OFFICE OF THE STATE CORONER

FINDINGS OF INQUEST

CITATION:	**Inquest into the death of Shealah Ann Woolgar**
TITLE OF COURT:	Coroners Court
JURISDICTION:	Brisbane
FILE NO:	COR 3894/07
DELIVERED ON:	20 March 2009
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HEARING DATE(s):	24 – 25 February 2009
FINDINGS OF:	Coroner John Lock
CATCHWORDS:	CORONERS: Inquest – unexpected outcome of medical procedure, Mitral Valve heart surgery, perforation of heart during surgery, Blake's tubing

REPRESENTATION:

Counsel Assisting:	Ms J Rosengren
Greenslopes Private Hospital:	Mr J Allen instructed by Minter Ellison Lawyers
Doctors Conias and Tam:	Mr D Atkinson instructed by Flower and Hart Lawyers

CORONER'S FINDINGS AND DECISION

1. These are my findings in relation to the death of Shealah Ann Woolgar who died at Greenslopes Private Hospital on 22 August 2007 in an unexpected outcome of heart surgery. These findings seek to explain how the death occurred and consider whether any changes to policies or practices could reduce the likelihood of deaths occurring in similar circumstances in the future. Section 45 of the *Coroners Act 2003* ("the Act") provides that when an inquest is held into a death, the coroner's written findings must be given to the family of the person who died and to each of the persons or organisations granted leave to appear at the inquest. These findings will be distributed in accordance with the requirements of the Act and also placed on the website of the Office of the State Coroner.

The scope of the Coroner's inquiry and findings

2. A coroner has jurisdiction to inquire into the cause and the circumstances of a reportable death. If possible he/she is required to find:-
   (a) whether a death in fact happened;
   (b) the identity of the deceased;
   (c) when, where and how the death occurred; and
   (d) what caused the person to die.

3. There has been considerable litigation concerning the extent of a coroner's jurisdiction to inquire into the circumstances of a death. The authorities clearly establish that the scope of an inquest goes beyond merely establishing the medical cause of death.

4. An inquest is not a trial between opposing parties but an inquiry into the death. In a leading English case it was described in this way:- *"It is an inquisitorial process, a process of investigation quite unlike a criminal trial where the prosecutor accuses and the accused defends… The function of an inquest is to seek out and record as many of the facts concerning the death as the public interest requires."* [1]

5. The focus is on discovering what happened, not on ascribing guilt, attributing blame or apportioning liability. The purpose is to inform the family and the public of how the death occurred with a view to reducing the likelihood of similar deaths. As a result, the Act authorises a coroner to make preventive recommendations concerning public health or safety, the administration of justice or ways to prevent deaths from happening in similar circumstances in future.[2] However, a coroner must not include in the findings or recommendations, statements that a person is or maybe guilty of an offence or is or maybe civilly liable for something.[3]

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[1] *R v South London Coroner; ex parte Thompson* (1982) 126 S.J. 625
[2] Section 46 of the Act
[3] Sections 45(5) and 46(3) of the Act

The admissibility of evidence and the standard of proof

6. A coroners court is not bound by the rules of evidence because the Act provides that the court "*may inform itself in any way it considers appropriate."*[4] That does not mean that any and every piece of information, however unreliable, will be admitted into evidence and acted upon. However, it does give a coroner greater scope to receive information that may not be admissible in other proceedings and to have regard to its origin or source when determining what weight should be given to the information.

7. This flexibility has been explained as a consequence of an inquest being a fact-finding exercise rather than a means of apportioning guilt; an inquiry rather than a trial.[5]

8. A coroner should apply the civil standard of proof, namely the balance of probabilities but the approach referred to as the *Briginshaw* sliding scale is applicable.[6] This means that the more significant the issue to be determined; or the more serious an allegation; or the more inherently unlikely an occurrence; then in those cases the clearer and more persuasive the evidence should be in order for the trier of fact to be sufficiently satisfied that it has been proven to the civil standard.[7]

9. It is also clear that a coroner is obliged to comply with the rules of natural justice and to act judicially.[8] This means that no findings adverse to the interest of any party may be made without that party first being given a right to be heard in opposition to that finding. As *Annetts v McCann*[9] makes clear, that includes being given an opportunity to make submissions against findings that might be damaging to the reputation of any individual or organisation.

10. If, from information obtained at an inquest or during the investigation, a coroner reasonably believes that the information may cause a disciplinary body for a person's profession or trade to inquire into, or take steps in relation to, the person's conduct, then the coroner may give that information to that body.[10]

The evidence

11. It is not necessary to repeat or summarise all of the information contained in the exhibits and from the oral evidence given, but I will refer to what I consider to be the more important parts of the evidence.

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[4] Section 37 of the Act

[5] *R v South London Coroner; ex parte Thompson* per Lord Lane CJ, (1982) 126 S.J. 625

[6] *Anderson v Blashki* [1993] 2 VR 89 at 96 per Gobbo J

[7] *Briginshaw v Briginshaw* (1938) 60 CLR 336 at 361 per Sir Owen Dixon J

[8] *Harmsworth v State Coroner* [1989] VR 989 at 994 and see a useful discussion of the issue in Freckelton I., "Inquest Law" in *The inquest handbook*, Selby H., Federation Press, 1998 at 13

[9] (1990) 65 ALJR 167 at 168

[10] Section 48(4) of the Act

Introduction and issues for determination

12. Shealah Ann Woolgar was described by her daughter, Mrs Pike, as a sprightly and active 83 year old who lived independently and was able to care for herself. Her husband had passed away in 1981. She was generally well and in good health for her age. Mrs Woolgar knew she was suffering from a heart condition and in 2007 she was receiving treatment for this by way of medication. She remained unwell and subsequently after many tests were done it was decided that she should have mitral valve and tricuspid valve repair heart surgery. Mrs Pike confirmed that it was known that this was major heart surgery but, if it was successful, it would lead to a better quality of life for Mrs Woolgar. The operation was due to take place on 22 August 2007.

13. Mrs Pike was shocked when called by staff at the Greenslopes Private Hospital on 22 August 2007 to be told that her mother would not survive the operation. She was told by the cardiothoracic surgeon, Dr Robert Tam, that damage had been caused to the heart by a "needle" during the surgery. It is to the credit of Dr Tam that he was open about this adverse event with the family. It is also to his credit that he personally reported the matter to the Coroner that day and was open about what he thought was the cause of her unexpected death. Those communications assisted in the subsequent investigation including the autopsy examination.

14. Mrs Pike explained in her letter to the Coroner[11] that she was not angry or seeking to find blame but hoped that the outcome of the inquest would go some way to ensure it does not happen again. That is of course very much the focus of this inquiry. To have Mrs Woolgar's family, who have lost a loved one, adopt this attitude has been very helpful to the Court and staff, and for this I thank them. I hope to be able to provide some answers.

15. A bevelled end of a soft rubber tubing (known as "Blake's" tubing) was attached to a syringe and inserted into the left ventricle to test the competency of the repaired mitral valve. It was reported by Dr Tam that this was the device which had caused a perforation to the left ventricle. He attempted to repair the source of the rupture but was unsuccessful and Mrs Woolgar's death was caused by the resultant uncontrolled bleeding. The autopsy examination found a perforation in the left ventricle, at an area which showed evidence of attempts at surgical repair. Very early in the investigation Dr Tam provided information to the Coroner confirming this version of events.[12] The focus of the investigation was concentrated on how that could occur and what steps could and have been taken to reduce the chances of such an event occurring in the future. Dr Tam and the Hospital have been cooperative

[11] Exhibit C3

[12] Apart from the oral report to the Coroner on the day of Mrs Woolgar's death, his first statement (exhibit C4.1) is dated 20 September 2007. His second statement dated 18 September 2008 (exhibit C4.2) did not resile from this version of events or the cause of the rupture.

in providing statements of witnesses and providing answers to questions asked in the course of the investigation

16. Subsequently[13], Dr Tam suggested that as a result of his interpretation of aspects of the autopsy findings there was an alternative explanation for the rupture. He opined that Mrs Woolgar had suffered a myocardial infarct at least 24 hours prior to surgery, resulting in some necrosis of the heart wall, which then ruptured when she went off the bypass machine during surgery. He stated that ventricular rupture is a well recognised complication of a full thickness myocardial infarct especially in elderly females where the ventricular wall is thinner. That latter statement is undoubtedly correct. As a result it has become necessary to consider that alternative hypothesis and whether there was any evidence which could support a finding that Mrs Woolgar had suffered a heart attack of some nature in the anterolateral aspect of the left ventricle in the days leading up to her surgery. For the reasons that will be set out I am confident that this was not the case and Dr Tam's hypothesis is not supported by the evidence. In making that finding I do not intend to be overly critical of Dr Tam. Sometimes in complicated medical cases there is often no absolute conclusive answer. Most often it is about weighing up what is known and determining what is the most likely or probable conclusion. Because of the importance of the conclusions being made in a case like this, of necessity, the standard of proof must be at the higher end of the *Briginshaw* standard to which I have previously referred.

17. Furthermore, as is often the case where matters proceed to inquest, there is a complexity about the evidence coupled with a singular event that is difficult to comprehend. In this case it has perplexed Dr Tam and others as to how soft rubber tubing, used in the hands of an undoubtedly experienced and competent surgeon, during a part of surgery which is at the lower end of its complexity, could cause such a catastrophic event.

18. The issues which became the focus of the inquest included:

(a) The cause of the rupture of the left ventricle including consideration of the alternative hypothesis of Dr Tam;
(b) The circumstances in which the competency of the valve came to be tested with a soft rubber tube which was bevelled;
(c) Whether any processes or procedures have been implemented to prevent this sort of death from happening in the future.

The medical intervention leading up to the operation

19. In a general sense, the family's understanding of Mrs Woolgar's health issues is confirmed by what has been subsequently obtained from her medical advisors. She had a longstanding history of an abnormal heart rhythm known as atrial fibrillation. This manifested itself with symptoms

[13] In his letter to the Coroner dated 10 October 2008 (exhibit C4.3)

of palpitations and increasing shortness of breath. She was on Warfarin, an anti-coagulant, amongst other medication. Her General Medical Practitioner considered she needed specialist advice. Since May 2007 she had been under the care of Dr Paul Watson, who is a very experienced interventional cardiologist. Dr Watson provided a statement[14] and gave evidence. He had initially attempted to manage Mrs Woolgar's condition conservatively with anti-diuretic therapy. This therapy resulted in an improvement in her atrial fibrillation but her symptoms of palpitations and shortness of breath continued. He diagnosed through a clinical examination and a subsequent echocardiogram that she had significant mitral valve pathology with evidence of grade 3/4 eccentric mitral valve regurgitation (incompetence of the valve) associated with anterior leaflet/posterior leaflet override and prolapse.

20. Conservative treatment was not successful and a repeat echocardiogram conducted by Dr Watson revealed significant regurgitation and elevated filling pressures associated with ongoing mild pulmonary hypertension and associated tricuspid valve regurgitation.

21. Mrs Woolgar was admitted to the Greenslopes Private Hospital on 9 August 2007 (as a result of experiencing ongoing dyspnoea and symptomatic palpitations) on the advice of Dr Watson for further investigations to be carried out.

22. These investigations concluded that the mitral valve pathology was associated with the recent episodes of cardiac failure.

23. A preoperative coronary angiogram revealed only minor non-obstructive coronary artery disease and normal left ventricle function. This placed her in a suitable category for surgery to repair her mitral tricuspid valves. Her quality of life was becoming significantly affected and her nocturnal dyspnoea was described by Dr Watson as equivalent to a feeling of drowning. Conservative treatment is not effective as a long term solution for this condition. She was experiencing recurrent congestive heart failure as a consequence of severe mitral and tricuspid valve leakage, which conditions can have long term morbidity and mortality

24. The medical opinion was that the best option was to consider a surgical repair. This was discussed at length with Mrs Woolgar and her family who agreed with this course of treatment.

The operation

25. It was for this reason that Dr Robert Tam, a cardiothoracic surgeon became involved in Mrs Woolgar's care. Dr Tam first examined her on 16 August 2007 and concluded that Mrs Woolgar suffered from severe

[14] Exhibit C7

recurrent pulmonary oedema (fluid collecting in the lungs) secondary to the moderate to severe regurgitation through the mitral and tricuspid valves. By way of explanation, the valves in the heart maintain the unidirectional flow of blood by opening and closing depending on the difference in pressure on each side. The mitral valve is on the left side between the left atrium and left ventricle. Its role is to make sure the blood flows in a forward direction from the left atrium to the left ventricle. Mitral valve regurgitation results in the abnormal leaking of blood back through the mitral valve, from the left ventricle into the left atrium. The tricuspid valve is on the right side between the right atrium and the right ventricle. Its role is to make sure the blood flows in a forward direction from the right atrium into the right ventricle. Tricuspid regurgitation is leakage of blood backwards through the tricuspid valve each time the right ventricle contracts. Dr Tam ascertained that the regurgitation was due to prolapse of the anterior leaflet of the mitral valve secondary to chordal rupture.

26. Dr Tam's medical notes on the hospital file indicate that the risk of mortality from the operation was between 5 – 10%. Dr Watson would have suggested something lower in the region of 3% with morbidity complications in the range of 5 – 10%. Whatever is the case there is no suggestion of criticism about the decision to operate. Dr Gardner, a cardiothoracic surgeon who provided an independent expert report, said it was entirely appropriate.

27. The surgery was performed at the Greenslopes Private Hospital on 22 August 2007. Dr Tam was the surgeon, Dr Anthony Conias was his assistant and Dr William Soong was the anaesthetist. The two scrub nurses were Registered Nurses Dene Dixon and Kym Stuart. There was also two scout nurses and anaesthetic nurse present in the operating theatre. The operation records indicate that the surgery commenced at approximately 6:30 am. The heart was exposed by an incision through the left atrium and both valves were repaired. The mitral valve was repaired first by creating new chords to support the anterior leaflet of the valve secondary to the ruptured chords. The repair was further supported by an annuloplasty ring.

28. In accordance with routine practice, Dr Tam injected normal saline through both the mitral and tricuspid valves after the repair to test the competency of each valve. This is normally done with a 50ml syringe with a short piece of relatively soft plastic tubing placed over the nozzle of the syringe and saline is injected through the valve into the left ventricle to test for any leakage. In this case "Blake's" tubing was used. Dr Tam described how this occurred in his evidence with the benefit of a diagram of the heart that he had drawn. Similarly Dr Gardner showed how he would approach the insertion of the syringe. Essentially it was the same. From the presentation in Court, if the tubing went in too far in the direction that was shown, the tubing potentially would come into contact with the anterolateral portion of the left ventricle wall in about

the position that was identified by Dr Tam as the area of perforation that he tried to repair.

29. Dr Tam found that the valves were competent. The heart was then deaired and the left atrium was closed. Mrs Woolgar was taken off the cardiopulmonary bypass machine. In his operation report Dr Tam noted that the table echo showed competent valves. During decannulation, significant blood was seen accumulating in the pericardium. Cardiopulmonary bypass was re-established and Dr Tam found a small perforation at the base of the antero-lateral portion of the left ventricle. Dr Conias described it as the posterolateral aspect. It is much the same area as generally described by Nurse Dixon when he saw it and in the area of the perforation found by the pathologist Dr Van Vuuren. Dr Tam, Dr Conias, Nurse Dixon and Dr Van Vuuren were each asked to mark it on a diagram of the heart and placed it in about that position.

30. Dr Tam attempted to repair what he considered to be a perforation on three occasions. Both internal and external approaches were unsuccessfully made to repair the rupture. The difficulties were largely encountered due to the repeated tearing of the repairs through the frail haemorrhagic tissue. It is understood that once this complication of rupture occurs it is a very difficult one to deal with and the mortality rate is high.

31. Various therapies to improve the situation including blood products and inotropes (which are drugs which make the heart beat more strongly) were used but there was further bleeding from the perforated ventricular site followed by hypovolaemia (low blood volume) and hypotension (low blood pressure). Adrenaline boluses were finally used to continue the resuscitation of Mrs Woolgar but with only a temporary effect.

32. By this stage it was unlikely that Mrs Woolgar would survive. She was transferred to the intensive care unit (ICU) at about 12:15 pm. At this time there was heavy bleeding into the cardiac drains which had been inserted. She was intubated, ventilated and sedated. She continued to receive blood transfusions and adrenaline. Within a short period of time it was apparent that it was both impossible and futile to continue transfusing blood at the required rate. Her condition quickly deteriorated and Mrs Woolgar passed away about 25 minutes later at 12:40 pm. The rapid and profuse blood loss and low cardiac output proved to be fatal.

33. Following the operation, Dr Tam noticed that the soft rubber tubing he had used to test the competency of the mitral valve had been cut on a bevelled angle thereby having a pointier end. His operation report, no doubt completed soon after the events, concluded that the soft rubber tube at the end of the syringe had perforated the left ventricle during the testing. He reported the adverse event to the Coroner.

34. An autopsy was performed by Dr Van Vuuren on 24 August 2007 and the cause of death was found to be haemorrhage following surgical repair of the mitral valve. This was based on her examination of Mrs Woolgar's heart with the naked eye and microscopically, her review of the hospital notes, the operation report, and discussions with Dr Tam.

35. Her report describes her examination of the heart in detail. She observed evidence of the surgical intervention. Relevantly to this case she found felt-like material sutured in the left ventricle visible just below the valve leaflets. Upon removal of the material she noted haemorrhage in the endocardium. There was haemorrhage near the apex. On multiple cross sections of the heart, linear areas of haemorrhage were visible in the apex with a few similar sections on the posterolateral aspect of the left ventricle. Autopsy photographs[15] show the linear area of haemorrhage. Dr Van Vuuren gave evidence that the perforation was not full thickness. It started in the endocardium into the myocardium but not through to the epicardium. She did not find any perforation in the basal inferior wall, which was suggested in the Transoesphageal Echocardiogram (TOE) done during the operation.

36. Histology examination of samples of heart tissue found an epicardial haemorrhage with a few acute inflammatory cells. There were multiple foci of myocardial haemorrhage consistent with laceration. Some of those areas contained fibrin with acute inflammatory cells. Contraction band necrosis was present and in some areas associated with acute haemorrhage and some acute inflammatory cells. Small areas of fibrosis were scattered throughout.

The cause of the rupture

37. Ultimately this became the most significant issue to be determined. There is often no absolute in medicine and as many witnesses said anything is possible. There were a number of possible scenarios mentioned in the statements and evidence and some of these can be excluded with some degree of certainty.

38. In his report[16], Dr Gardner suggested that it could have been due to tearing in the papillary muscle from the artificial chords. However, Dr Tam saw no evidence of damage visually. Further, the TOE taken at the time of the operation, and which had only recently been found and produced, found no evidence of this. In light of this extra information, Dr Gardner said that this possible cause could be excluded.

39. Dr Gardner also said it could not be discounted absolutely that there had been disruption of the atrioventricular ("AV") groove and this caused extensive bleeding. As I understand it, this could occur if the heart was picked up or moved in the course of deairing and the AV groove put under tension. It is in the area of the AV groove that the

[15] Photograph 1052 of exhibit A3
[16] Exhibit D1

annuplasty ring is placed. He said that bleeding at the AV junction usually occurs by disruption where a prosthetic valve is used to replace the mitral valve. Whilst this was a repair, the annuplasty or physio ring used here he considered was still pretty rigid. He had looked at the autopsy photographs and could see blood in the AV groove and was not convinced that it had not played a part. Dr Gardner also considered the TOE done at the time of the operation alluded to blood at the back of the pericardium and a suggestion that it may have been adjacent to the coronary sinus (which is in the AV groove).

40. The evidence which contraindicates this possibility is threefold. Firstly, Dr Tam says he did not have to lift up the heart to deair it. Dr Tam stated that his usual technique does not involve touching the left ventricle in any way and he would only touch the left ventricle if the TOE identified a pocket of air in this region. As the TOE showed no air in this region, Dr Tam concluded that he did not touch the left ventricle during the deairing process. Dr Gardner agrees that the coronary sinus catheter would be nowhere near the possible site of the bleeding. Secondly, Dr Tam says that he did not see or note any particular calcification at the AV junction and he had no difficulties in placing the physio ring. There was some calcification noted on the TOE but nothing significant is mentioned in the autopsy report. On this issue, although discounted by Dr Tam initially in questioning, he agreed under examination by his counsel that there was some possibility the annulus damaged the ventricular wall. Thirdly, at autopsy the mitral valve showed evidence of repair but there was no haemorrhage noted in these areas and the suture lines were in place. The rupture that was found by Dr Van Vuuren was not full thickness and only went to the epicardial fat. Dr Gardner agrees this would contraindicate what he says on this point, but says they can co-exist.

41. Ultimately Dr Gardner ended his evidence with a comment that he was not convinced there has not been some AV groove disruption which may explain the loss of blood and blood tracking from one site to another and why the repair at the site where Dr Tam saw the perforation did not successfully stem the blood flow. Accepting his expert view I would have to consider that it is one that cannot be discounted but is less likely than other explanations which will be discussed shortly.

42. The next hypothesis and the one raised by Dr Tam was that an alternative explanation for the rupture was due to Mrs Woolgar having suffered a myocardial infarct at least 24 hours prior to surgery resulting in some necrosis of the heart wall which then ruptured when she went off the bypass machine. Dr Watson, Dr Gardner and Dr Van Vuuren all considered that this was unlikely or only remotely possible. During evidence Dr Watson was asked to consider the results of the various tests including Electrocardiograms (“ECG’s”), echocardiograms (“echo’s”), angiograms and the TOE which were conducted between

May and 22 August 2007. It is convenient to list them here and the general comments of Dr Watson:

(a) The ECG on 23 May 2007[17] noted atrial fibrillation ("AF"). It also noted that there should be consideration of an anteroseptal infarct.

(b) The ECG on 25 May 2007[18] noted AF and probable anteroseptal infarct. In relation to the ECG results from 23 and 25 May 2007, Dr Watson explained that the reports are computer generated and tend to overcall a potential diagnosis to highlight possible concerns. Although there is some territorial overlap with the anteroseptal and anterolateral wall of the left ventricle he considered there had not been an anterolateral infarct.

(c) The Pathology report with results from 23 – 24 May 2007[19] noted raised troponin levels. Again this result, combined with the ECG's, could be indicative of an ischaemic event but Dr Watson considered them to be non specific.

(d) The echo on 24 May 2007[20] was a pivotal investigation and showed conclusive evidence of mitral and tricuspid regurgitation. Dr Watson said the results of this test would pick up an ischaemic event and none was found. He considered that this excluded a significant infarct at this time.

(e) The ECG on 27 June 2007[21] noted AF. There was no suggestion of an infarct.

(f) The echo on 28 June 2007[22] showed no evidence of a myocardial infarct but showed worsening mitral valve and tricuspid valve regurgitation. It was at this stage that consideration for a more formal assessment for surgery was considered.

(g) The ECG's conducted on 15 and at 7:55 am on 21 August 2007[23] confirmed AF but again showed no evidence of an anterolateral infarct.

(h) The echo at 7:11 pm on 21 August 2007[24] showed the severe mitral valve and tricuspid valve incompetencey with a normal left ventricle and possible suitable for repair but unlikely that there was an infarct in the anterolateral wall.

(i) The angiogram on 16 August 2007[25] showed minor obstructive coronary disease but was not likely to cause symptomatic angina pain.

(j) The TOE conducted during the surgery on 22 August 2007[26] should have indicated an infarct if one had occurred during surgery.

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[17] Page 122 of exhibit E1
[18] Page 120 of exhibit E1
[19] Page 136 of exhibit E1
[20] The report generated from the echo is at page 159 of exhibit E1
[21] Page 114 of exhibit E1
[22] The report generated from the echo is at page 158 of exhibit E1
[23] Pages 111 – 113 of exhibit E1
[24] The report generated from the echo is at page 157 of exhibit E1
[25] Exhibit C7.1

43. Dr Watson also had the advantage of clinically examining Mrs Woolgar. He considered that it was unlikely that she had suffered an infarct in the anterolateral wall at the time of or in the days prior to the operation. The angiogram conducted by him on 16 August 2007 showed only minor coronary disease and this was consistent with that found at autopsy. He said, consistently with other evidence, that for there to be a full thickness rupture on the ventricle there would need to be a three to five day period for the development of necrosis and damage sufficient to weaken and tear the wall. He accepted that a rupture could occur at the anterolateral aspect but that there are more common sites where a rupture would occur. If a myocardial infarct had occurred he would expect that there would be evidence of necrosis at autopsy, it would have been seen on the TOE taken during the operation, it would be observable by the surgeon/s at the operation, there would be blood in the pericardium, it would show on the ECG monitoring in the days leading up to the operation and he would expect to have seen clinical signs of the infarct. There was no evidence of any of those things and his last examination of Mrs Woolgar indicated she was stable. He considered that her shortness of breath was consistent with her medical problems and was not likely to be evidence of an infarct. He accepted there can be silent infarcts (presumably one where the patient shows no clinical signs) but on all of the available evidence he considered it was very unlikely that Mrs Woolgar suffered a silent infarct.

44. Dr Van Vuuren was confident in excluding that Mrs Woolgar had suffered an infarct. Principally this was for four reasons. Firstly, Dr Van Vuuren was able to identify a perforation at the area that was sutured, presumably in an attempt to repair it. The perforation would need to be full thickness to have been caused by a myocardial infarct and in this case the perforation was not full thickness. It went from the endocardium to the myocardium but not through to the epicardium. Secondly, the perforation was clearly linear. In a myocardial infarct rupture the perforation would look quite different. Thirdly, there was no evidence of acute necrosis. For an infarct she would expect to find haemorrhage and acute inflammatory cells in greater number and more widespread than seen at autopsy. The contraction band necrosis that she found at autopsy could be the result of a number of causes that could have occurred here including a reperfusion injury, the body's adrenaline response, part of the resuscitation and/or hypovolaemic shock but she opined this necrosis was not associated with a myocardial infarct. Fourthly, the perforation she saw was traumatic in origin and did not have the appearance of an infarct. She discounted the suggestion that there could have been a silent infarct. Whilst she conceded anything is possible, her opinion was that the perforation with the associated contraction band necrosis that she observed was not from a myocardial infarct.

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[26] ExhibitE2

45. Dr Gardner was of the view that it was “extremely remote and most unlikely” that the rupture was from an infarct or silent infarct. He considered that there was no evidence of the degree of inflammatory changes at the site of the perforation indicative of an infarct. The echo’s and TOE performed prior to and at surgery showed no evidence of wall abnormality. Dr Gardner considered that the rupture would not have to be large but it would have to be full thickness to have been caused by a myocardial infarct, which was not the case here. He agreed that there would need to be a three to five day delay for the infarct to cause sufficient damage to rupture and he would expect this to show on the TOE and be evident at autopsy. It was not. He said that although it is possible for a rupture to occur spontaneously it was most unlikely and is the type of thing he would expect to happen after trauma.

46. Taking all of the evidence into account I can confidently exclude that the rupture was caused by a myocardial infarct.

47. Aside from the AV groove disturbance theory, that then leaves the explanation to be that the rupture occurred in the course of surgery by trauma. Dr Gardner was of the opinion that it was more likely that the rupture was trauma related and all the evidence pointed towards this. There were two scenarios suggested. Firstly, that it occurred through the use of forceps or other instruments during the operation. The second is that it occurred through the use of the rubber tubing as first reported by Dr Tam.

48. There are a number of reasons why the first scenario can be discounted. Certainly Dr Tam rejected it on the basis that it would have been obvious to him at the time. He also said that the pathologist did not find a surgical perforation. Further he said that surgical instruments went nowhere near the area of the perforation. For essentially the same reasons, Dr Gardner would think the use of a surgical instrument was less likely the cause than the rubber tubing. Dr Van Vuuren was of the same opinion. The evidence therefore supports a finding excluding forceps or other surgical instruments as responsible.

49. One of the difficulties with this case is accepting that a soft rubber tube, bevelled or not, could cause damage to the heart wall such that it perforated and ruptured. The most likely scenario, as accepted by Dr Gardner, is that if the tube is responsible, the tip of the tubing caused a partial perforation and when the heart was placed under pressure after the bypass machine was turned off, the perforation tore further.

50. The fact that soft rubber tubing could cause such a partial injury was the source of some conjecture. Dr Conias would have put it low on his list of culprits and he has seen other surgeons use much more rigid devices which are potentially more traumatic. Dr Van Vuuren said it was possible that the tubing caused the damage. Dr Gardner thought the tube was certainly soft and flexible but it could cause a partial

perforation. He said that the tip would be under some pressure and rigidity when the saline fluid is injected through the tube, and if at that moment the tip was resting on the heart wall, then it could be sufficient to disrupt the wall slightly and cause the initial perforation. Dr Tam also said he uses forceps towards the end of the tube to help guide it as it passes through the valve into the ventricle. That may also provide some degree of rigidity to that end.

51. Putting aside how the device is used; it is not unknown that soft rubber tubing introduced into the heart can cause damage to the heart wall such that a rupture occurs.[27] None of the clinicians in this case could say that they have seen it occur with this type of instrument before and I accept that there is nothing about the instrument in the context of how it is expected to be used which makes it inherently a problem. I accept what Dr Gardner says about the bevelling. He sees no need for it to be bevelled and it does have the potential to be more problematic.

52. On how the tubing should be used, Dr Gardner's view is that the principle is that it should not need to be inserted much past the entrance of the valve and only sufficient enough to push the leaflets back. Dr Tam speaks about pushing it one to two centimetres into the ventricle. There does not seem to be a lot of difference in the methods used although I had the impression that Dr Tam inserts it further into the ventricle than Dr Gardner would. Dr Gardner for instance said that two centimetres is not far, but he would still not need to go that far. I note in Dr Tam's statement to the Coroner dated 18 September 2008[28] that *"sometimes multiple testing is required to assess the valve and in doing so, the tube can accidentally push in too far. However, with the blunt soft tube, this is usually non traumatic to the ventricle."*

53. When assessing the probability that it was the tubing that caused the perforation the following can be noted:

(a) This was the first impression of Dr Tam who was in the best position of anyone to understand what occurred;

(b) That impression stayed with Dr Tam until only recently when he raised the possibility of a myocardial infarct;

(c) The area of the perforation found at autopsy and as identified by Dr Tam, Dr Conias, and Nurse Dixon is essentially in the same area;

(d) The action showed to the Court by Dr Tam in introducing the tubing into where the valve meets the left ventricle is the one that would be adopted by Dr Gardner. On that description Dr Gardner said that introducing the tubing at that angle the part of the wall of the left

[27] See the findings in relation to the inquests of Maureen Fitter (who died as a result of cardiac tamponade due to a peripherally inserted central catheter) and Jacinta Kate Robinson (who died as a result of cardiac tamponade due to leaching from the tip of a catheter).
[28] Exhibit C4.2

ventricle that the perforation was found would potentially be the place you would expect to see trauma if any trauma was to occur;

(e) The wall of the left ventricle for a woman of the age of Mrs Woolgar and with her medical conditions would be weak and thin and potentially more susceptible to damage, even from such an unlikely source. Dr Van Vuuren said that with congestive heart failure the wall can become thinner and you can get ischaemia in the wall which would probably be softer than normal. Dr Gardner said that a syringe full of saline might provide some additional rigidity and that the tip might have been sufficient to disrupt the endocardium slightly and injecting the saline under pressure produced a shearing effect to an aged myocardium which caused a split; and

(f) The tubing was not blunt but was bevelled and it is more likely that a pointed end could create more damage, even slight, without any significant force being used.

54. From all of the evidence, I can confidently come to a conclusion that the perforation of the left ventricle was caused by the use by Dr Tam of the bevelled rubber syringe in testing the mitral valve. The totality of the evidence points to that being the most likely cause and other alternatives have been excluded to my satisfaction.

The circumstances in which the competency of the valve came to be tested with a soft rubber tube which was bevelled

55. The extent to which the bevelling of the tubing is complicit in causing the perforation is not certain by any means but as a matter of common sense it must be of some significance. A small pointier end is more likely to lodge itself in a soft heart wall and so it would be more likely to cause damage in that way. Of course the tube had to be passed through the ventricle sufficiently far enough for it to come into contact with the heart wall in the first place.

56. Dr Tam immediately considered that the tube should not be bevelled and gave instructions that in future the tube should be cut off straight. Dr Gardner certainly sees no reason why it should be bevelled. Dr Conias could not disagree that cut straight the tubing would potentially be less traumatic. I think it can be taken that this is now a consensus view of the medical practitioners who gave evidence. The tubing should not be bevelled.

57. It would seem from the evidence of Nurse Dixon that he cut the tubing at an angle for the chest drains and then simply used the left over tube for the syringe tester. His evidence is that he would have used a similar method on 5 – 10 occasions, some involving Dr Tam, who had not raised it as an issue. It seems that it is for the surgeon to tell the nurses if something is not to their satisfaction and different surgeons may have different practices. On that basis I can see how it would be difficult for

this to be set out in a policy or procedure of a hospital. Dr Tam now ensures the tubing is cut straight across.

58. Dr Conias confirms that the practice was that the nurses just used bits and pieces left over for the tubing. He wondered how this bevelled tubing could have caused the damage and has seen more potentially traumatic devices used both bevelled or not.

59. I had some concerns about the response of Greenslopes Private Hospital to a request for information about what has been done procedurally within the Hospital to ensure something like this does not occur again. I received a more fulsome response just prior to the inquest which, together with the evidence I have heard, now satisfies me. It would seem that the surgeons are not employees of the Hospital. Dr Houston, the Director of Medical Services at Greenslopes Private Hospital, discussed the case with Dr Tam. He reviewed the event from the perspective of hospital processes as they related to patient preparation. This was an issue of cardiac surgical technique that was a matter for the surgeon to control. An open audit process also concluded that no steps needed to be taken regarding changes to processes or equipment. The use of bevelled or straight tubing is a matter for the surgeon to decide upon and to change if that is what the surgeon requires.

60. In this case Dr Tam accepts the evidence of Nurse Dixon that bevelled tubing has probably been used by him in the past and was not commented upon by him at the time or made part of any instructions to Nurse Dixon or others present at the operation. He says it would normally be blunt. His explanation as to how he missed it on this occasion was not altogether satisfactory. It seems he may not focus on the end of the tube and he does not check every item of equipment, but he did say that ultimately he is the one accountable. He agreed that he had not noticed it was bevelled on previous occasions and never gave directions one way or the other. He simply wanted soft tubing which is what he got and I do not think the issue of it being bevelled or not ever crossed his mind, probably because he did not consider it was a risky instrument one way or the other.

Referral to the Medical Board of Queensland

61. I have come to a conclusion that I should not refer Dr Tam to the Medical Board of Queensland. The threshold for my doing so is quite low but after hearing the evidence I do not think it could be said that reasonable skill was not used. Mrs Woolgar's death was a tragic loss for her daughter. Her death occurred in an unusual manner. She was having complicated and complex heart surgery. She was an elderly woman who had congestive heart failure and atrial fibrillation. Her heart was stable enough but there would be risks attached to the operation which were discussed with her. There was a risk of mortality of around 3%, although Dr Tam put it higher than this. The course of the surgery involves very invasive opening of the chest cavity and heart. The

patient is on a bypass heart machine. Very delicate surgery takes place. Towards the end of the surgery the accepted practice is to test the valves by injecting saline through a syringe with rubber tubing attached. That testing was at the lower end of complexity of the surgery and it would not be expected that something could go wrong in the way that it occurred here. In this case it did.

62. Dr Tam must have placed the rubber tube into the left ventricle far enough to reach the ventricle wall. It could not be damaged otherwise. Dr Gardner would only have the tip just inside the ventricle sufficient to separate the leaflets. Dr Tam would seem to place it in further than that, at least 1 to 2 cm. Perhaps he and other surgeons who use that technique might want to consider the adverse outcome here. There is no doubt that there is a risk of the tubing coming into contact with the heart wall. That risk is generally managed by using a soft tube. In this case it was bevelled and this may have been a contributory factor. It would seem that the issue of whether it is used as bevelled or not is for surgeons to decide. Objectively, I can accept that the tubing is not inherently dangerous or unsuitable. There is no evidence which suggests that undue force was used. It has simply been placed further into the ventricle than may have been necessary or intended. That is the main problem. If there had been no contact then there would not have been a potential for a perforation but it is a fine line.

63. The bevelled point came into contact with the left ventricle and a small perforation occurred. Dr Tam accepts responsibility for the fact that he did not notice the bevelled point. This may not have been evident to him at the time. It was probably caused by the tip resting on the heart wall and becoming more rigid as the saline solution passed through which caused the initial damage. When the heart was placed under pressure, after the bypass machine was turned off, the perforation extended on the same line through to the epicardium and then the bleeding occurred.

64. Although I can confidently conclude on the balance of probabilities that it was the tubing that caused the perforation, I also have to consider the evidence of Dr Gardner where he repeated on a number of occasions that it could not be discounted absolutely that there had been disruption of the AV groove which caused or contributed to the extensive bleeding. That may be one of the risks that attach to this type of surgery. In those circumstances, I do not consider it appropriate to make the referral to the Medical Board of Queensland.

Findings required by section 45

65. I am required to find, as far as is possible, who the deceased was, when and where she died, what caused the death and how she came by her death. I have already dealt with the last of these issues, being the circumstances of Shealah Ann Woolgar's death. As a result of considering all of the material contained in the exhibits and the

evidence given by the witnesses, I am able to make the following findings in relation to the other aspects of the death:

(a) The identity of the deceased was Shealah Ann Woolgar;
(b) The place of death was Greenslopes Private Hospital, Greenslopes, Queensland;
(c) The date of death was 22 August 2007;
(d) The formal cause of death was:
    1(a) Haemorrhage, due to, or as a consequence of
    1(b) Mitral valve disease (surgically repaired)

Concerns, comments and recommendations

66. Section 46 of the Act provides that a coroner may comment on anything connected with a death that relates to public health or safety, the administration of justice or ways to prevent deaths from happening in similar circumstances in the future. The state of the evidence is such that no such comments or recommendations can be made. Dr Gardner made the comment that any intra ventricular manipulation with tubular structures needs to be done with the utmost care and that the devices used should be the least traumatic as possible and manipulated with great care. That is a fairly obvious statement and hardly surprising. The extent to which this very adverse outcome can provide some clinical education for future purposes may be limited. Dr Conias referred to the fact that it has been discussed around the profession and it did form part of a cardiac review of cases conducted by Greenslopes Private Hospital. I will send a copy of this decision to the Royal Australasian College of Surgeons on the basis it can be used to advance some areas of technique or promote discussion within a clinical education context.

67. My condolences are expressed to Mrs Pike and the family and friends of Mrs Woolgar.

I close this inquest.

John Lock
Brisbane Coroner
20 March 2009