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 31st day of January 2006, the 1st day of February 2006, the 12th day of April 2006 and the 21st day of April 2006, by the Coroner's Court of the said State, constituted of Mark Frederick Johns, State Coroner, into the death of Ruby Mabel Edwards.

The said Court finds that Ruby Mabel Edwards aged 77 years, late of Gilbert Valley Senior Citizens' Home Incorporated, 20 Masters Road, Riverton, South Australia died at the Riverton and District Soldiers Memorial Hospital, 23 Moorhouse Terrace, Riverton, South Australia on the 30th day of September 2003 as a result of haemorrhagic diathesis due to warfarin toxicity. The said Court finds that the circumstances of her death were as follows:

1. Introduction and reason for Inquest

1.1. Ruby Mabel Edwards died on 30 September 2003 at the Riverton and District Soldiers Memorial Hospital, having been admitted on 8 July 2003. She was 77 years of age. A post mortem examination conducted by Dr Karen Riches gave the cause of death as haemorrhagic diathesis due to warfarin toxicity. Ms Edwards had a history of Alzheimer's type dementia, hypertension, osteoporosis, anaemia and mild renal failure. She had undergone an aortic valve replacement in 1995 for which she was on anticoagulant therapy with Warfarin.

1.2. A report of death form was transmitted to the State Coroner on the same day. It was completed by Dr George Alcorn who was the visiting medical officer to the Riverton and District Soldiers Memorial Hospital and the medical practitioner responsible for the care of Ms Edwards during her hospitalisation. In the report of death form

Dr Alcorn suggested Ms Edwards cause of death was "acute blood loss due to Warfarin therapy due to aortic valve replacement".

1.3. On 3 October 2003 a warrant was issued for the seizure of the medical casenotes relating to Ms Edwards and once obtained those notes were submitted to Professor Roger Byard, Forensic Pathologist, who reported that an autopsy was warranted to determine the precise cause of death and the relationship between that cause and the coagulation times. Accordingly, an autopsy was carried out by Dr Karen Riches on 9 October 2003. A report of the autopsy results was provided by Dr Riches. The report is dated 24 October 2003 and was admitted as Exhibit C3 in the Inquest. As already noted, Dr Riches gave the cause of death as haemorrhagic diathesis due to warfarin toxicity.

1.4. The case was reviewed by Professor Alex Gallus who provided a report dated 22 March 2005. In that report Professor Gallus commented that as a patient, Ms Edwards represented a difficult management problem. However, he considered that, on the information then known to him, the anticoagulant management of Ms Edwards was deficient during the three weeks preceding her death.

1.5. Accordingly, the State Coroner determined that an Inquest was necessary and desirable.

2. Autopsy findings – evidence of Dr Karen Riches

2.1. Dr Riches gave evidence at the Inquest. Dr Riches holds a Bachelor of Science degree with honours from the University of Western Australia, a Bachelor of Medicine and a Bachelor of Surgery with honours from the University of Western Australia and a Fellowship of the Royal College of Pathologists of Australasia which she obtained in 2005. At the time of the post mortem examination she was a full-time trainee pathologist working in the Forensic Science Centre in South Australia. The supervising forensic pathologist was Dr John Gilbert. Dr Riches was a careful and considered witness and gave her evidence clearly and helpfully. I have no hesitation in accepting her evidence.

2.2. Dr Riches explained that haemorrhagic diathesis may be described as haemorrhagic disorder in the sense that the deceased had evidence of bleeding from different organ sites or from different sites in the body, suggesting that there was some underlying

defect of the normal clotting cascade that would occur in a healthy person (T7). Dr Riches gave evidence at T10 to T11 as follows:

> 'The cause of death was bleeding or haemorrhage manifest in the gastro-intestinal tract, lungs, skin and around the ovaries. The most significant of those would have been in the lungs. The effect on the body would have been two-fold; both due to the loss of blood and also due to the filling of the lungs with fluid which would have inhibited breathing and exchange of oxygen from the air into the blood.' (T11)

2.3. Later at T11 Dr Riches explained that it is in the nature of haemorrhagic disorder that the sites of bleeding can be multiple. She described it as an oozing of blood from multiple different places and tissues with a number of small defects in vessels. She contrasted this with the example of a traumatic injury which might sever a particular blood vessel in one place, in which case it is much easier to localise the site of bleeding.

2.4. Dr Riches also noted that the deceased had an artificial aortic valve which was in good working order.

2.5. Dr Riches gave evidence about the Warfarin therapy prescribed to Ms Edwards. She explained at T13 that the reason for the Warfarin therapy was because of the past medical history of the prosthetic aortic valve in 1995. Dr Riches explained that when a person has an artificial heart valve, the body recognises the foreign body and there is an increased risk of a clot forming around the heart valve. Warfarin is administered to inhibit the clotting process and reduce the risk of a clot forming on the artificial heart valve (T14).

2.6. Dr Riches gave evidence that Warfarin is a drug that is metabolised very differently in different people and the same volume of the drug can have different effects in different people. Accordingly, the effect of the drug is measured using a blood test called an INR test. INR is an abbreviated for International Normalised Ratio (T12).

2.7. Dr Riches gave evidence that treatment with Warfarin is a balancing act between the risk of clotting on the one hand and the risk of bleeding on the other. She said that as you increase one you decrease the other and therefore the aim of the therapy with Warfarin is to find a safe middle ground where the risks of both extremes are minimised (T21).

2.8. From her careful examination of the hospital notes, and in particular, the IMVS blood INR results, Dr Riches prepared a chart showing the INR measurements on the dates that it was measured, and the Warfarin dose on the days that Warfarin was administered. The chart covers the whole of the period from 8 July 2003 to Ms Edwards death on 30 September 2003. It was tendered and admitted in evidence as Exhibit C5. The chart is reproduced hereunder:

DATE	INR	WARFARIN 5MG	WARFARIN 1 MG	TOTAL WARFARIN	OTHER READINGS
8 JUL	6.6	Defer	-	-	
9	4.4	Defer	-	-	
10		Defer	-	-	
11	2.9	5	-	5	
12		5	-	5	
13		5	-	5	
14	1.6	5	-	5	
15		5	-	5	
16		5	-	5	
17	1.8	5	1	6	
18		5	1	6	
19		5	1	6	
20		5	1	6	
21	2.4	5	1	6	
22		5	1	6	
23		5	1	6	
24		5	1	6	
25		5	1	6	
26		5	1	6	
27		5	1	6	
28	3.3	5	1	6	
29		5	1	6	
30		5	1	6	
31		5	1	6	
1 AUG		5	1	6	
2		5	1	6	
3		5	1	6	
4		5	1	6	
5		5	1	6	
6		5	1	6	
7		5	1	6	
8		5	1	6	
9		5	1	6	
10		5	1	6	
11		5	1	6	
12		5	1	6	
13		5	1	6	
14		5	1	6	
15		5	1	6	
16		5	1	6	
17		5	1	6	
18		5	1	6	
19		5	1	6	
20		5	1	6	
21		5	1	6	
22		5	1	6	

23		5	1	6	
24		5	1	6	
25		5	1	6	
26		5	1	6	
27		5	1	6	
28		5	1	6	
29		5	1	6	
30		5	1	6	
31		5	1	6	
1 SEPT		5	1	6	
2	8.9	W/H	W/H	-	
3		W/H	W/H	-	
4		W/H	W/H	-	
5	2.6	W/H	W/H	-	
6		W/H	W/H	-	
7		?	?	?	
8	1.2	?	?	?	
9		5	-	5	
10		5	-	5	
11		5	-	5	
12		5	-	5	
13		5	-	5	
14		5	-	5	
15		5	-	5	
16		5	-	5	
17		5	-	5	
18		5	-	5	
19		5	-	5	
20		5	-	5	
21		5	-	5	
22		5	1	6	
23		5	1	6	
24		5	1	6	
25	8.1	5	1	6	
26		5	-	5	
27		5	-	5	
28		W/H	-	-	
29	12	W/H	-	-	
30		W/H	-	-	

2.9. The chart, Exhibit C5, is self-explanatory. Dr Riches explained that she was unable to determine from the notes whether Warfarin was or was not administered on the 7th and 8th of September (T42). Furthermore, she was uncertain, having read the notes, about the administration of Warfarin on each of the 22nd, 23rd, 24th and 25th days of September, at least so far as the dosage of 1 mg for those days was concerned, because the relevant dose chart contains a nursing signature suggesting that the 1 mg dose was administered on each of those days. However, for reasons which were never explained by the evidence, a line appears through those initials. It impossible to know what to make of the line through the initials, particularly bearing in mind for the ensuing days until Ms Edwards' death, there is another notation and mark in the hand of Dr Alcorn clearly showing that further administration of 1 mg Warfarin was

cancelled. I am inclined to think that the 1 mg of Warfarin was in fact administered in the period between 22nd and 25th September inclusive, and I cannot account for the line which has subsequently been drawn through the initials for those days.

2.10. Exhibit C5 is a very helpful summary of the course of the Warfarin therapy during Ms Edwards' stay in the hospital. It also helpfully shows the results of the INR tests for the days on which they were done. It shows that INR was tested on twelve occasions between 8 July and 29 September.

3. INR levels during hospitalisation

3.1. The hospital notes were tendered and admitted as Exhibit C2. The copies of the original IMVS test results are contained within the notes. Each of the IMVS reports contains a statement to the following effect in relation to the INR measurement:

> 'The recommended therapeutic range for oral anticoagulant therapy is 2.0-3.0. For patients with prosthetic heart valves the recommended ranges are 2.5-3.5 (low risk valves) and 3.0-4.5 (high risk valves).'

3.2. The evidence at Inquest did not reveal whether Ms Edwards had a low risk valve or a high risk valve.

3.3. As already noted, Ms Edwards was admitted on 8 July 2003. Her course of treatment shows that her INR levels waxed and waned. The table prepared by Dr Riches which appears earlier in these reasons shows a high INR of 6.6 on 8 July, dipping to a low of 1.6 on 14 July, following a withdrawal of treatment in response to the earlier higher reading. The INR was not checked for a substantial period from 28 July until 2 September when the reading was above acceptable limits at 8.9. At this point Warfarin treatment was withheld again with the result that the level dipped to an unacceptable low of 1.2 on 8 September.

3.4. In referring to these levels as unacceptably high or low I am having regard to the recommended therapeutic range referred to in the IMVS reports. I am also having regard to the consensus guidelines for Warfarin therapy published in the Medical Journal of Australia in 2000 which were tendered in the proceedings and became Exhibit C10. According to the abstract appearing on the front page of the guidelines, the anticoagulant effect of Warfarin should be kept at an international normalised ratio (INR) of about 2.5, the desirable range being between 2.0 and 3.0. The risk of bleeding increases exponentially with increasing INR and becomes clinically

unacceptable once the INR exceeds 5.0. The evidence given at Inquest was to the effect that an INR of less than 2.0 gave rise to an unacceptably high level of risk of clotting around the prosthetic heart valve.

3.5. The next INR reading was on 25 September when it was 8.1 and then again on 29 September when it was 12. Unfortunately on the following day Ms Edwards died as a result of the haemorrhagic diathesis before her INR could be restored to therapeutically acceptable levels.

4. Warfarin therapy during hospitalisation – Dr Alcorn – the nature of his practice

4.1. Dr George Bruce Alcorn was called to give evidence at the Inquest. Dr Alcorn is a general practitioner who practises at Riverton. The evidence showed that, like many country GPs, Dr Alcorn has a very heavy workload and must face a very high demand for his services.

4.2. In 2003, Dr Alcorn had approximately 2300 people registered as patients at his practice (T64). He was also the visiting medical officer at the Riverton and District Soldiers Memorial Hospital which was a 28 bed hospital at the relevant time (T64). In 2003, and for that matter, at the time of the Inquest, Dr Alcorn was on-call for the hospital twenty-four hours a day seven days a week, apart from Wednesday afternoons when he normally comes to Adelaide. He described this in evidence as his "sanity break". I do not consider that Dr Alcorn was trivialising when he used that description. I accept that his workload and responsibilities as the only GP at Riverton are such that one afternoon per week could be regarded as a very modest allocation for his regular rest and recreation.

4.3. He gave evidence at T66-T70 about his working hours. The evidence was to the effect that he has consultations with patients at his surgery Monday, Tuesday, Thursday, Friday and Saturday (morning only). Dr Alcorn also gave evidence that he regularly visited the hospital prior to consulting at his surgery in the morning on Monday, Tuesday, Thursday and Friday, and then would return to the hospital after surgery on Monday, Tuesday, Thursday, Friday and Saturday. Furthermore, he would attend at the hospital on Wednesday morning and also on Sunday morning until around one o'clock. Apart from those regular hours, he would be on-call for the hospital.

4.4. Dr Alcorn gave evidence that between 17 and 31 August he took leave of absence from his surgery and also from the hospital. He was absent altogether from Riverton during that period and a locum practitioner looked after his practice for him. It is notable that he returned on 1 September 2003 and ordered an INR test almost immediately, the result of that coming in on 2 September 2003 at 8.9.

4.5. At T83 he gave evidence that when Ms Edwards came under his care in July he effectively maintained the Warfarin doses that she had been on prior to admission apart from the initial withholding after the high reading on 8 July. At T83-T84 he asked about the reading on 8 September of 1.2 and stated:

> 'It's far too low for someone who I thought had a heart valve.'

Later on that page he was asked about his treatment strategy as at 25 September when the reading was again 8.1. He evidence was as follows:

> 'Looking at the two previous occasions when I had stopped it the level had dropped far too low and I thought if I dropped it 1 mg and re-checked after three or four days that there may be a reduction back into the normal range and that was what I was trying to achieve without stopping the Warfarin altogether and getting a very low reading just to try and ease the level back.'

4.6. Dr Alcorn was asked about the possibility of treatment with intravenous vitamin K. At T85 he explained that he did not pursue that strategy because with intravenous vitamin K there can be an unpredictable drop in INR levels and also because there is a small risk of anaphylaxis with vitamin K. Furthermore, there would be an element of distress to Ms Edwards in the intravenous administration of vitamin K.

4.7. At T98 Dr Alcorn again reiterated his earlier evidence that as at 25 September his state of mind was that if he stopped or withdrew Warfarin altogether, Ms Edwards might have dropped her INR levels to a very low level of 1.2 or least less than 2, with a risk of embolus or clotting. Therefore his aim was just to try and reduce it slowly because he thought that with her clinical condition he would have time to do that.

4.8. Dr Alcorn also gave evidence of the practical difficulties involved in obtaining blood test results from approximately midday on Saturday until Monday morning because, between those times, regular couriers do not attend from the IMVS at Riverton.

4.9. Ms Edwards' hospital notes, Exhibit C2, record that Ms Edwards had a blood transfusion on 5 September 2003. Dr Alcorn was asked why that treatment was

ordered at T120 and he answered that it was ordered because her haemoglobin was 79 which was low and he arranged for the transfusion in the hope of pushing her haemoglobin up to improve her clinical position.

4.10. After reviewing the evidence about Warfarin dosages, I believe that the most likely position is that Warfarin was given to Ms Edwards on 7 and 8 September (T113-T114). There was some doubt about whether the 1 mg Warfarin was administered on each of the 22, 23, 24 and 25 September (T123-T124). On the basis of that evidence, and also an entry in the progress notes in Exhibit C2 for 1700 hours on 25 September 2003 which records that Warfarin 1 mg was "ceased by MO" (where "MO" is an abbreviation for medical officer). I infer that Warfarin 1 mg was being administered between 22 and 25 September (inclusive).

4.11. It was clear from Dr Alcorn's evidence that when the high INR reading of 8.1 was reported on 25 September 2003, he ordered that the Warfarin 1 mg be ceased, but not the Warfarin 5 mg. He was reluctant to cease Warfarin altogether, having regard to the previous experience when, on doing that, the INR levels had dipped to a dangerously low reading. He therefore ceased the Warfarin 1 mg but did not cease the Warfarin 5 mg. However, he did cease it altogether on 28 September, and from that day onwards until the time of her death, no Warfarin was administered to Ms Edwards.

5. Evidence of Professor Gallus

5.1. As already noted, Professor Alex Gallus provided a report at the request of Counsel Assisting. Professor Gallus is a haematologist currently working at the Flinders Medical Centre. He is also the director of the pathology services at Flinders Medical Centre and anticoagulant treatment has been one of his major clinical and research interests for the past thirty years.

5.2. At T135 Professor Gallus explained the anticoagulant effect of Warfarin. He said that it is a vitamin K antagonist and it prolongs the blood clotting time by interfering with the manufacture of blood clotting factors. He explained that the effect of Warfarin is measured with the INR which he described as the standardised process to determine the intensity of the anticoagulant effect. He explained that an INR of 1 represents normal blood coagulation, and an INR of 2 means that in the test tube the time taken

by the blood to clot is doubled. An INR of 3 means the coagulation time is tripled, and so on.

5.3. Professor Gallus gave evidence that the target levels of INR which have already been referred to, are also set out in the Consensus Guidelines for Warfarin Therapy (Exhibit C10) of which Professor Gallus is co-author. He said the Consensus Guidelines for Warfarin Therapy were developed on behalf of the Australasian Society of Thrombosis and Haemostasis with the aim of assisting medical practitioners to negotiate a path between either too little Warfarin or too much Warfarin.

5.4. Professor Gallus said that the effectiveness of the Warfarin and the resulting INR levels will vary with a number of factors particular to the individual. At T138 Professor Gallus said:

> 'I mean, Warfarin is a notoriously tricky drug, it fluctuates in terms of its effect within individuals, between individuals and while there are people in whom the effect is quite stable for many years on end, if things then happen to them that effect will change. There are some people in whom the effect is actually quite unstable or brittle and that has to do with age and other medications and regularity of intake and dose absorption and other illnesses and so on and so forth.'

5.5. Professor Gallus explained that in a patient such as Ms Edwards the risk of too low an INR measure is that there will be clotting from the prosthetic heart valve, and the risk of too high an INR reading is that the patient will be at risk of bleeding.

5.6. Professor Gallus did not criticise Dr Alcorn for not ascertaining what sort of prosthetic valve Ms Edwards had (T143).

5.7. At T144-T145 Professor Gallus explored the difficulties confronting a general practitioner in a country practice in applying the publishing consensus guidelines for Warfarin therapy. At T145 he said:

> '…but practitioners need help in that regard.'

5.8. Professor Gallus had no criticism of the Warfarin therapy administered by Dr Alcorn during the month of July. It will be remembered the no INR measurements were carried out during the month of August, and that for half of that month, Dr Alcorn was on leave from Riverton altogether. Professor Gallus was asked (T154) whether there should have been more testing after 28 July. He replied that he thought "should" is a

very strong word but that it might have been better to test earlier and suggested two to three weeks as a time frame. At T154 Professor Gallus made the following remark:

> 'Sure and let's face it, if one looks at the degree to which anticoagulant control in say an anticoagulant clinic environment, actually manages to achieve that 2-3 or two and a half to three and a half, any series you look at will tell you that the ability to keep people within that range is very variable and the reports have ranged from as low as 40% within range and maybe 25 below and 15 above or something, as high as 80 or 90% and yes one is constantly trying to do better but I know in my clinic I'm doing well if I have 75% of people within range. It is just not possible to have perfect anticoagulant control in everybody, which is why if you are very wary about these things, you sort of start to develop maybe a bit of an instinct which will never be always right as to when you test a little sooner and when you don't, but nobody gets it right all the time I tell you.'

5.9. The table referred to earlier shows that there were no INR tests between 8 September and 25 September. Professor Gallus expressed the view at T163 that it would have been prudent to have tested INR every two to three days during that period.

5.10. Professor Gallus expressed the view initially that Dr Alcorn's decision to reduce Warfarin by 1 mg only after receiving the INR report of 8.1 on 25 September 2003 was a puzzling response (T164). He was informed of Dr Alcorn's explanation that he only reduced it by that amount because of his previous experience with the INRs dropping to a low figure after complete cessation. Professor Gallus was asked, bearing in mind the past experience that Dr Alcorn had had with Ms Edwards, whether his action was appropriate. Professor Gallus said:

> 'I have enormous sympathy. Anti-coagulation with Warfarin is not easy and you're always caught in the jaws of this dilemma where you are either too high, or if you over react to that, you're too low. …To me that was a mistake, but an understandable one in logic.'

5.11. At T166 Professor Gallus noted that it is always possible to break tablets in half to give greater flexibility in dosing, an option which Dr Alcorn did not seem to have considered.

5.12. Counsel put to Professor Gallus some of Dr Alcorn's further concerns about the difficulties that he had in taking blood from Ms Edwards because of the distress it caused her and because of her various medical problems. His response was as follows:

> 'You're painting a very vivid and entirely plausible picture and I mean you're now taking us into the area of how you manage the late sort of follow up of an intervention which in 1995 when the lady was younger and fitter and weller was obviously an important thing

> for her to have, but then as she gets older as we get older, it just becomes more and more difficult to manage some of these issues. Anticoagulation in the elderly is a huge challenge and you actually manage as best you can and as best the circumstances allow and you may well be in a situation where you start feeling that the blood-letting is difficult to achieve etc. etc. etc. How one would manage this face to face and on the day I do not know.'

5.13. I think it is fair to summarise Professor Gallus' evidence as being that while death from haemorrhage could have been prevented by closer attention to Warfarin monitoring, Warfarin dose adjustment and reversal of excessive Warfarin effects, he was very sympathetic to the difficulties confronting Dr Alcorn as a country GP attempting to manage coagulation under very difficult circumstances (T174).

5.14. At T178 Professor Gallus conceded:

> 'Really what we're talking about is not could we have prevented death, but what is our choice of death. The patient at some stage would die, happened to have died a bleeding related death, but there are an awful lot of other things going on which as you say, were limiting things seriously. One of the things that we probably ought as a profession be doing more and we do very uncomfortably, is address these issues of life and death and when we pass certain thresholds in terms of what level of management we really are going to apply, given the individual's sort of overall sort of integrity as a human being. I'm not sure I want to go there.'

5.15. At T180 Professor Gallus referred to one option which might have assisted the management of Ms Edwards in the country hospital setting. He said that there are now starting to become available what he described as "point of care devices" which are little handheld devices that one can use to take a small drop of finger-prick blood and by means of a cartridge and a slide, feed the sample into the device and obtain an INR result. According to Professor Gallus these devices are simple and easy to use and not necessarily terribly expensive.

5.16. Professor Gallus provided further information on point of care devices by letter dated 1 March 2006:

> 'Point of Care testing and particularly self testing and/or self management using Point of Care testing are evolving areas of clinical practice, and I refer you to the following recent review articles partly or wholly concerned with this:
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> J Ansell et al, The Pharmacology and Management of the Vitamin K Antagonists: Seventh ACCO Conference on Antithrombotic and Thrombolytic Therapy, Chest, Volume 126 September, 2004 Supplement: 204S-233S

DA Fitzmaurice et al, An Evidenced Based Review and Guidelines for Patient Self Testing and management of Oral Anticoagulation: British journal of Haematology, 2005; 131 : 156-165

C Heneghan et al, Self Monitoring of Oral Anti-coagulation: A Systematic Review and Meta Analysis, The Lancet, 2006; Volume 367, Pages 404-411.

There is now a point of Care testing trial in a general practice setting funded by the Commonwealth government at the university of Adelaide with Briony Glastonbury as the Trial Manager. The address is Data management and Analysis Centre, Department of Public Health, mail Drop 511, The University of Adelaide, SA 5005. Alternatively, the other contact person is Carolyn Laurence, Trial Evaluation Manager and Senior Research Fellow, Department of General Practice, Faculty of Health Sciences, Level 3, Eleanor Harrold Building, Royal Adelaide Hospital, The University of Adelaide, SA 5005.'

5.17. At T183 Professor Gallus suggested that it would be useful in the INR reports which are supplied to doctors if there were some form of warning which he described as "a sort of a blinking light on the computer screen which says, this is a hazardous level of INR, here are the guidelines, consider this". He referred to work being done in association with a project called the Oasis project of clinical information system management which might assist in computer-aided management of patients. I see considerable merit in this notion of particular warnings being provided to a reporting practitioner when reports from pathology services exceed a particular threshold level.

6. Conclusion

6.1. Professor Gallus placed the circumstances in which Dr Alcorn found himself in a very helpful perspective. He very frankly acknowledged the plight of the general practitioner describing it as "the world's most difficult job". He acknowledged that there is a great deal of difference between practising at a very high level in a narrow area of expertise on the one hand, and being a general practitioner having to deal with "not half a dozen guidelines, but with dozens and dozens of them, covering all the various aspects of their practice. It's a major challenge to the system to actually get all of those sort of bits of wisdom to somehow be applied."

6.2. Dr Alcorn's position is not, and was not in 2003, an enviable one. He is trying to service a very large group of people in a country area who are highly reliant upon him. He stated at T132 "but there has always been an expectation for the whole time that I've been in rural practice that if you are the doctor in the town, you provide services to all patients in the hospital." He explained that "at Riverton because I'm

the only doctor, I provide services to every patient in the hospital." The following passage appears at T133:

> 'Q. So if you were feeling that everything was too much for you, to whom would you speak if anyone.
>
> A. The only two options I have if the load gets too great, are either to retire quick and leave the board to provide another medical officer. To put that in context, before I arrived in 1999 … there was no resident doctor in Riverton from 1997 to 1999 and the community have tried to get a doctor available from that time.'

6.3. In the circumstances, I find that Dr Alcorn provided as good a service as could have been expected in the circumstances. Perhaps with the aid of warning systems such as might be offered by the Oasis project, or with the use of the portable devices referred to by Professor Gallus, the situation of patients in remote country hospitals could be improved. There is clearly a very strong public interest for the community in remote areas in attracting and retaining people dedicated enough to provide services as a country general practitioner. Practitioners need to be encouraged to work in regional and remote communities, and it is important to resist the temptation to judge their efforts by the benchmark of the standards expected in major public hospitals. In making these remarks, I am in no way suggesting that people in regional and remote areas should not receive the very highest standards of care. However, the full burden for that should not be laid entirely on GPs who are dedicated enough to practise in such remote and isolated locations.

7. Recommendations

7.1. I recommend that the Department of Health consider the research referred to in the letter from Professor Gallus quoted above, with a view to assisting General Practitioners in remote areas with anti coagulation therapy management.

7.2. I further recommend that the Department of Health consider the use of the Oasis project of clinical information system management with a view to providing a warning to clinicians to whom test results are directed that the report is showing a hazardous level of INR.

Key Words: Hospital treatment, Warfarin therapy

In witness whereof the said Coroner has hereunto set and subscribed his hand and Seal the 21st day of April, 2006.

State Coroner

Inquest Number 11/2006 (2811/03)