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Emily Burston
Senior Solicitor, Solicitor assisting the enquiry
Special Commission of Inquiry into LGBTIQ hate crimes

Dear Ms Burston,

Re: Special Commission of Inquiry into LGBTIQ hate crimes: expert report in relation to the death of Richard Slater

Thank you for your letter of the 21st of December 2022 asking me to provide an expert witness report regarding the above case. I have read the expert witness code of conduct and agree to be bound by it. I am a practicing cardiologist in the metropolitan Sydney area and am currently employed as the head of the Department of Cardiology at the Royal Prince Alfred Hospital; my qualifications are outlined in the attached copy of my curriculum vitae (attachment 1). Also attached is a letter of instructions on which this report is based (attachment 2).

I have reviewed the material supplied including the running sheet summary of events leading to the death of Richard Slater, the report of death to the Coroner, autopsy reports and statements of Drs Bennett, Newton and Bookallil. My report is based on these resources. Below I have addressed the questions outlined in the letter of instruction.

1. **Whether it is possible for cardiac issues to be precipitated or exacerbated by injuries obtained in the course of an assault, with particular reference to the medical history of and injuries suffered by Mr Slater.**

   It is most likely that Mr Slater's cardiac events of 20 and 22 December 1980 were precipitated by the assault he suffered and the injuries he sustained at that time.

   In 1980 the understanding of the pathophysiology of myocardial infarction, heart failure and other events was poorly understood, similarly there was no effective treatment available to deal with these clinical events. For example, in 2023 Mr Slater would have undergone urgent revascularization on 20 December 1980 and been supported through this and likely would have survived, this was not the case in 1980, where treatment consisted of digoxin and frusemide for the subsequent heart failure. Treatments for myocardial infarction only advanced once it was realized that the pathophysiology involved thrombosis within the coronary artery on top of a coronary plaque and that dissolution of this clot with thrombolytic therapy or directly with

percutaneous coronary intervention (PCI) was urgently required to improve outcomes (Van de Werf, The history of coronary reperfusion, European Heart Journal 2014, attachment 3). Multiple studies since the 1980s have found that the natural history of coronary artery disease is that plaques develop in the coronary arteries over decades often starting in early life. These plaques then become unstable leading to thrombosis following events such as plaque rupture, the cause of the plaques becoming unstable is not well understood but it certainly appears that many people live for a long period of time with coronary artery disease before it clinically manifests (Libby and Theroux, Pathophysiology of coronary artery disease, Circulation 2005, attachment 4). Whilst in some cases the transformation from stable coronary artery disease to an unstable picture with myocardial infarction there are no obvious causes it is also clear that a number of internal and external factors often play a role in the development of the acute events.

A number of studies have demonstrated the connection between external factors and development of an acute coronary syndrome such as myocardial infarction. For example, during the 2006 FIFA world cup in Germany there were large spikes in the incidence of acute myocardial infarction within that country every time that Germany played (Wilbert-Lampen et al, Cardiovascular events during World Cup soccer, NEJM 2008, attachment 5). It has been recognized that several studies that both physical and psychological stress can lead to the development of acute coronary syndromes due to a number of factors including activation of the sympathetic nervous system and adrenergic stimulation caused by stress, in addition things such as physical trauma and bleeding can lead to dehydration and activation of clotting factors making transition to an acute coronary syndrome much more likely (Muller et al, Mechanisms precipitating acute cardiac events, Circulation 1997, attachment 6).

A connection has also been observed between the brain injuries and acute cardiac events, either due to stroke or traumatic brain injury, particularly in patients that have underlying cardiac issues, although it can also occur in patients without pre-existing cardiac disease (Chen et al, Brain-Heart interaction, Circulation Research 2017, attachment 7). In patients that have suffered severe head trauma, over half have been observed to develop cardiac complications, and this is associated with a significant increase in mortality rates. Even in 2016 these mortality rates were over one third of patients in this group (Hasanin et al, Incidence and outcome of cardiac injury in patients with severe head trauma, Scandinavian journal of trauma and resuscitation 2016, attachment 8).

In December 1980 Mr Slater was a 69-year-old man with a history of a myocardial infarction 12 years before and a more recent history of stable angina, he had also suffered a cardiac arrest under anesthesia 5 year prior to 1980, although the exact nature of this is unclear. Despite this it appears that he was well functioning and able to mobilize well without obvious distress, for example in the period before the events of December 1980 his grandson mentions that he would walk across the park to visit the store selling lottery tickets. This would suggest that at the time of the assault Mr Slater had advanced coronary artery disease, but this was stable with no signs of impending acute coronary syndrome or myocardial infarction.

Following Mr Slater's assault and admission to hospital with significant head and facial injuries, lacerated spleen, internal retroperitoneal bleeding, and numerous superficial bleeding he suffered a myocardial infarction. According to postmortem findings he had suffered a very large myocardial infarction involving most of the heart. At postmortem it was found that he had severe coronary artery disease with

only a pinpoint opening, presumably this refers to the orifice of the left main coronary artery as this would also fit for the distribution of the myocardial infarction. This type of lesion is unlikely to be survivable due to the damage to almost the whole heart, however given that Mr Slater had stable symptoms prior to this it is most likely his problem was that the plaque in his left main coronary artery became unstable around 20 December 1980. This temporal relationship supports the role of his assault in precipitating this and there are clear mechanisms as to why this might have occurred including the extensive bleeding activating his clotting system, the events causing physical stress and emotional stress leading to sympathetic activation as well as the observed role that significant head trauma can have on cardiac risk, as explained above.

2. **With regard to your conclusion as to issue 1 above, the extent to which Mr Slater's death is attributable to a myocardial infarction and/or the injuries occasioned by the assault on 19 December 1980, and/or any other cause.**

   I think that Mr Slater's mode of death was most likely the extensive myocardial infarction he suffered between 20 and 22 December 1980 as this led to cardiogenic shock that was irreversible, however as explained above I think that this event was precipitated by the assault and extensive injuries he sustained from the assault. I base this largely on the clinical course and the postmortem findings of extensive infarction involving almost the whole left ventricle.

3. **Any recommendations for further investigations with respect to determining the manner and cause of Mr Slater's death; and**

   I wonder if it might be worth having an expert opinion from a neurosurgeon regarding his intra-cranial postmortem findings. To my untrained eye the findings on examination of the intracranial cavity appear more serious than I would have expected from a clinical perspective, in particular the finding of extensive cerebral oedema with cerebellar coning. These are usually not good prognostic features of head injury, and I wonder if this might have carried a worse prognosis that thought by Dr Bookallil (based on clinical findings) and that death might have occurred anyway had not a myocardial infarction occurred.

4. **Any other matters you wish to raise within your expertise that may be of assistance to the inquiry.**

   Nil.

If I can provide any further information, please do not hesitate to contact me.

Thank you.
Yours sincerely,

Mark Adams