesthetist, should have been on-call, which, as a result of a review, is something that now occurs. Dr Ruigrok also noted that locum doctors on duty were unfamiliar with the MCH systems and equipment. If it has not already done so, the MCH might consider reviewing whether the orientation system in place for locums is sufficiently robust.

I am not able to find that Mr Broomhall’s outcome would have been different had he been correctly treated at an earlier time.
Dangers of methylamphetamine use

This finding is published together with my finding in respect of the death of another male who also died after consuming methylamphetamine whilst playing football. It is therefore appropriate to make the following comments regarding the dangers of the consumption of methylamphetamine generally and, more particularly, whilst playing sport or after exertion. I hope that publication of these findings will emphasise the tragic consequences and significant harm that results from its use.

Methylamphetamine (also known as methamphetamine) belongs to the stimulant class of drugs, which also includes amphetamine, ecstasy, and cocaine. These drugs stimulate the brain and central nervous system, resulting in increased alertness and physical activity.

There are three main forms of methylamphetamine: powder (speed), base, and crystal. The research shows that of particular concern is the crystalline form of methylamphetamine, known as “ice”. This is the substance likely consumed by Mr Broomhall before his death.

In 2013, 7% of Australians reported that they had used methylamphetamine in their lifetime, and 2% reported using in the past 12 months. Whilst the same proportion of Australians use methylamphetamine now as in 2007, the frequency, form and method of administration has changed, resulting in greater harms.1

The availability of crystal methylamphetamine has increased, with more users reporting using crystal methylamphetamine rather than lower purity powder methylamphetamine and more regular use. Amphetamine related helpline calls, drug treatment, arrests and hospital admissions per amphetamine disorders and psychosis have all increased steeply since 2010. The increased availability and use of crystal methylamphetamine have been associated with increased regular use and harms.2

It is estimated that in 2013-2014 there were 268,000 regular methylamphetamine users aged 15 to 54 years in Australia. This equates to population rates of 2.09% as regular users and 1.24% for dependent use. This study also noted that the rate of dependent use had increased since 2009–2010, most markedly among young adults aged 15 to 34 years.3

The National Coronial Information System records that from the period 1 January 2011 until 31 December 2015 there were 1193 deaths reported to a coroner in Australia where consumption of methylamphetamine was determined to be the primary cause of death. Males comprised the majority of these deaths. The National

1 Roche et al; Methamphetamine Use in Australia; National Centre for Education and Training on Addiction, Flinders University.
2 Degenhardt et al; Crystalline methamphetamine use and methamphetamine-related harms in Australia; Drug Alcohol Rev 2016 Jun 11;
3 Degenhardt et al; Estimating the number of regular and dependent methamphetamine users in Australia, 2002 – 2014; MJA 204 (4) 7 March 2016
Coronial Information System report notes that the actual number of deaths is likely to be greater due to the number of cases still open.

Dr Bell has provided me with a summary of the effects of the use of methylamphetamine, derived from the medical literature. I have, below, extracted the main points from his summary.

Methylamphetamine stimulates the central nervous system, producing behavioural and physiological effects. It has a high potential for abuse and addiction. It may result in fatality in some individuals when used alone on a single occasion.

Use is associated with serious cardiovascular events, including sudden death in patients with pre-existing structural cardiac abnormalities or other serious heart problems. At higher doses there may be dramatic increases in heart rate, blood pressure, respiration and temperature. Hypertensive crisis, hyperthermia, and refractory arrhythmias are associated with severe intoxication.

Methylamphetamine use can cause or exacerbate focal neurologic deficits (impairments of brain, spinal cord and nerve functions) such as central nervous system ischemia, infarction, or haemorrhage. Use may lead to new onset or breakthrough seizure activity. Headaches, nausea and insomnia are also commonly associated with use.

Both acute and chronic methylamphetamine use is strongly associated with a variety of psychiatric symptoms, including anxiety, paranoia and psychosis, delusions, homicidal and suicidal ideation, aggressive and hostile behaviour. Choreiform movement disorders (rapid, jerky involuntary movements) are a relatively common finding in acute methylamphetamine intoxication. Hypervigilance and akathisia (a need to move constantly) may be present in mildly intoxicated persons, while severe intoxication may cause abrupt changes in behaviour, becoming extraordinarily violent. Psychiatric symptoms are often the chief complaint of patients presenting to the emergency or acute care setting.

The main psychological effects of methylamphetamine include wakefulness, alertness, a decreased sense of fatigue, mood elevation, increased self-confidence, and a decreased appetite. Ingestion prevents the safe performing of tasks which require mental alertness. Methylamphetamine use does not create extra physical and mental energy but merely affects the perception of fatigue and pain. Therefore, a person engaged in athletic activity may feel as though they are moving faster and getting stronger when this is not the case. Moreover, methylamphetamine is notable for distorting the user's perception of reality and impairing judgement, which may cause an athlete to participate while ill or injured, possibly leading to exacerbation of pre-existing injuries or illness. Methylamphetamine also predisposes the user to excessive exertion which can lead to heatstroke and rhabdomyolysis (muscle breakdown).

In Mr Broomhall’s case, I am not able to determine the extent of his past use of methylamphetamine or the dose ingested during the football match. However, the
evidence at autopsy shows that his heart displayed methylamphetamine-related
damage, and also that the pre-existing brain injury suffered at work was significantly
worsened by his ingestion of methylamphetamine during a period of physical
exertion. The worsening of his condition, being increased haemorrhage, led to his
tragic death.

I extend my appreciation to investigating officer, Senior Sergeant Darren Pendlebury,
for his thorough investigation and report.

I convey my sincere condolences to the family and loved ones of Mr Broomhall.

Dated: 12 September 2016 at Hobart in the State of Tasmania.

Olivia McTaggart
Coroner