CORONERS ACT, 2003

SOUTH  *AUSTRALIA*

FINDING OF INQUEST

An Inquest taken on behalf of our Sovereign Lady the Queen at Adelaide in the State of South Australia, on the 9th and 10th days of November 2005 and the 25th day of January 2006, by the Coroner's Court of the said State, constituted of Mark Frederick Johns, State Coroner, into the death of Glenn Howard Raphael.

The said Court finds that Glenn Howard Raphael aged 74 years, late of 15 Railway Terrace Wingfield died at Queen Elizabeth Hospital, South Australia on the 21st day of June 2001 as a result of cardiac and renal failure complicating treatment of infected ischaemic toes resulting from diabetes mellitus. The said Court finds that the circumstances of his death were as follows:

1. Introduction and reason for Inquest

1.1. Glenn Howard Raphael died on 21 June 2001 at the age of 74 years. He was a patient at the Queen Elizabeth Hospital to which he had been admitted on 4 June 2001. He presented with an infected left big toe. He had first noticed a blister on the toe 4 weeks previously. He had been seen by his local doctor who had placed him on antibiotics for the previous week but the toe infection had worsened to the point of being inflamed and cellulitic with several blisters and early signs of gangrene. The admitting doctor noted that he had a history of Non Insulin Dependent Diabetes Mellitus. He also had peripheral vascular disease, hypertension, cancer of the prostate, high cholesterol and asthma. However, he was alert, oriented and not distressed.

1.2. Accordingly he was referred to the Vascular Department for treatment. Doctor Roach gave evidence (T13) that diabetes affects the large arteries and contributes to

peripheral vascular disease. It narrows the small arteries in the toes and feet and impairs the immune system. The resultant lack of blood supply to the peripheral areas of the body means that when there is an infection of the toes for example, the ability of the body to heal itself is significantly reduced. It was for this reason that the primary department chosen for Mr Raphael's treatment was the vascular department, although, as we shall see, there was a multidisciplinary approach to Mr Raphael's treatment.

1.3. The subsequent course of Mr Raphael's condition was well summarised by Dr Gilbert, forensic pathologist, who carried out an autopsy after Mr Raphael's death on 21 June 2001. Dr Gilbert stated that death was due to cardiac and renal failure complicating treatment of ulcerated infected and ischaemic toes due to diabetes mellitus. Dr Gilbert states that initially Mr Raphael's:

> 'renal function was stable but by 20 June 2001 he was in acute renal failure. This was attributed to the side effects of treatment with three classes of drugs known to potentially impair renal function. These were Ramipril (an ACE inhibitor class antihypertensive), celecoxib (Celebrex, a non-steroidal anti inflammatory) and gentamicin (a potentially nephrotoxic antibiotic). Of concern was the fact that renal function, as assessed by plasma creatinine measurement, was not tested between 12/6/01 and 18/6/01 nor were serum gentamicin levels checked after 9/6/01. The creatinine level on 9/6/01 and 12/6/01 was 0.100 mmol/L (normal 0.05 to 0.12 mmol/L) but had increased to 0.249 on 18/6/2001 and 0.265mmol/L on 19/6/2001 indicating renal failure. By the next day (20/6/2001) it had increased to 0.319 mmol/L . The potassium level had also increased from 4.8 on 20/6/2001 to 5.7 on 21/6/2001 necessitating treatment with Resonium. He was admitted to the Intensive Care Unit on 21/6/2001 but deteriorated with worsening renal and cardiac failure, became bradycardic and arrested. Prolonged resuscitation was unsuccessful. The deceased's cardiac failure was contributed to by significant coronary atherosclerosis involving the left anterior descending and right coronary arteries and would have been exacerbated by acute renal failure. The failure to monitor renal function by creatinine measurement between 12/6/2001 and 18/6/2001 and gentamicin levels after 9/6/2001 despite use of three drugs known to impair renal function could be criticised. Earlier recognition of the development of renal failure may have prevented the death. Review of this case by a renal physician is suggested.'

1.4. A report was obtained from Dr A Clarkson, Consultant Renal Physician on 6 May 2002. It was tendered at the hearing and admitted as exhibit C13. A further short report was provided by Dr Clarkson on 13 September 2005 which was admitted as exhibit C13a. In the earlier report Dr Clarkson stated that on admission Gentamicin and Aminoglycoside antibiotic was administered intravenously in a dose of 240 mg daily for 14 days with no monitoring of blood concentrations of the drug (or no

recorded monitoring) after the second day of administration. He stated that Gentamicin is a nephrotoxic drug and its dose should have been reduced or the drug ceased and blood concentrations measured when decline in renal function was documented.

1.5. Significantly, Dr Clarkson noted that for the most part of his stay in hospital, the hospital records document that Mr Raphael's toe slowly improved:

> 'but a large number of other investigations were performed which were intended to delineate potential cardiac, visual, pulmonary or renal complications. Interventions occurred with treatment of cardiac problems with Perhexeline and Carvedilol, renal/hypertensive problems with Ramipril while he was continued on Gentamicin, Metronidazole, Cephalothin, Celecoxib, Diamicron and Metformin. The Gentamicin Celecoxib and ACE inhibitor Ramipril are potentially nephrotoxic. Mr Raphael's deterioration commenced on 15th June 2001 approximately 24 hours after commencing Ramipril. Subsequently there was progressive deterioration in his condition until he died on 21st June 2001.'

1.6. Dr Clarkson stated:

> 'Review of the case notes suggest strongly that the administration of Ramipril was the straw that broke the camel's back as the clinical deterioration can be dated from that time. In a diabetic patient with severe vascular disease already being treated with Gentamicin and Celecoxib this prescription without careful clinical and biochemical monitoring was dangerous and should have been avoided. The dangers of potentially nephrotoxic drugs in patients such as Mr Raphael cannot be over emphasised, as with the increasing numbers of elderly diabetics in the community, such prescription mistakes are more and more likely.'

1.7. It was against this background that an inquest into the circumstances surrounding the death of Mr Raphael was held.

2. Representation and parties granted leave to appear

2.1. When the Inquest was called on Ms Cliff sought leave to appear for the Queen Elizabeth Hospital and Drs Roach, Gibb and Fitridge. In the event I granted leave to those Doctors to be represented, and also the North Western Adelaide Health Service, which I was informed by Ms Cliff to be the incorporated body that is responsible for the provision of health services at the Queen Elizabeth Hospital. I was not prepared to grant leave to the Hospital under its own title, as I would be reluctant to make a grant of leave to what might be regarded as an indefinite group of people who might loosely be employed by or associated with the Queen Elizabeth Hospital. I was

prepared to grant leave to the body corporate itself. I was assisted at the Inquest by Ms Shaw.

3. **The Witnesses**

3.1. Evidence was given by Doctors Roach and Gibb, both of whom treated Mr Raphael during his stay in hospital. Evidence was also given by Doctor Fitridge, a consultant on the vascular ward, as to the operation of the ward. And Dr Clarkson also gave evidence in addition to his written reports referred to above.

3.2. Dr Roach is a vascular surgeon. In June 2001 she was employed in the Queen Elizabeth Hospital as a first year vascular registrar. Her duties included attending a daily ward round first thing in the morning , although not every day. The routine was that one of the registrars on the ward would attend the ward round with the interns. On two or three days per week they would be joined by a consultant or consultants. There were two registrars on the ward at that time.

3.3. Dr Roach first saw Mr Raphael soon after his admission on 4 June 2001 and took much of the history referred to above. She prescribed the IV antibiotics of Cephalothin, Gentamicin and Flagyl. She stated that these are standard antibiotics for diabetic infections (T15). She said that the Gentamicin dose is dependent upon the level of renal function which is assessed by reference to the patient's creatinine level. A creatinine level was taken on that day and shown at 0.108 which was within the normal range. A dosage of Gentamicin was worked out on the background of that test. At T17 she stated that the creatinine level can change over time and that it needs to be tested regularly, every three to four days, and the Gentamicin dose varied accordingly. At T38 under cross examination by Ms Shaw she conceded that, in retrospect, having regard to Mr Raphael's vascular disease and diabetes and the two nephrotoxic drugs (Gentamicin and Celocoxib) that he was on initially, the creatinine tests should have been done every two days.

3.4. The Hospital Medical records were tendered and admitted in evidence as exhibit C9. They contain biochemistry test results for Mr Raphael's creatinine levels for blood taken on the following days: 4, 6, 8, 9, 18, 19, 20 and 21 June 2001. The readings for those days were, in chronological order, 0.108, 0.134, 0.115, 0.100, 0.249, 0.265, 0.319 and 0.360 mmol/L. A normal reading would have been in the range 0.050 – 0.120 mmol/L. Two observations may be made immediately. The first is that

between 9 June and 18 June 2001, creatinine levels were not tested. The second is that, apart from one reading on 6 June 2001 when the level exceeded normal limits by a modest amount, the readings were normal before the period when no testing was done after 9 June 2001 until 18 June 2001. From that later day until Mr Raphael's death, readings were done daily. They were all well in excess of normal limits.

3.5. Dr Roach said that Mr Raphael's presenting condition was one that was commonly seen by the staff of the vascular department, including the interns who should be able to arrange for the regular monitoring of the creatinine and the appropriate modification of the drug dosage. She gave evidence (T25) that the creatinine levels should have been checked between 9 June 2001 and 18 June 2001. She could not explain why they were not. She stated that there had been several references in the notes to the effect that she and Dr Gibb had directed that the creatinine level and Gentamicin level should be checked. She pointed out that on average each intern would have 20 patients and if each patient had two or three blood tests a day, that would be around 60 blood tests per day to review. She implied that this may have been the reason that the tests were not done. She stated that the responsibility for the testing of those levels rested with the vascular department.

3.6. Dr Roach gave evidence of changes to the processes in the vascular department intended to ensure that levels are tested as and when required. She stated that there are now a lot more staff in the vascular unit, there is a permanent resident who is an older doctor who is very experienced and his job is primarily to assist the interns, and this has proved very successful. She stated that there is also a ward Pharmacist who reviews the patients two or three times a week. She stated that the unit does a lot more consultant ward rounds – that either she or Dr Fitridge would do a consultant ward round almost every day to ensure the smooth running of the ward. She stated that she would expect that with these changes there is less likelihood that tests such as those overlooked in the case of Mr Raphael would be overlooked today.

3.7. Dr Roach said (T41) that the gentamicin was ceased on 17 June 2001 because by that stage the left infected toe had improved. The intravenous antibiotics were ceased, and he was changed over to oral antibiotics. The nephrotoxic effect of the Gentamicin would therefore have started to abate from that time.

3.8. Dr Roach gave evidence (T43) that when attending Mr Raphael, she would not necessarily look back over the notes (Exhibit C9) to see whether previous instructions to junior staff had been carried out. She acknowledged by way of example that Dr Gibb had made an entry on 13 June 2001, when prescribing Ramipril, that Mr Raphael "*will need regular monitoring of electrolytes as will get increased creatinine secondary to ACE inhibitor*". She stated that she would expect the intern to conduct that monitoring. However, she conceded at T35 that she now tends to check the results even with the level of staff that the unit now enjoys. At T40, in answer to a question from Ms Shaw whether she could offer any explanation why the tests were not done between 9th June and 18th June, she said "*I can only presume that it was due to overwhelming other requirements for the intern and it was not done"*. The import of this sentiment is that she is not accepting personal responsibility for this failure. She gave evidence that she had made notes that the tests be done, that they were not done, and that the only explanation she could offer is that the intern was overwhelmed. This evidence needs to be carefully examined in view of the evidence given by Dr Fitridge and Dr Clarkson on the matter of responsibility for checking and following up monitoring in this situation. I return to this issue later.

3.9. Dr Gibb is a Consultant Physician. In June 2001 she had the same qualification and was then (as she is now) Director of Physician training at the Queen Elizabeth Hospital, the physician in charge of the high risk pre-operative assessment clinic, and a general physician in the cardiac unit. She no longer has the final of those roles, but her peri – operative role with surgical patients has increased to the extent that she now been recruited to work on the orthopaedic unit doing that work.

3.10. She said that she would have been consulted by the vascular unit to look at Mr Raphael as he was being considered for surgery and her role was to "*look at a short term management plan to optimise his medical condition as much as possible before any surgery*" might take place (T47). When she first saw Mr Raphael (T47) she noted his diabetes and peripheral vascular disease. She also noted a history of supraventricular tachycardia and left bundle branch block on his ECG. The left bundle branch block is an abnormality found on ECG testing that shows a disruption of the normal pathways for electrical conduction though the left ventricle. It is a condition associated with an increased risk of cardiac abnormalities and placed Mr Raphael at high risk of ischaemic heart disease. Dr Gibb particularly noted that

Mr Raphael suffered shortness of breath with very limited physical exertion such as walking to the toilet. Furthermore he had a history of poor self management of his diabetes, in that he did not follow a diabetic diet and had not had his eyes checked for over a year.

3.11. Dr Gibb was particularly concerned about Mr Raphael's shortness of breath after mild exertion, because if he were to have surgery one of the key factors in survival or cardiac complication with surgery is exercise tolerance. The fact that he could only walk five to ten metres suggested that he was very frail and had significant co-morbidities apart from his ischaemic toe.

3.12. Dr Gibb also had an eye to Mr Raphael's long term well being, as well as the short term plan for managing any surgery that might become necessary. She took a number of steps to this end, including an increase in his diabetes control medication, Metformin. She arranged a diabetic educator review because he had a poor understanding of how to control diabetes and how important it was in long term health. She arranged an HBA1C, a 24 hour urine protein and an ophthalmology review. The HBA1C would give more information about the progress of the diabetes. Dr Gibb arranged an echocardiogram and pulmonary function tests and suggested that if he was likely to have surgery she would do a nuclear cardiac scan. She noted that he was a candidate for preoperative Beta blocker as these are almost the only medications that have been shown to decrease the risk of cardiac death or infarction after surgery.

3.13. Dr Gibb summarised the position as follows:

> 'The immediate concern was that this was a gentleman who was likely to tolerate surgery poorly and the question was if there was anything that I could do that could improve his cardiac risk with surgery. Often the things that I may do before surgery actually improves (sic) people's general medical condition which can then carry on into further community management.' (T9)

3.14. The surgery that was in contemplation was never clearly defined. Dr Gibb speculated about the possible surgical interventions that might have been contemplated at T60. The clinical record for Mr Raphael contains several references to the possibility of amputation below the left knee at an early stage of his stay. Presumably this possibility receded as the intravenous antibiotics began to stabilise the infection. It is not necessary to resolve this matter for the purposes of these findings.

3.15. The Echocardiogram Report ordered by Dr Gibb was tendered and admitted as Exhibit C10b. Dr Gibb gave evidence about the report at T62 to 64. She noted that Mr Raphael's heart was significantly enlarged. This meant that the heart was struggling to cope and this had been a chronic process. The ECG reported three measures of his left ventricular ejection fraction, which is the percentage of blood that comes into the left ventricle that is pumped with each heart beat. A normal left ventricular ejection fraction is over 55%. The three measures were 36%, 26% and 15%, the last being the Doppler measurement which in Dr Gibb's view was a more accurate measure for this patient of his heart function. She commented:

> 'So you can see that this was severe heart failure and really would, I would not be expecting this gentleman to walk more than five to ten metres without being limited by heart failure with that kind of ejection fraction. It really is severe.'

3.16. Dr Gibb further analysed the degree of heart failure suffered by Mr Raphael, using the notions of plumbing, pumping and wiring as metaphors for the heart's various functions. She noted that he had a problem with his pumping as evidenced by the poor ejection fraction. He had a problem with the wiring of the heart, which in the metaphor is the electrical activity that makes the heart beat regularly, in that he was in atrial fibrillation which means that the heart rhythm is disturbed with the result that the heart beats irregularly. He also had a problem with the plumbing of the heart which in the metaphor is the blood supply to the heart muscle which if cut off causes heart attacks.

3.17. Doctor Gibb summarised the position by saying that he had New York Heart Association class three heart failure. A copy of the New York Heart Association classification was tendered and admitted as exhibit C10c. Class III is noted as "*marked limitation on ordinary physical exercise with no symptoms at rest*".

3.18. On 13 June 2001 Dr Gibb formulated a plan of management for Mr Raphael's diabetes, his lungs and his heart. For his diabetes she was waiting for his test results still. For his lungs she suggested broncho dilators. As to his heart, she was concerned about his use of the drug Verapamil, which he had been on for some time prior to his admission. She noted that Verapamil is contraindicated in cases of left ventricular failure, so she decided to stop that. She prescribed Perhexiline. She also prescribed an ACE inhibitor, namely Ramipril. She directed that he would need regular

monitoring of his electrolytes because his creatinine may increase because of the Ramipril ACE inhibitor.

3.19. As to the Ramipril, Doctor Gibb said that a large body of evidence has developed over the last 20 years that in treatment of heart failure ACE inhibitors improve morbidity. There was also evidence that Ramipril or other ACE inhibitors reduce the progression of diabetic renal patients to end stage renal failure. I interpolate the remark that there is a paradox here because there is also evidence that, at least in the short term, Ramipril is nephrotoxic. At T68 Dr Gibb explained this. She noted that ACE inhibitors actually decrease the filtration pressure across the glomerulus, thus decreasing the glomerulus filtration rate. This increases the creatinine levels. However, this effect is reversible, but is a well recognised effect of ACE inhibitors in the short term. In the long term however their decreasing kidney function and rise in creatinine will be much less than if they were not on the ACE inhibitor. As will appear, this was the subject of discussion in the evidence of Dr Clarkson.

3.20. Dr Gibb stated that by regular monitoring of creatinine levels, she meant every 2 to 4 days (T69). She stated that the responsibility for the regular monitoring rested with the Vascular Unit. She said "*as a consultant physician, if I ask for something to be done, I expect it to happen*".

3.21. Dr Gibb next saw Mr Raphael on 15 June 2001. By this time the HBA1C diabetes test result was in and it showed poor long term control of the condition. Apart from that, there was little progress and Dr Gibb made no changes at that time.

3.22. The Drug Chart, or "Regular Medication Record" in Mr Raphael's Hospital records (Exhibit C9) records that the Ramipril was commenced on 13 June 2001, and continued daily until the entries for the 19th and the 20th which record that on those days it was "held", by which I infer that the drug was not administered on either of those days. The entry for the 21st states that the Ramipril was cancelled. Dr Gibb's recollection of events was, understandably, no longer clear at inquest, as some four years had passed by then. She was unable to take matters further than the medical records in relation to the timing of the decision to cease the Ramipril. The records show that there was a Vascular note on 19 June 2001 which recorded an increase of creatinine to 0.249. As we have already seen, that result was for the 18th, and on the 19th the result was even more alarming, at 0.265. Presumably the result for the 19th

was not available at the time of the Vascular note to which I am referring. In any event, the note records that the vascular team was then proposing to discuss with Dr Gibb the possibility of holding the Ramipril, presumably in light of the increased creatinine.

3.23. A further vascular entry in the record on 20 June 2001 at some time earlier than 1040 hours on that day records what must have been the contemplated discussion with Dr Gibb, and contains the notation "Stop Ramipril". Some 7 hours later that day at 1716 hours, there is an entry in the hand of Dr Roach stating "Pt in acute renal failure at present". The note also stated:

> 'in view of increased oliguria and increased creatinine ? will need renal support. D/W Renal Reg – she will review later today'.

The next entry was at 1830 hours the same day, when Mr Raphael was reviewed by the Renal Unit.

3.24. At T79 counsel for Dr Gibb asked her to comment on Dr Clarkson's view that it was a mistake to prescribe Ramipril. Her response was that it was certainly not a mistake, but a deliberate therapeutic strategy. She said that Mr Raphael came into hospital with ischaemic toes, but his major functional limitation was related to his cardiac disease, and that ACE inhibition is one of the best strategies for improving morbidity and mortality in heart failure cases. She stated that she was aware of the potential risks involved in prescribing Ramipril, but that she had done it in the expectation that the monitoring she had ordered would happen. She acknowledged that "*something in the system didn't work*", and that "*the back up .....obviously failed and the monitoring did not occur*". However, she maintained that Mr Raphael was in danger of rapid cardiac death even if he had not had the renal failure, and maintained that the ACE inhibitor was appropriate in light of that.

3.25. At T88 Dr Gibb gave evidence that Ramipril has a beneficial effect on mortality in heart patients within 30 days. She also stated that the effect on the kidney can be reversed and the half life of Ramipril is about 24 hours, so it would take two and a half days for the Ramipril to be cleared from the system normally so the effects should have been completely reversed within about five to seven days.

3.26. At T93 Ms Shaw asked Dr Gibb whether she thought that the result would have been different had the monitoring been undertaken between the 10th and the 17th as

directed. Her response was say that on the assumption that the acute renal failure developed purely because of the medication, earlier monitoring may have prevented his death. She pointed out that the renal failure may also have developed from the septicaemia from his toes. She pointed out that Mr Raphael was not a well gentleman anyway, having regard to his cardiac disease, and stated that, even had he survived this episode, "*he was going to die in short order anyway*".

3.27. Before leaving the evidence of Dr Gibb, I note that, in answer to a question from me she made the following remark about Dr Clarkson's evidence:

> 'I think with Dr Clarkson's evidence, he is coming in at the end of the day having been told that this gentleman died of acute renal failure. He is a kidney specialist. I have a different viewpoint in that I actually saw this patient and I actually say his function. My concerns as a general physician were probably more global than just purely one end organ. So I think that might be why there may be different perspectives in this gentleman.'

3.28. Dr Fitridge gave evidence at the inquest. He is a Vascular Surgeon, and in 2001 was a Consultant Vascular surgeon and head of Vascular Surgery at the Queen Elizabeth Hospital. He stated that he did not have any direct involvement with the care of Mr Raphael during his time in the vascular ward. Although Mr Raphael was admitted under Dr Fitridge because he was head of the Unit, he was actually managed by Mr Subramanian, one of the other consultants.

3.29. Although Dr Fitridge did not treat Mr Raphael, I thought it useful to hear from him in his capacity as the head of the unit concerned. He gave evidence very frankly, and readily conceded that there were shortcomings in the treatment of Mr Raphael. He was able to tell the court about the changes that have been implemented since, and was also able to give an impartial and expert view about the roles and responsibilities of medical staff on the vascular ward now, and in 2001.

3.30. Dr Fitridge gave evidence at T101 that the presentation of a patient with diabetes and an infection to a peripheral part of the body is becoming a common event in the Vascular ward. He said that type 2 or maturity onset diabetes is increasing in the community, and that has a number of effects. It affects the sufferer's arteries with blockages, it affects kidney function and it affects the nervous system with the result that the patient does not feel his or her feet terribly well. So for example, if a person buys a new pair of shoes that are ill fitting and rub the skin off, the patient may not

notice until the point where there is blistering and ulceration. As a result, the "diabetic foot" is an increasing problem and is responsible, on Dr Fitridge's estimation, for between 50 and 70 percent of amputations. He said that a multidisciplinary approach is required to treat patients in Mr Raphael's condition because their management is quite complex and difficult.

3.31. He said that one of the effects of diabetes is that patients do not respond to infection very well. In the case of feet, the infection tends to be caused by a broad spectrum of organisms. This means that the antibiotic treatment needs to cover the spectrum of possible organisms to combat the infection effectively, and the three antibiotics prescribed for Mr Raphael on admission were the normal standard used on the ward. He gave evidence that the Unit has developed formal protocols for admission antibiotics which incorporate the use of those prescribed for Mr Raphael.

3.32. At T104 Dr Fitridge was asked whether there would have been any alternative to the prescription of broad spectrum antibiotics including the nephrotoxic Gentamicin. His evidence was that, as so often is the case, it is a balancing exercise. The surgeon is forced to look at the range of alternatives. Dr Fitridge said that the treatment options are to attempt to heal the infection with antibiotics as a first resort. The next resorts are surgical, and include angioplasty (a balloon in the artery) of an artery near the foot in an effort to get greater blood circulation in the foot. Another option is a vascular by-pass, or in the last resort a major amputation which is usually below the knee or even above the knee. This is very much a last resort because, as Dr Fitridge pointed out, in a fairly frail patient such as Mr Raphael, the impact of the amputation usually is that the patient goes to a nursing home which is associated with a poorer quality of life than was being experienced prior to admission.

3.33. Dr Fitridge was questioned by his counsel on the matter of the responsibility for monitoring creatinine levels. He said that with a nephrotoxic drug such as gentamicin, they should have been monitored every two to three days at least (T105) and that the intern is responsible for ordering the test and "chasing" the results and then "reports back to the registrar" (T105). He went on to say:

> 'Obviously at the end of the day you expect the registrar to keep an eye of (sic) these sorts of results, because it's fairly important for the management of the patient.'

3.34. Dr Fitridge noted that the creatinine level was normal on the 9th June but that by the 17th it was getting towards .25 (in fact a result of .249 was obtained from blood taken on the 18th, no test having been done on the 17th but I do not cavil with the thrust of his evidence) "so between that period of time when gentamicin levels weren't done that was around the time that the kidney function deteriorated".

3.35. At T113 Dr Fitridge stated in relation to the monitoring of gentamicin:

> 'I'd say that's inadequate basically as we've sort of discussed before I'd be doing it every three days.'

3.36. At T113 Dr Fitridge stated in relation to the monitoring of creatinine:

> 'I'm afraid that was inadequate as well really…'

3.37. At T113 it was put to Dr Fitridge by his own counsel that those levels should have been done more frequently and that should have been something set in train by the intern and perhaps supervised by the registrar. His response to that was: "*Correct*".

3.38. At T115 Dr Fitridge was taken by his counsel to the topic of the introduction of the Ramipril – also a nephrotocic drug - on the 13th June. He was asked who bore the responsibility for the continued monitoring of creatinine and gentamicin levels at that point bearing in mind that the Ramipril had been introduced by Dr Gibb. His answer was that the intern (on the Vascular Ward) would follow the instructions of the consultant (Dr Gibb) but that would be supervised by the registrar or registrars on the Vascular Ward.

3.39. It will be recalled that Dr Roach was not prepared to accept responsibility for the monitoring of creatinine levels in her evidence (T40) as set out above, even though she was one of the Registrars on the Vascular Ward at the time. This position cannot be reconciled with the views of her senior colleague Dr Fitridge, who clearly attributed supervisory responsibility to the Registrars, which includes Dr Roach.

3.40. At T128 Dr Fitridge gave evidence that one of the Registrars would see the patient every day – normally both of them would. In fact, the Hospital record (Exhibit C9) shows that Dr Roach saw Mr Raphael on each of the 4th, 8th, 10th, 11th 12th . The next entry at which it can be positively asserted that Dr Roach saw Mr Raphael was on the 20th when she wrote that he was in acute renal failure. There were Vascular Unit ward rounds recorded on each of the intervening days, but the record does not tell us

whether or not Dr Roach was present on those occasions. I infer that it is likely that she would have been present on at least one or two occasions between the 12th and the 20th on the basis of Dr Fitridge's evidence about the frequency with which the Registrars saw patients. It is possible but unlikely that Dr Roach did not see Mr Raphael at all in those seven days, so I am unable to reach any firm conclusion on this point.

3.41. Dr Fitridge gave evidence at T129 that the registrars normally review the results that are considered important. And he said:

> 'I mean, the registrars essentially run the unit on a day-to-day day basis and are expected to know all the appropriate results and give instruction in terms of tests to be ordered.'

He was asked whether he would expect the registrars to have picked up that there had not been a creatinine test since the 9th of June and said:

> 'I would have expected that to be the case, yes.'

3.42. Finally, Dr Fitridge told the court about the improvements made in the organisation of the Vascular Ward since Mr Raphael's death. He said that a number of strategies have been adopted. Firstly the unit has accepted that having a number of sick patients to look after is challenging for an intern, so it has recruited a career medical officer who is a senior resident who is not on a training scheme but was a GP for a number of years. This doctor is involved in the day-to-day management of the patients. The Unit also has a pharmacist who attends the ward two or three times a week and is actively involved in the foot clinic where the diabetics tend to be first seen. There is also discussion taking place with the IMVS to try and have some way of flagging patients who are on nephrotoxic drugs such as those involved in this case to ensure that the monitoring does occur. The unit has also developed a practice of having more ward rounds per week so that there are at least two consultant ward rounds per week rather than one.

3.43. Before leaving the evidence of Dr Fitridge, I mention an issue which I raised with him. I was (and remain) intrigued by the fact that there were no creatinine tests done between 10th June and 17th June inclusive, but that on 18th June the test was done again. I had wondered if there was some explanation of what had triggered this reintroduction of testing. Certainly there was nothing in the notes that explained the reintroduction (any more than there was any explanation in the notes or in the

evidence to explain the failure to test during that period). At T136 Dr Fitridge could not provide any answer. Indeed I noted that there was reference in the Vascular ward note for the 18th June to a plan for discharging Mr Raphael the following day after consultant review. Dr Fitridge could not explain this incongruity. He suggested that the note must have been written before the creatinine result came back on that day after the long period without any testing being done. I think that must be so. However, it demonstrates just how unexpected the impending renal failure was to those caring for Mr Raphael at that time. This was a mere three days before his death.

3.44. Dr Anthony Clarkson gave expert evidence at the inquest. He had, until just before the inquest, held the position of Director of Renal Services in the Australian Capital Territory. Prior to that he was Director of the Renal Unit at the Royal Adelaide Hospital and Senior Consultant in Nephrology at the same Hospital.

3.45. Dr Clarkson explained to the court that there are different ways in which drugs can be nephrotoxic (damaging to the kidneys). One is that the patient can have an idiosyncratic reaction to the drug. The other way is that the drug is directly poisonous. Some drugs can be directly poisonous to the kidneys because they are excreted entirely unchanged by the kidney and they are directly poisonous to the cells of the kidney.

3.46. Dr Clarkson noted that the Gentamicin was ceased on the 17th June and the measurement of Gentamicin concentration in the blood on the 21st June was toxic and inferred that the level on the 17th would have probably been much higher. He was asked what impact such a level would have had on renal function and replied that it was not good. When asked to elaborate on this laconic response, he stated that the effect would have been to impair kidney function with the result that the excretion of other drugs would also be impaired and the total body function would become abnormal. For example, the electrolytes in the body and enzyme levels would be upset by an increase in acidity (T155).

3.47. He stated that Gentamicin is a cationic drug. It must be given intravenously because it is not absorbed abdominally. Other drugs can be excreted by the liver, but Gentamicin is only excreted by the kidney. Because of its cationic character, it is peculiarly attracted to the tubular cells of the kidney which take the urine out to where

it drains into the bladder. It causes damage because it accumulates and is not excreted. As it accumulates, the amount of damage increases (T163).

3.48. He stated that Ramipril does two things – it lowers blood pressure, and it is used in patients with heart failure to allow the left ventricle to work more efficiently by reducing the after-load or the pressure which the heart has to act against (T157).

3.49. Dr Clarkson was asked about the effect of Gentamicin in conjunction with other potentially nephrotoxic drugs. He referred to Celecoxib or Celebrex and said that it can impair the blood flow into the filter of the kidney. The filtration rate will be affected by the pressure of blood going into to the kidney, but also by the pressure applied by the blood vessel taking the blood out. Normally, if the filtration rate required by the body of the kidney needs to be increased, the blood vessel taking the blood out will constrict in order to permit that to happen. This occurs by the influence of a hormone called angiotensin. He referred also to Ramipril and stated that it is also an angiotensin converting enzyme inhibitor, so that the angiotensin cannot act to constrict the blood vessel taking the blood out of the kidney. The vessel remains dilated, and there is no back pressure to increase the filtration capacity of the kidney (T155-156).

3.50. Dr Clarkson stated that the creatinine levels should have been taken immediately prior to the commencement of Ramipril in order to make a decision whether to administer the Ramipril. It should not have been administered if there was evidence of kidney failure as measured by creatinine levels (see T159 and T168).

3.51. Dr Gibb had given evidence (T69) that Mr Raphael had been on Celecoxib for a long time (prior to admission) and despite that (nephrotoxic drug) his creatinine was still normal (on admission) "so that was reassuring in itself that his glomerular filtration rate was reasonable". This hypothesis was put to Dr Clarkson who responded negatively stating that it is the interaction of the Ramipril with the Celecoxib that would cause the problem (T160). Neither was support for the hypothesis forthcoming from Dr Fitridge, who stated that he did not take comfort from the fact that Mr Raphael had been tolerating Celecoxib prior to admission (T123).

3.52. In his report dated 6 Mary 2002 Dr Clarkson had said "review of the case notes suggest strongly that the administration of Ramipril was the straw that broke the camel's back as the clinical deterioration can be dated from that time". This opinion

was the subject of strong exception by Dr Gibb. It was a position that Dr Clarkson seemed at first blush to retreat from in his oral evidence. For example, at T160 he said he had no qualms with the prescription of Ramipril for patients like Mr Raphael, but added that is was the monitoring that he would worry about. He said at T161 that he was not saying that the drug had been prescribed inappropriately, but that he was critical of the monitoring of the kidney function following its prescription, particularly in association with the other drugs Mr Raphael was on. He said at T165 that his use of the expression "*prescription mistakes*" in his report of May 2002 meant that while the prescription of Ramipril in diabetics is appropriate and has potential advantages, the presence of other drugs in the patient's system must be taken into account. He gave as an example Celecoxib, and described the combination of those types of drugs as a "*not uncommon*" cause of acute kidney failure "*because of the way they work together, not on their own but together*".

3.53. Counsel for Dr Gibb asked Dr Clarkson how he would have dealt with Mr Raphael's cardiac problems, for example his very low ejection fraction of 15%. He said that he did not think the cardiac function would have been compromised (in the absence of the Ramipril) any more than it was before the prescription of Ramipril because Mr Raphael was not in gross cardiac failure before it was prescribed. He pointed out that these was no evidence that the Ramipril actually improved Mr Raphael's cardiac function (T169), however he pointed out that it would certainly have made his renal function worse (T169-170).

3.54. Dr Clarkson elaborated on this theme at T170 when he said:

> 'when he came into hospital his cardiac function was a problem but it was not a major problem, when he came into hospital his kidney function was not a problem, but it became a major problem that would have impacted unfavourably on his heart function.'

When asked how to support Mr Raphael's heart function without Ramipril he said:

> 'It may not have needed supporting, that's what I am saying; there was no evidence that he was having difficulties with his heart before it was given.' (T171).

3.55. Counsel for Dr Gibb put to Dr Clarkson that there was evidence of heart problems and cited the findings of Dr Gibb. Dr Clarkson responded:

> 'I understand; there are a lot of people walking around quite comfortably with an ejection fraction of 15%, when they walk quicker they get into trouble.'

3.56. Counsel for Dr Gibb pointed out that one of Dr Gibb's objects was to look not only to how the patient might cope with surgery, but to the future after discharge from hospital, and to put him in a good position with respect to his heart function. Dr Clarkson responded by pointing out:

> 'Yes, the problem was he was not discharged, he was discharged out the back door.'

3.57. In fact Dr Gibb herself had stated in evidence that the Ramipril would not have an immediately beneficial effect on the cardiac function but that it can take up to 30 days to have a beneficial effect on mortality (T88). This seems to be the problem. The Ramipril was obviously indicated for Mr Raphael's general well being. However, he was not in hospital to look to his long term well being. He was in hospital because of his infected toe. I put to Dr Clarkson the proposition that it might be reasonable to assume that a less aggressive approach to the heart problem during the middle part of Mr Raphael's stay in hospital might not have been deleterious to him. He agreed (T173).

3.58. At T162 Dr Clarkson was asked to comment on whether the outcome might have been different if the monitoring had been sufficient. He stated that Mr Raphael's death might still have occurred but it would have been less likely.

3.59. Finally Dr Clarkson was asked whether it was his view that the registrars should be going back over the hospital notes and reviewing the earlier instruction to ensure that they have been followed up by the intern. He responded by saying that is part of the registrar's job.

4. Conclusions

4.1. In light of the evidence I have come to the following conclusions:

4.2. The monitoring of the test results and the responsibility for ensuring that the tests were actually undertaken was, to a significant extent, that of the registrars. The interns are trainees. They are junior doctors. Furthermore their stay on the vascular ward is brief – two months only – as part of their training. It is unrealistic to assume that they can be held solely to account for tests that were as crucial as those required for Mr Raphael in such circumstances. The common, constant presence in the ward at the relevant time was the registrar. That is where primary responsibility rests,

although the consultant under whom the patient is admitted bears ultimate responsibility.

4.3. Counsel for the doctors and the hospital was instructed by her clients – very properly – to concede that there was a failure to monitor and that it was a mistake.

4.4. The matter of whether the proper monitoring of Mr Raphael's creatinine and Gentamicin levels would have prevented his death is one to which there is no clear answer. However, as Dr Clarkson pointed out, his death would have been made less likely had they been done.

4.5. The administration of Ramipril would not, in itself, be a mistake in a diabetic patient. However, its administration in conjunction with the other nephrotoxic drugs does appear to have been erroneous. The motivation of long term improvement to Mr Raphael's heart function was laudable. However, he was admitted with an infected ischaemic foot, which required that he be given the nephrotoxic drug Gentamicin. That could not have been avoided, and was clearly the correct thing to do. The evidence showed that whatever beneficial effects Ramipril might have brought about would not have occurred in sufficient time to be decisive in the decision whether or not to operate on him in the event that his foot did not respond to the Gentamicin. In the event, the Gentamicin was withdrawn on 17 June when the foot was improving. The Ramipril was introduced on 13 June. It is difficult to see that he would have suffered any short to medium term detriment if the Ramipril had been withheld. As events showed, only 4 days later, surgery would not be required to deal with the foot. On the other hand, the Ramipril clearly did have a detrimental effect in the short term, which careful monitoring may have detected in sufficient time to prevent catastrophic harm. The point is that it was not necessary to introduce it at all.

4.6. Dr Gibb argued that Mr Raphael was headed for acute heart failure in the short term, and that Ramipril would assist in preventing that. I do not accept this argument as a justification for the introduction of a nephrotoxic drug that appears not to have been immediately necessary for the problem with which he presented. On the other hand, I fully acknowledge the precarious state of Mr Raphael's general health and particularly his heart. In addition he had diabetes, underlying renal problems and the infection from the foot.

4.7. I cannot see why the Ramipril could not have been introduced at a time subsequent to discharge from hospital, after Mr Raphael's immediate episode was over.

4.8. I note the changes that have been introduced in the organisation and staffing levels in the Vascular Unit at the Queen Elizabeth Hospital. They are well considered and impressive responses to ensure that the chances of a repetition of the events leading to Mr Raphael's death are unlikely to occur. I commend those involved in bringing those changes about. Having regard to those system changed, I do not propose to make any recommendations under S25(2) of the Coroners Act 2003.

Key Words: Hospital treatment; Failure to monitor; Ramipril; nephrotoxic drugs;

In witness whereof the said Coroner has hereunto set and subscribed his hand and Seal the 25th day of January, 2006.

State Coroner

Inquest Number 27/2005 (1542/01)