OFFICE OF THE STATE CORONER

FINDINGS OF INQUEST

CITATION:	**Inquest into the death of Maureen Fitter**
TITLE OF COURT:	Coroner's Court
JURISDICTION:	Brisbane
FILE NO(s):	COR 2351/05(6)
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HEARING DATE(s):	11, 12 & 13 July 2007
FINDINGS OF:	Mr John Lock, Coroner
CATCHWORDS:	Coroners: Inquest, Hospital/health procedures death, Cardiac Tamponade, Peripherally Inserted Central Catheter

REPRESENTATION:

Counsel Assisting:	Ms J Rosengren
Counsel representing Dr Brown & Dr O`Connell:	Mr D Tait SC instructed by Blake Dawson & Waldron Lawyers
Counsel representing Holy Spirit Northside & Dr Lloyd and T. Kratzmann:	Ms S Gallagher instructed by Minter Ellison Lawyers

The *Coroners Act 2003* provides [1] that when an inquest is held, 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 are my findings in relation to the death of Maureen Fitter. They will be distributed in accordance with the requirements of the Act and a copy will be sent to the Office of the State Coroner and placed on the State Coroner's website.

Introduction

Mrs Fitter had been admitted to the Holy Spirit Northside Hospital for treatment for polyps found in her large intestine. A colonoscopy performed in Bundaberg found several polyps of which all but one had been removed. This polyp was removed by a laparoscopic right hemicolectomy on 15 September 2005 at Holy Spirit Northside. The surgery was uneventful but some 2 days later Mrs Fitter developed abdominal distension which was being treated.

Due to a deterioration in her nutritional levels, a physician namely Dr Wagenaar ordered Total Parenteral Nutrition (TPN) fluids to be commenced. This necessitated the insertion of a Peripherally Inserted Central Catheter (PICC line). This was performed by a radiologist, Dr O'Connell on 22 September 2005. The PICC line was inserted into a vein of her right arm and passed through the arm and around into the heart, with the intention of an optimum placement resting at the junction of the superior vena cava (SVC) and the right atrium. Such a placement minimises significantly the chance of the PICC line perforating the wall of the heart.

On 24 September 2005 Mrs Fitter's condition deteriorated rapidly and unexpectedly, and within hours and despite Intensive Care Unit (ICU) treatment, she suffered a cardiac arrest and passed away.

A post mortem examination found TPN in the pericardium and the pathologist opines that the cause of death was due to a cardiac tamponade as a consequence of a ruptured heart as a consequence of total parenteral nutrition.

These findings seek to explain how the death occurred and consider whether any changes to health policies or practices could reduce the likelihood of deaths occurring in similar circumstances in the future.

The Coroner's Jurisdiction

Before referring to the evidence I will say something about the nature of the coronial jurisdiction.

[1] s 45 *Coroners Act 2003*

The scope of the Coroner's inquiry and findings

A coroner has jurisdiction to inquire into the cause and the circumstances of a reportable death. If possible he/she is required to find:-

- whether a death in fact happened;
- the identity of the deceased;
- when, where and how the death occurred; and
- what caused the person to die.

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.

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.* [2]

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.[3] However, a coroner must not include in the findings or any comments or recommendations statements that a person is or maybe guilty of an offence or is or may be civilly liable for something.[4]

The admissibility of evidence and the standard of proof

Proceedings in a coroner's court are not bound by the rules of evidence because the Act provides that the court "*may inform itself in any way it considers appropriate."*[5] 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 provenance when determining what weight should be given to the information.

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.[6]

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[2] R v South London Coroner; ex parte Thompson (1982) 126 S.J. 625
[3] s46
[4] s45(5) and 46(3)
[5] s37
[6] *R v South London Coroner; ex parte Thompson* per Lord Lane CJ, (1982) 126 S.J. 625

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.[7] This means that the more significant the issue to be determined, the more serious an allegation or the more inherently unlikely an occurrence, the clearer and more persuasive the evidence needed for the trier of fact to be sufficiently satisfied that it has been proven to the civil standard.[8]

It is also clear that a coroner is obliged to comply with the rules of natural justice and to act judicially.[9]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*[10] 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.

The investigation

The matter was investigated by the State Coroner's office and it was determined that an inquest should be held. A pre-inquest conference was held in Brisbane on 24 April 2007. Ms Rosengren was appointed Counsel Assisting. Leave to appear was granted to the Holy Spirit Northside Hospital and its nurses and medical staff and at the hearing, Ms S. Gallagher instructed by Minter Ellison Lawyers appeared. Leave was also granted to Mr Tait SC instructed by Blake Dawson Waldron Lawyers to appear on behalf of Dr J Brown and Dr T.B. O`Connell. The family of Mrs Fitter was not separately represented but they consulted with those assisting me before and throughout the inquest. The inquest then proceeded over three days commencing on 11 July 2007. Nine witnesses gave evidence and 37 exhibits were tendered. After legal argument, 2 exhibits were withdrawn by consent.

Issues and findings to be determined

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

One of the most significant findings at the autopsy was a collection of 300mls of white milky fluid around the pericardium which is the membrane covering the heart. The white milky fluid was analysed as being very similar to TPN. The TPN was being introduced by the PICC line. The autopsy investigation found evidence consistent with a perforation of the right ventricular wall by the PICC line. The mechanism of how and when this occurred was the main focus of the investigation and the inquest and is an issue of some controversy.

The second main issue for investigation was whether the diagnosis of cardiac tamponade should have been made prior to Mrs Fitter's death and in circumstances where treatment could have been provided which would have

[7] *Anderson v Blashki* [1993] 2 VR 89 at 96 per Gobbo J
[8] *Briginshaw v Briginshaw* (1938) 60 CLR 336 at 361 per Sir Owen Dixon J
[9] *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
[10] (1990) 65 ALJR 167 at 168

enabled her to survive. This also involved an assessment of the level of care provided to her by nursing and medical staff. Ultimately, the findings on that aspect of the enquiry are not controversial.

The Evidence

I will commence by making some general findings of fact which are not controversial and which place some of the issues in context.

Medical Procedures up to 25 September 2005

In early September 2005, Mrs Fitter had a colonoscopy performed in Bundaberg, at which time several polyps were found. The local specialist was able to remove all but one. She was referred to Dr R. Roberts, a gastroenterologist in Brisbane, for removal of this final polyp which was in the part of the large intestine called the caecum. Dr Roberts[11] performed a colonoscopy on 14 September 2005 but due to the location of the polyp he was unable to remove it.

A further referral was then made to Dr Andrew Stevenson, a colorectal surgeon[12]. Treatment options were discussed and Mrs Fitter decided to undergo a surgical removal of the polyp because she was concerned about the possible development of cancer. Surgery was performed on 15 September 2005 by Dr Stevenson. A laparoscopic right hemicolectomy was performed, which essentially removes about half the large intestine including the section where the polyp was situated.

The surgery itself was uneventful. However some 2 days later, Mrs Fitter developed upper abdominal distension. An x-ray showed this was most likely due to an ileus or a small bowel obstruction. An ileus is an obstruction of the bowel that results when the intestinal contents back up because peristalsis fails. Peristalsis is the successive waves of involuntary contraction passing along the intestine and forcing the contents onward. The other possibility was an actual occlusion or twisting of the intestine causing the obstruction.

Dr Stevenson requested the nursing staff to insert a Nasogastric (NG) tube to decompress any potential gastric dilatation. This was unsuccessful because of a previous procedure to treat reflux called a Nissen fundoplication had been performed in 1996. However, her bowels had opened and on reviews by Dr Stevenson over 18 and 19 September 2005, she was reasonably comfortable albeit with some continued moderate distension.

In the early evening of 19 September 2005, Dr Stevenson made a telephone call to the ward with regard to another patient and at the same time also enquired of Mrs Fitter. He was informed that Mrs Fitter had developed shortness of breath. Dr Monica Wagenaar, a physician was requested to review her.[13] Dr Wagenaar arranged a chest x-ray to exclude pulmonary

[11] Statement is exhibit B12
[12] Statement is exhibit B15
[13] Statement is exhibit B17

oedema and it showed no abnormalities. It was her opinion that the shortness of breath could be explained by the distended abdomen.

Dr Stevenson reviewed her the following morning on 20 September and an abdominal x-ray was performed which showed continued gastric and small bowel distension. Dr Stevenson considered Mrs Fitter still required a NG tube and because of the previous unsuccessful attempts to insert one, he arranged for Dr Roberts to insert it under a general anaesthetic later that day. This resulted in significant improvement in related symptoms over the following 12 hours.

On 22 September 2005, a further CT scan of the abdomen was done which Dr Stevenson thought still showed an ileus or small bowel obstruction. Mrs Fitter was reviewed by Dr Wagenaar that morning. Dr Wagenaar thinks that family members were present for this consultation and were generally concerned about Mrs Fitter's condition, including her nutrition levels. She gave evidence that Mrs Fitter had been essentially fasting for a period of at least 9 days, her phosphate levels were into a very low range and it was not likely that she would get any reasonable nutrition in the next few days. Dr Wagenaar has recorded in the chart that she considered Mrs Fitter to be malnourished and has explained in her statement that whilst she would not ordinarily write this as a diagnosis in a patient's chart, she felt that in Mrs Fitter's case, it was becoming a second significant problem. For this reason she ordered TPN fluids to be commenced at a rate of 20ml/hr and this necessitated the insertion of the PICC line.

The PICC line was inserted by Dr O`Connell and I will refer to his evidence later in this decision.

On the evening of 22 September Mrs Fitter developed atrial fibrillation (irregular rapid breathing of the upper chambers of the heart). Dr Wagenaar reviewed her on 23 September and looked for the usual precipitants including electrolytes and found her to be hypokalemic (a condition where the body fails to retain potassium). An electrocardiograph or ECG was taken which showed ischaemic chambers but this seemed to have resolved on a subsequent ECG.

Dr Stevenson reviewed Mrs Fitter the following morning on 23 September. She continued to have a distended abdomen but was not complaining of pain. He arranged for a gastrografin follow-through which is a radiological investigation to determine whether Mrs Fitter's continuing problems could be explained by an ileus or small bowel obstruction. A clear distinction could not be made so Dr Stevenson made arrangements for Mrs Fitter to return to the operating theatre on Monday 26 September for a laparotomy if her symptoms did not resolve prior to then.

Dr Stevenson arranged for Dr David Clark to cover for him over the weekend and Dr Wagenaar arranged for Dr Jenny Brown to cover for her. Dr Clark[14] saw Mrs Fitter on his morning rounds on 24 September and Dr Stevenson

[14] Statement is exhibit B3

telephoned him at approximately 2pm to enquire as to her well being. He was informed that there had been no clinical changes with respect to her abdomen and for this reason he left the plan in place to return her to the operating theatre on the Monday.

Dr Brown saw Mrs Fitter because Dr Wagenaar had told her that she was concerned about her potassium and fluid balance, her TPN management and the large amount of nasogastric contents she had aspirated. Dr Brown considered her condition to be stable but because of the large nasogastric losses she increased the TPN to 60ml/hr.

Findings as to medical procedures up to 25 September 2005

I will briefly note that no matters arose during the inquest or the earlier investigation which would have anticipated any adverse findings concerning Mrs Fitter's treatment during the course of the various medical procedures in Bundaberg and then at Holy Spirit (other than the PICC line insertion). Members of the family had some firm concerns regarding her treatment by nurses and doctors whilst an inpatient. There may be some issues concerning communication of decisions and the exchange of information but these are not strictly related to the cause of death and are not within the parameters of issues for which I could make any recommendations.

In any case, Dr Barnett from the Hospital, met with the family and it is apparent from Exhibits C16 and C17 that those issues were discussed and he passed them on to the nursing administration. Areas regarding poor communication with medical staff were to be taken to the Medical Advisory Committee.

There was an issue raised by the family concerning whether an X-ray of the chest should have been taken after insertion to ensure that the site of the tip was known. Dr O'Connell used an Image Intensifier to assist in guiding the catheter along the vein to the right atrium. This is effectively an X-ray taken in real time and at the conclusion the final image is taken and kept. This was the image which was the subject of his report referred to earlier.

Dr Clarke agrees. The image Intensifier is a better system for a PICC line and a chest X-ray would not provide or would be unlikely to provide further information.

In the evidence from Mrs Coad, the family raised the issue of a protocol that stated that X-rays be taken at the time of insertion of a PICC line and subsequent to insertion. She stated she had a meeting with the doctor in charge of ICU after the death and saw a document marked as a "Protocol" which dealt with a policy of subsequent X-rays for PICC lines being done. In evidence there was a page of the medical records placed immediately before page 157 headed "Peripherally Inserted Central Catheters (PICC) - Quick Reference". This seems to be a reference for nursing staff. Relevantly, it states "Ensure you have confirmation of correct tip placement before

connecting fluids to the PICC – check in chart for CXR report."[15] This document makes no reference to subsequent X-rays being done. This was not the document that Mrs Coad recalls seeing.

Mrs Coad was also shown Exhibit C7 which is the Holy Spirit Hospital Policy and Procedures Manual on PICC lines. She says this was not the document shown to her as it was headed as a Protocol. It also makes no reference to a policy of requiring subsequent X-rays to be performed. Exhibit C8 is a more recent version of C7 but again makes no reference to subsequent X-rays being performed. Exhibit C9 is headed a "Protocol for Unblocking Occluded PICC" and may have been a document shown to Mrs Coad but it also makes no reference to subsequent X-rays being taken.

No "Protocol" dealing with subsequent X-rays being taken was identified. Mrs Coad denied that what was being shown to her or discussed was a protocol dealing with future care.

I am satisfied that at the time of death there was no policy or protocol in place that stated that subsequent X-rays should be done. All medical staff gave evidence that the best X-ray was the Image Intensifier and that subsequent X-rays would not have been indicated.

The autopsy found that the small intestine and part of the large bowel were markedly distended with gas but they were surgically joined and the surgical sutures were intact. There was no leakage of fluid or twisting or obstruction of the bowel. The ileus (or obstruction of the bowel) was clearly present on clinical findings but there was no evidence from the autopsy of a reason for this. Further the Hospital medical file and notes clearly show that, at least in a clinical sense as distinct from some of the communication issues, both medical and nursing staff were attentive to her needs, as attested by the number of consultations, tests, scans, referrals to other specialists and procedures which were performed to alleviate the position.

Dr Boots gave evidence in relation to the episode of atrial fibrillation as being an indicator of the catheter impinging on the heart wall or valve or of the cardiac tamponade that was to come. He said that as she was a very sick lady with a severe gut obstruction and exceedingly low potassium both of these conditions in their own right would be a cause for atrial fibrillation. He considered that doing an ECG would not have told anyone very much about the tip of the catheter. Clinically he would not have called for a chest x-ray at that time either.

I note that Dr Roberts' report to Dr Strahan of 14 September 2005 states that Mrs Fitter may be at more risk to remove the residual polyp than through conservative observation but the risks were discussed with Mrs Fitter and there was a verbal request by Dr Roberts it seems, to have the operation. There is no issue concerning the need for the operation.

[15] Document tendered as exhibit C24

Whatever may be the case, the operation to remove the polyp and the subsequent treatment for the abdominal distension were only indirectly related to the cause of death in that the need for a PICC line would not have occurred but for the operation and post operative complications. The further treatment may very well have resolved the ileus or it may have resolved itself.

Events leading up to death

Accepting that the cause of death is due to a cardiac tamponade, it is helpful to explain in general what that is. The heart is covered by a membrane called the pericardium which is a thin doubled layered sac. It usually has a small amount of about 20mls of fluid contained within the layers and that area is called the pericardial space. The purpose of the fluid is to lubricate the 2 surfaces which constantly rub together.

Increased fluid in the pericardial sac results in increased pressure and this interferes with the mechanism of the heart's pumping action and prevents the ventricles of the heart from fully expanding so they cannot adequately fill or pump blood. If the fluid builds up there is a compression of the heart caused by blood or fluid accumulating in the space between the heart muscle and the pericardium. Dr Olumbe describes it as like a bear hug around the heart which restricts it.

A large collection of fluid is life threatening and is known as a cardiac tamponade. The efficiency of the heart's pumping mechanism deteriorates and lowers the body's blood pressure with low stroke volume, shock and often death within hours. In this case 300mls of TPN fluid were found in the pericardial sac at the autopsy so the situation was very serious.

The condition if identified must be treated as a medical emergency. There are no specific laboratory tests to diagnose a tamponade although an echocardiogram will help establish the diagnoses. Many of the signs of tamponade are non specific in that they may also indicate other conditions. It is treated by needle evacuation of the fluid guided by ultrasound which has the effect of lowering the pericardial pressure. If the condition is diagnosed and treated promptly then there is a reasonable prognosis for survival.

At about 11.45pm on 24 September 2005, Registered Nurse Trevor Kratzmann[16] went into Mrs Fitter's room to aspirate her NG tube. She complained of a sore throat and requested pethidine which Nurse Kratzmann declined after reviewing her medication chart. About half an hour later, Nurse Kratzmann went in to see Mrs Fitter again at her request and she complained of feeling hot, a sore throat and difficulty hearing. She was pale, sweating profusely and was cold and clammy to touch. Her observations were of concern to Nurse Kratzmann. He requested another nurse to perform an ECG (RN Shaw[17]) and a short time later phoned Dr Ben Lloyd in the

[16] Statement is exhibit B8. RN Kratzmann was not able to be contacted or served with a summons to appear. However his statement is corroborated by the hospital medical notes and other witnesses on most points.
[17] Statement is exhibit B14.

Intensive Care Unit (ICU) and explained to the doctor that he was concerned about her condition. Dr Lloyd immediately attended and in summary introduced some treatment regimes, sought advice from the ICU consultant and made arrangements for Mrs Fitter to be transferred to ICU.[18] She arrived in ICU at about 1.30am, and she arrested shortly before 2am, CPR was performed for about 10mins and was ceased after it was decided that further attempts were unlikely to be successful.

In his evidence Dr Lloyd stated that he wished to amend his previous statement and thought that death was more likely to have been around 1.45am to 2am and not 2.15am. He considered that on the symptoms he was faced with that the most likely diagnosis was either pulmonary embolism or a myocardial infarct. He did not consider cardiac tamponade as it was uncommon and her reasons for being in hospital were to do with her bowel not her heart. He did not consider problems associated with the PICC line as the usual problems occur at insertion and this was some days later.

He was asked about an X-ray being taken which may have indicated that a cardiac tamponade was occurring. He had considered and made a telephone call for a Computer Tomography Pulmonary Angiogram (CPTA) but the call was not answered. In any event she needed to be stabilised first for that to occur and had to be taken to the ICU for stabilisation. She arrested soon after she got to ICU.

Dr Rob Boots is highly qualified practitioner in intensive care. He examined the hospital file and other documents and provided three reports and gave evidence.[19] Dealing only with the issue of the treatment in the ICU, he said that given that perforation by the PICC line must have occurred some time after insertion and with the sudden development of symptoms there was little opportunity for a clinical diagnosis of cardiac tamponade to be made. There was no haemo dynamic evidence of tamponade and it would not be on the top of his list particularly in a person who was not a heart patient. In her condition, being very sick, and with a nasty bowel obstruction, the restriction on cardiac output by the amount of detected TPN fluid would have led rapidly to cardiac arrest.

Dr Boots considered that resuscitation procedures followed were in line with current standards of practice and Dr Lloyd's treatment was appropriate. He said that her chart reads as a pulmonary oedema or cardiac arrest and it is noted that this was the assessment of Dr Lloyd.

On the issue as to whether a chest X-ray should have been done when she deteriorated which may have indicated the fluid in the pericardium he said the X-ray would not necessarily show the fluid. In any event evacuating the fluid was not a straightforward procedure. It requires much training and had many risks. You could not run in blindly.

[18] Statement is exhibit B9
[19] Exhibits D1,D2 & D3 are his reports.

On the basis of this evidence and a consideration of all other relevant evidence the court is satisfied that the treatment provided by nursing and medical staff on the night of 24 September and the early hours of the 25 September 2005 were appropriate and in line with current standards of practice and no adverse findings or recommendations should be made.

Issues concerning the PICC line

Accepting for the moment the opinion of the pathologist that there is evidence consistent with a perforation of the right ventricular wall by the PICC line, the substantial issue of controversy is how the tip of the PICC line could have ruptured the wall of the right ventricle if it was in fact resting at the top of the atrium.

The other significant issue for determination is when this occurred. These issues will now be explored.

Exhibit C18 is a diagram of the heart. The heart has 2 upper chambers, called the right and left atria and 2 lower chambers called the right and left ventricles. The right atrium receives blood from the upper and lower body through the SVC and the inferior vena cava (IVC) respectively. The right atrium opens into the right ventricle through the tricuspid valve which only allows the blood to flow from the atrium into the ventricle but not in the reverse direction.

As is known a few days before Mrs Fitter's death, Dr O'Connell inserted a double lumen PICC line. It is a long, thin, flexible catheter which is inserted into one of the large veins of the arm near the bend of the right elbow. It is then advanced through increasingly larger veins, towards the heart. An examination of the PICC line itself shows it is of a soft plastic/silicone nature with the tip being even softer and it is difficult to see how it could of itself, cause a perforation of the relatively thick wall of the heart. In this case, there is evidence of a perforation and it would seem from the medical literature such instances occur, albeit rarely.

The catheter[20] was an Arrow brand PR-05052-LW and 50 cm in length. A sticker in Mrs Fitter's chart[21]at pg 61 shows that the exposed catheter length once it was inserted was "0". This means that there was no catheter left outside the skin and the entire 50cm had been inserted.

Dr O'Connell provided a statement to the Coroner[22] and gave evidence at the inquest. Not unusually, he has no independent recollection of performing this procedure at 3.20 pm on 22 September 2005. In his statement he describes his usual practice when inserting such a line under image intensification. Following insertion, a final image from the Image Intensifier was taken and the report of this[23] states that the tip of the PICC line *"lies in the right atrium"*.

[20] An example is exhibit C19
[21] page 61 of medical records
[22] Statement is B11
[23] page 27 of medical records

Dr O'Connell explains in his statement that he has reviewed a copy of that x-ray and considers that more accurately it in fact shows the position of the tip being in the right atrium/SVC junction which is in a slightly higher position than that recorded in the report.

At the end of the catheter tube outside the body, each lumen has a special cap to which a drip line or syringe can be attached. There is also a clamp to keep the tube closed when it is not in use.

There is little controversy as to the methods adopted to insert the PICC line. All witnesses agreed with the appropriate method and there is nothing to suggest this was not the method used by Dr O'Connell.

There is however, a wide divergence of opinion among the experts as to a number of crucial issues. These include:

1. The positioning of the tip of the PICC line as shown on the Image taken from the Image Intensifier;
2. When or even if a perforation of the right ventricle occurred from the PICC line; and
3. The appropriate length of catheter introduced.

The Position of the Tip of the PICC Line and length of catheter introduced

Dr O'Connell examined the X-ray in Court and maintains the tip of the catheter can be seen at the junction of the SVC and right atrium.

From a lay person's point of view what can be seen and only when it is pointed out, is a line progressing to a slightly darker and thicker point and then follows what seems to be a discernible line further down into what would be the right atrium. The bottom of the right atrium is not seen. All experts agree that the quality of the imaging is faint.

Dr O'Connell sees the tip just at or above the darker point described above.

Dr Robert Clarke is an experienced radiologist. He provided 2 reports and gave evidence.[24] In his first report dated 2 July 2007[25]he states that the X-ray supplied does not clearly indicate where the tip terminates. He said there was a faint impression of a catheter passing off the bottom of the film beyond the right atrium.

In his second report of 6 July 2007[26] he altered his position as to the PICC line position. He reviewed 10 separate patients who had Arrow catheters inserted and reviewed an uninserted catheter on fluoroscopy. He now says that he agreed with Dr O'Connell that the tip lies at the junction of the SVC and right atrium. The perceived faint linear density which appears to lie medially cannot be accounted for. On the basis that there was no

[24] Reports are exhibits D5 & D6
[25] exhibit D5
[26] Exhibit D6

redundancy of catheter it would be impossible for the catheter to have migrated more distally.

In his evidence Dr Clarke said he had considered further material and information he had been given and changed his view as set out in his second report. He says that in his opinion it remains unclear as to the position of the tip and that the density lying further from where he thought the tip was, may be the catheter.

Dr Clarke gave evidence that taking into account a person of Mrs Fitter's height and weight and the position of insertion in her arm, that it would take about 40 cm of catheter or a bit under to reach the junction of the SVC and the right atrium. On that basis he also thinks there was too much catheter placed.

Dr Clarke raised an issue of possible looping of the catheter. He said that looping would be difficult to reproduce and to exactly superimpose on itself and cannot see how this could be done in the part shown on the X-ray, which is from the axilla through to the SVC and into the atrium. If the wire was used to insert the catheter it would be unlikely to loop. If the wire was not used it was possible to loop at some place in the arm.

Dr Clarke suggested that on the basis the whole 50cm was introduced and accepting a perforation occurred in the right atrium then he postulated that the guide wire was reversed and the stiffer end perforated fully or partially the right atrium at initial insertion. He stated that the distance between the junction of the SVC and right atrium and the area of perforation found at autopsy in the right ventricle was 8 to 10 cm. He opined that the catheter may have gone through the right ventricle. The tip with TPN fluid was being infused through the top hole of the tip and inside the pericardium and the saline solution was being infused through the other lumen and through the other hole in the catheter and was still in the ventricle or the heart wall, thus explaining why mainly TPN fluid was found in the pericardium and why 300mls of fluid was found and not 5 litres.

As to the possibility of a catheter migration which then perforated the ventricle wall, Dr Clarke said it is not impossible but the heart wall would have had to be pretty weak. He discounted the possibility of the catheter without the wire insertion being capable of perforation as the tip was quite soft.

Although Dr Clarke was unsure as to the location of the tip, with the overall circumstances of the entry point of the catheter, the length inserted, evidence of a perforation of the right ventricle and with a pericardium full of TPN there were very few other explanations. He said that some of the TPN fluid could have been absorbed through the lymphatic system but he was not an expert in that area.

During cross examination Dr Clarke said that he can see the PICC line distally but agreed that it is a matter of interpretation. On the basis that the wire was still inserted at the time of perforation the PICC line would have

perforated and stayed in the wall for the whole time from insertion to death. He agrees that if pushed through 3 days earlier one should have seen detectable abnormalities much earlier.

Dr Gregory Slater is also a radiologist who provided a report to Blake Dawson and Waldron[27] and he also gave evidence. He states that the tip of the catheter appears to lie just below the level of the carina in the region of the distal SVC and cannot be traced further distally. It does not pass into the right atrium or right ventricle. He said it was not uncommon to put the whole 50 cm of PICC line in and disagreed with the assertion that it should have only been 40 cm for a person of her height and weight. He estimates the distance to SVC/right atrium at 45 to 50 cm. On his view he says that the pathologist has to be wrong about his findings as to a perforation. He finds it hard to imagine a hidden loop.

Dr Robert Boots located the tip of the PICC line in the general area located by Dr O'Connell and Dr Slater. He said the film quality is very poor but he cannot see the line distally going into the right atrium/ventricle. He did not have a satisfactory answer for how the wall came to be perforated, but that it most probably occurred by catheter migration. On the basis the tip is where he says, then migration could only occur from looping. Looping is possible and not uncommon when inserting a line through a blind technique, that is where a PICC line is inserted without the use of a continuously viewed image intensifier and an X-ray is only taken to confirm its final placement. You would not necessarily see it with an Image Intensifier, although the likelihood of this is less given you are monitoring the PICC line's path.

Dr Boots thought it was unlikely that the heart muscle was perforated at time of insertion. There would have been symptoms from bleeding and there would have been pain.

He would discount the view that there was a tear in the ventricular wall as there would be more blood found in the pericardial sac. He was asked for his opinion regarding Dr Olumbe's view that there was a partial perforation, a necrosis of the muscle and that TPN fluid leaked through in the hours before and the wall gave way. Again he thought that there would be more blood than TPN found in the pericardium if that was the case and that *"a hole is a hole"* and what would seep through was the total contents of the chamber which would mainly be blood with a small amount of TPN.

Dr Boots agreed that there could have been an earlier perforation with a partial healing and even necrosis of the wall but if the wall had just given way then it would be the scenario referred to above. The TPN found in the pericardium is more in keeping with a slow ooze from the catheter with it being in the pericardium for some time. When it perforated into the pericardium is a difficult question to decide.

[27] Report is Exhibit D8

He discounts Dr Clarke's view that there was an inadvertent perforation of the wall at insertion when wires were put in backwards and the tip perforated the pericardium. This is because it would have had to be inside the pericardium for 57 hours and he would have expected more symptoms earlier. There was no progressive rise in blood pressure and the heart would react to a perforation rapidly. There would have been pain, increasing tachycardia, increasing pulse and decreasing blood pressure. That was not found in the nursing observations.

Dr Boots' view was that there had been a redundancy in the PICC line probably around the subclavian area and that the tip appeared to be positioned at the junction of the right atrium and SVC. He thinks the PICC line migrated and forced itself through in the hours before death and the catheter must have been in the pericardium.

Dr O'Connell identifies the tip as being at the junction of the SVC and right atrium. He can see the faint linear density referred to by Dr Clark but sees it as a radiolucent line going to a vessel. He disagrees with the view that with a person of Mrs Fitter's height and size that inserting 50cm of catheter would mean there must be some redundancy as there are a number of variables and if placed under image intensifier it is the safest method possible.

Dr O'Connell is at a loss as to how it occurred. He does not agree with Dr Boots' scenario of line migration. He has heard of it happening in other cases but does not accept it occurred in this case. He disagrees with the possibility of looping because the size of the vein is 6 to 8 mm and it would be impossible to loop it with the wire. He would consider it very unlikely that he could put the guide wire in backwards. He loads it and always checks.

In relation to a partial perforation Dr O'Connell said the autopsy report shows no evidence of a haemorrhagic track which would have been expected if the wire or catheter had gone through. He was informed of the evidence given by the pathologist that the pathologist had found there was an earlier perforation, necrosis of the heart muscle by trauma and not infarction, and seepage of TPN fluid. He said that the heart wall then gives way. The pathologist also had found evidence of a 4mm tear and tracks. Dr O'Connell could not comment on these matters.

One issue that arises is the length of catheter introduced. In this case it was the full 50cm. Dr O'Connell does not agree with the view of Dr Clarke that 50cm was too much catheter and that warning bells should ring if that occurred. Dr Slater is of the same view as Dr O'Connell.

Autopsy evidence

Dr Alex Olumbe performed a post mortem examination on 26 September 2005. [28] I do not intend to set out in detail his findings other than as is necessary. He has been a forensic pathologist for 14 years and has worked

[28] His reports are exhibits A2 & A3

at the John Tonge Centre for 5 years. He would have performed 18,000 autopsies.

He found that the small and large bowels were markedly distended with gas and this would be consistent with the clinical findings that there was an ileus or small bowel obstruction. The small bowel and large bowel were connected, there was no leakage and the surgical sutures were intact. There was no twisting or obstruction of the bowel. He concurred with the opinion of Dr Stevenson that there was no abnormality in the bowel that would have caused death.

There was evidence of severe coronary atherosclerosis which of itself can cause a sudden and unexpected death but there was no evidence that there had been a myocardial infarction with a subsequent rupture of the heart.

There was evidence of mild to moderate emphysema in the lungs but he was of the view it would need to be more widespread or severe to cause death. There was a liver tumour which was benign and not related to death.

The significant finding was the collection of 300ml of milky bloodstained fluid in the pericardium. He was of the opinion that this amount would cause death from a cardiac tamponade.

Samples of this fluid were compared by Linda Jones, Scientific Officer, with a bag of TPN fluid obtained from the Holy Trinity Hospital Pharmacist. That analysis showed the two samples had a very similar fatty acid composition.[29] She stated in her evidence that there was a very, very high likelihood they were the same type of fluid. She excluded any suggestion that it was a chylous fluid as suggested by Dr Clarke.

The fluid from the pericardium was stained with blood and she opined that it consisted of 50% TPN, 25% blood and 25% of other fluid. It is noted that the double lumen PICC line was placing both TPN fluid and saline through the two lumens of the PICC line. Dr Olumbe opined that the blood staining of the fluid was indicative of leakage of blood at the time of perforation.

I am satisfied beyond any possible doubt that the fluid found in the pericardium at autopsy included the TPN fluid that was being given to Mrs Fitter through the PICC line.

It is Dr Olumbe's opinion that the tip of the line perforated the wall of the right ventricle in the area shown and marked on the diagram of the heart[30] and that as a result of the perforation, the TPN fluid from the line seeped or leaked into the pericardium.

Dr Olumbe found there was no evidence of trauma to the right atrial wall. On naked eye examination there was no obvious perforation in the right ventricle

[29] Exhibit A4
[30] Exhibit C18

but he found that microscopic appearances were consistent with myocardial necrosis due to trauma from the tip of the PICC line. The outer surface of the right side of the heart showed a milky white area with 2 discontinuous areas of bleeding.

Dr Olumbe found evidence of a blood clot and a perforation in the lower right ventricle which he considers to be the place of the perforation. This was the same area of localised necrosis and he was able to define a track under microscope. He also found droplets of fatty lipids localised within the tract which would be coming from the fluid because you do not expect those fat droplets in between the muscle fibres. He considered that the breach into the heart wall was not an infarction because it was localised and not widespread.

He considered that there was not a complete perforation as death would be very quick and there would have been substantial blood found in the pericardium. Rather he considered that it must be due to a partial perforation.

He agreed that it was possible that the tip of the PICC line could have rested in the heart muscle and the hypertonic nature of the TPN fluid eroded further into the wall. He was unable to be dogmatic about which of those possibilities was most likely.

Dr Olumbe was of the opinion that it was possible for there to have been an incomplete perforation earlier and even possibly at the time of insertion and that it slowly seeped through in the days after that. It was also possible for the tip of the PICC line to be sitting through the hole and TPN fluid flowing directly into it. However, if there was a complete flow without sealage of the tip by the pericardium then there would have been almost only TPN fluid without much blood.

Another possibility was that the tip was sitting almost on the surface of the heart and then impinging on the pericardium and you would have TPN fluid flowing slowly into the pericardial sac and then with the tear there would then be blood flowing in. If the tip had perforated in the hours before death then he would not have expected to find the blood clot.

Summary

One of the difficulties that the inquest faced was due to the unfortunate removal of the PICC line by mortuary attendants before the commencement of autopsy. Locating its final resting position may have resolved some of the mysteries of this case.

I am certainly satisfied that death is due to cardiac tamponade from the TPN fluid entering the pericardium. The mechanics of how this occurred are problematical and the evidence is in such dispute that it is not possible to be definitive on this.

I am satisfied on balance that there was not a total perforation at the time of insertion as death would have been likely within a few hours and significant would have been observed.

I am also satisfied that as TPN fluid was found in the pericardium, it could only have been by some mechanism relating to the PICC line. That would not have occurred unless the PICC line somehow found its way into the pericardium, and for that to have occurred there must have been some migration of the line post insertion. If the tip of the PICC line remained in situ at the junction of the SVC and the right atrium this would simply have not been possible.

A partial perforation explains some outstanding issues, but as to whether there was a subsequent tear and TPN leaking in, it is difficult to say. I agree with Dr Boots on this issue that logically there would be more blood found in the pericardium than there was. One possibility is a partial perforation at some time after insertion which explains the localised necrosis and a blood clotted perforation. With partial perforation the TPN fluid dissolves away the heart wall and then makes its way through in the hours before death and hence more TPN fluid building up over a few hours at 60ml per hour.

Partial or total perforation at insertion is discounted if you accept that the Image Intensifier image shows the tip at the SVC/right atrium. Dr Boots, Dr O'Connell and Dr Slater generally agree about that. Dr Clarke also came to the same view after some consideration and then came to a different view. His opinion, which I am sure he held honestly, did change on three occasions over a short period and perhaps is indicative of the difficulties faced in this case.

Another possible explanation is that there was a migration of the PICC line after insertion and a perforation by the tip resting on the heart muscle and then making its way through the heart wall. That is known in the medical literature[31] and is supported by Dr Boots. Looping of the catheter was suggested but it seems an unlikely event although it is technically possible. Migration of the catheter due to arm movement is suggested in the literature.[32]

There is some support for an earlier perforation as evidenced by the microscopic evidence, the track with fatty lipids and the blood clot. Maybe there were two events, that is, an earlier partial perforation as evidenced by the blood clot, and then a later perforation by the migrating PICC line which caused the fatal event evidence of which was not found at autopsy.

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[31] See *Fatal Cardiac tamponade as a result of a peripherally inserted central venous catheter: a case report and review of the literature*, British Journal of Anaesthesia, Advance Access, published 4 July 2007. In that case the X-ray taken at insertion showed the PICC tip at the junction of the SVC and right atrium. Post mortem examination showed no sign of perforation but an examination of the fluid found in the pericardium was the same as the fluid being introduced by the PICC. At inquest, a verdict of accidental death was recorded. Movement or migration of the PICC line due to arm movement was surmised to be a possible risk.

[32] ibid

Unfortunately the state of the evidence is such that this is all very much speculation.

All witnesses agree, as does the medical literature, that cardiac tamponade is a known but extremely rare complication of insertion of PICC lines. Symptoms are non-specific in nature and makes diagnosis difficult as it can also indicate other conditions. In some cases there is no examinable clear point of perforation and there have been examples where leaching is surmised to be the cause.[33]

Although no doubt unsatisfactory for the family, with the differing opinions and wide variations as to possible scenarios, it is not possible for me to make a positive finding as to the mechanics of how the TPN fluid made its way into the pericardium other than that there must have been a perforation at some stage.

Findings required by s45

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 Mrs Fitter'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.

Identity of the deceased	The deceased person was Maureen Fitter
Place of death	She died at The Holy Spirit Northside Hospital, Brisbane, Queensland
Date of death	She died on 25 September 2005
Cause of death	1(a) Cardiac Tamponade, due to or as a consequence of 1(b) Ruptured heart due to or as a consequence of 1(c) Total Parenteral Nutrition due to or as a consequence of 1(d) Caecal Polyp (surgically treated)

Concerns, comments and recommendations

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.

[33] See Coroners Findings into the death of Jacinta Kate Robinson, handed down by Deputy State Coroner Clements, 4 May 2005 and the article referred to in footnote 31 above.

I am unable to make any recommendations as to how to avoid such deaths in the future. The risks associated are rare but known in the medical literature. I am also unable to make any findings which are adverse to any medical practitioner which would warrant any referral to a disciplinary body.

On that basis I do not intend to otherwise make any other formal comments or recommendations.

I again offer my condolences to the family.

I close this inquest.

John Lock
Coroner, Brisbane
26 August 2007