



MAGISTRATES COURT of TASMANIA

CORONIAL DIVISION



Record of Investigation into Death (Without Inquest)

*Coroners Act 1995
Coroners Rules 2006
Rule 11*

I, Rod Chandler, Coroner, having investigated the death of Roderick David Charles Ham

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

- a) The identity of the deceased is Roderick David Charles Ham;
- b) Mr Ham was born on 30 December 1956 and was aged 59 years;
- c) Mr Ham died at the Royal Hobart Hospital (RHH) in Hobart on 1 October 2016; and
- d) The cause of Mr Ham's death was pulmonary thromboemboli due to deep vein thrombosis following right hip replacement.

Background

Mr Ham was the long-term partner of Ms Jenni Strang. His relevant medical history included hypertension, gout, chronic headache, osteoarthritis of the right hip and polycythaemia (elevated haemoglobin level).

On 15 August 2016 Mr Ham attended orthopaedic surgeon, Mr Michael Pritchard, for a pre-operative assessment. An x-ray of his pelvis had confirmed degenerative disease in the right hip joint. Mr Pritchard recommended a right total hip replacement utilising a direct anterior approach. A pre-operative workup confirmed mild polycythaemia, normal blood coagulation, mildly impaired renal function, slightly elevated liver function results, a normal electrocardiogram and weight of 95kg (BMI 33.26). A review by anaesthetist, Dr Yarrow followed. Mr Ham's polycythaemia was noted. He was assessed to be a low risk for deep vein thrombosis (DVT).

On 7 September 2016 Mr Ham was admitted to Calvary Health Care's hospital at Lenah Valley (Calvary). His hip surgery was straightforward and uneventful. In recovery he experienced low oxygen saturation along with agitation. He was treated by Dr Yarrow and his condition settled. Anti-embolic AV boots were employed and his aspirin, previously taken for his chronic headache, was re-commenced. Post-operatively his management was routine and he was discharged on 10 September.

Ms Strang reports that Mr Ham was '*up and walking*' by the time he was discharged. When he left hospital he was provided with an information sheet setting out exercises which he was advised to do daily. He was also told to avoid sitting for too long.

Circumstances Surrounding the Death

Ms Strang reports that on Thursday 22 September Mr Ham experienced a sharp pain in his back while they were out walking. She says: '*He thought he was going to faint. He went all hot, sweaty and clammy*'. The following morning he was '*really short of breath*' so Ms Strang took him to Calvary's Emergency Department where he was examined by Dr Clive Stack. An ECG showed sinus tachycardia with right heart strain. He was treated with enoxaparin (an anticoagulant or blood thinner) by injection and intravenous fluid was administered. A CT scan of the pulmonary arteries showed bilateral near occlusive pulmonary emboli involving the main pulmonary artery and the lobar and segmental branches. An ultrasound showed an occlusive thrombus within the right femoral vein extending over a length of about 20cm from above the knee crease to the profunda junction. Input was sought from a cardiologist, cardiothoracic surgeon and an intensivist and the decision was then taken to transfer Mr Ham to the RHH.

Mr Ham arrived at the RHH at 5.18pm and was later admitted to a ward under the care of respiratory physician, Dr David Stock. Cardiologist, Dr Phillip Roberts-Thomson, was consulted and he together with Dr Stock agreed that thrombolysis (a therapy utilising medications designed to break up blood clots) was not indicated. Enoxaparin was ordered with a dose of 1 mg/kg twice daily by subcutaneous injection.

In the late evening of 23 September Mr Ham had a fainting episode when attending the toilet. He was treated by the Medical Emergency Team and given alteplase, a thrombolytic drug. He was then transferred to the Intensive Care Unit. In the morning of 24 September a right thigh swelling was noted and a later orthopaedic review reported no evidence of compartment syndrome. Mr Ham's condition was stable the following day but he remained unwell. There was evidence suggesting renal failure and an unfractionated heparin infusion was commenced. Later a renal ultrasound showed normal renal structures. A CT scan of the thigh showed a haematoma but an ultrasound indicated no residual clot.

Mr Ham's condition remained stable during 26 September although a transthoracic echocardiogram showed significant right heart dysfunction. That afternoon he was transferred to the respiratory ward. At 4.30pm there was a spike in his temperature with associated tachycardia. He complained of sweats and rigors. An examination of Mr Ham's respiratory system found bibasal coarse crepitations. Enoxaparin was continued but at a dose of 70mg twice daily. Mr Ham remained febrile on 27 September with a spiking temperature. The following day he reported feeling well. His vital signs were stable. His platelet count was 49 n/l. Physiotherapy was commenced. Over the next two days it was considered that Mr Ham's condition was slowly improving.

At 7.30am on 1 October clinical observations were recorded and did not cause concern. However, there was later a sudden deterioration in Mr Ham's condition with his blood pressure and oxygen saturation levels falling markedly. He became unconscious. Resuscitation was commenced but Mr Ham could not be revived. He was declared deceased at 9.22am.

Post-Mortem Examination

This was carried out by State Forensic Pathologist, Dr Christopher Lawrence. In his opinion the cause of Mr Ham's death was pulmonary thromboemboli due to deep vein thrombosis following right hip replacement. I accept this opinion.

Investigation and Findings

This has been informed by:

1. Affidavits provided by Ms Strang.
2. An affidavit of registered nurse, Ms Bernadette Phillips-McGrath.
3. A review of Mr Ham's general practice and hospital records undertaken by clinical nurse, Ms L K Newman.
4. A report provided by Mr Pritchard.
5. A report provided by Dr A J Bell.
6. A report provided by orthopaedic surgeon Professor David A F Morgan.

The investigation shows that persons who have undergone total hip replacement surgery are at risk of suffering from thromboembolic disease (i.e. the formation of a clot or thrombus in a blood vessel which can be dislodged and carried by the blood to another vessel which it plugs or blocks). Its incidence is significantly greater if measures are not taken to reduce the risk. These measures, known as thromboprophylaxis, can be chemical and/or mechanical. The post-mortem examination has established that Mr Ham's death was directly attributable to a thrombus which evolved as a result of his hip surgery. This investigation has therefore focussed upon the adequacy or appropriateness of the steps taken to reduce the risk of this outcome.

I have already noted that Mr Ham, following his pre-operative assessment, was considered a low risk for developing a DVT. In his report, Mr Pritchard advises that in managing this level of risk he followed the 2016 guidelines of the Arthroplasty Society of Australia ("the Guidelines"). This meant that an AV Impulse Foot pump was utilised in the recovery suite, he was encouraged to mobilise on the first day post-surgery, that anti-embolic boots were employed and his aspirin was resumed post-surgery.

It is noted that the Guidelines include an Appendix which itemises those conditions which increase the risk of DVT or pulmonary embolism. These include '*Marked obesity*' and '*Known hypercoagulable states*'. Several observations need to be made.

First, the Guidelines do not define the term '*Marked obesity*', although it is my understanding that this phrase is commonly considered to be synonymous with 'morbid obesity' which means a BMI >40. Of relevance it is noted that the current edition of the Guidelines published in 2018 show a minor criteria increasing the risk to be '*Obesity, BMI >30*'. As I have noted Mr Ham's BMI was 33.26, thus less than the morbidly obese threshold but greater than the level set by the latest Guidelines.

Secondly, it is my understanding that polycythaemia is a condition which involves an increased concentration of haemoglobin in the blood. It gives rise to a 'stickiness' in the circulatory system and predisposes an individual to the formation of deep venous thrombi. In the result it qualifies as a hypercoagulable state and hence raises the question whether Mr Ham should have been treated under the Guidelines as being high-risk and hence warranting the use of a chemotherapeutic anti-thrombotic agent in response to that elevated level of risk.

Professor Morgan is an experienced orthopaedic surgeon based in Brisbane. He has assisted this investigation by providing a report upon Mr Ham's medical care and treatment. He offers these opinions upon the issue of risk:

1. That under normal circumstances the regime set by the 2016 Guidelines and applied by Mr Pritchard would have been appropriate for Mr Ham. However, in his case two factors were present which increased his risk of developing a DVT/PTE. These were his polycythaemia and his elevated BMI.

2. That when risk factors exist, the Guidelines include not only the use of compression devices with aspirin but also the use of supplemental anticoagulants such as Warfarin. In cases of very high risk inferior vena caval filters or umbrellas are also recommended.
3. That Mr Ham's '*...polycythaemia may not have been of any great magnitude and similarly, the elevated body mass index is not far above the accepted threshold. It is possible that these conditions influenced Mr Pritchard in not proceeding with the addition of another chemotherapeutic agent. Given the outcome and the benefit of hindsight, it is possible that Mr Ham's outcome may have been more gratifying had that bolstered regimen been recommended.*

It is self-evident that the Guidelines are the best yardstick to assist surgeons in managing the risk of DVT following hip replacement surgery. However, they are, as self-described, guidelines and do not require strict adherence. As they applied to Mr Ham the Guidelines identified obesity and his polycythaemia as factors relevant to his level of risk. However, strictly read Mr Ham did not seemingly qualify as being 'marked(ly) obese' and whilst he did qualify as having a 'known hypercoagulable state' that condition was only mild and had not required active treatment. All of this leads me to the conclusion that Mr Pritchard's decision to treat Mr Ham as being at low risk of DVT was not unreasonable and could not be fairly criticised. However, hindsight, as Professor Morgan has noted, now suggests that Mr Ham's tragic death may have been avoided if he had been assessed to be at high risk of DVT and more proactive steps taken in response.

Dr Bell has advised me that with one qualification the care and treatment provided to Mr Ham at the RHH was of a good standard. The qualification concerns the dosage of enoxaparin. I have noted above that on 26 September Mr Ham's dose was adjusted to 70 mg twice daily. Dr Bell advises me that at this time Mr Ham's renal function was recovering and hence an adjustment of his dose was not required. Instead he should have been maintained on the previous dose of 100 mg twice daily. I accept this advice and it follows that Mr Ham was under anti-coagulated. However, I am not able to make a positive finding that the outcome would have been different if the dosage had not been reduced.

This is the second case this year where I have had to investigate the death of a patient following hip replacement surgery. These cases should serve as a reminder to the public-at-large that complications do attach to this procedure which can lead to a catastrophic outcome and hence should not be embarked upon without serious contemplation.

Comments and Recommendations

The circumstances of Mr Ham's death are not such as to require me to make any comments or recommendations pursuant to Section 28 of the *Coroners Act* 1995.

I convey my sincere condolences to Mr Ham's family and loved ones.

Dated: 16th day of April 2019 at Hobart in the State of Tasmania.

Rod Chandler
Coroner