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 2nd, 3rd and 4th days of June 2009 and the 4th day of February 2010, by the Coroner's Court of the said State, constituted of Anthony Ernest Schapel, Deputy State Coroner, into the death of Olga Krivitch.

The said Court finds that Olga Krivitch aged 81 years, late of 12 Adaluma Drive, Pooraka, South Australia died at the Royal Adelaide Hospital, North Terrace, Adelaide, South Australia on the 29th day of January 2006 as a result of hypovolaemic shock following bleeding from the site of a femoral angiogram. The said Court finds that the circumstances of her death were as follows:

1. Introduction

1.1. Ms Olga Krivitch was 81 years of age when she died on 29 January 2006 at the Royal Adelaide Hospital (RAH). Ms Krivitch had been admitted to the RAH on 22 January 2006.

1.2. Ms Krivitch's death was reported to the State Coroner. Although no post mortem examination was carried out, Dr Barbara Koszyca, a Forensic Pathologist, reviewed Ms Krivitch's RAH clinical record. Dr Koszyca prepared a report dated 31 January 2006 that was subsequently verified by way of her affidavit[1]. In her report Dr Koszyca expresses the opinion that the cause of Ms Krivitch's death was hypovolaemic shock following bleeding from the site of a femoral angiogram. Hypovolaemic shock is the product of acute blood loss that in Ms Krivitch's case was the result of over-anticoagulation and persistent coagulopathy. Anticoagulation is a

[1] Exhibits C3 and C3a

measure designed to prevent or inhibit clotting of the blood. A femoral angiogram is a procedure designed to facilitate radiological assessment of blood perfusion within a person's leg. It involves the puncturing of the femoral artery and the insertion of a catheter at the puncture site.

1.3. Ms Krivitch's care was also reviewed by an intensive care specialist, Professor Jack Cade, who is the Principal Specialist in Intensive Care at the Royal Melbourne Hospital. Professor Cade has reviewed the RAH clinical record regarding Ms Krivitch's admission in January 2006. Professor Cade prepared a number of reports that were tendered to the inquest. In one of his reports[2] Professor Cade also expresses the view that the cause of Ms Krivitch's death was hypovolaemic shock following bleeding from the site of a femoral angiogram. Having considered the reports to which I have referred, and the opinions of Dr Koszyca and Professor Cade, I find that the cause of Ms Krivitch's death was hypovolaemic shock following bleeding from the site of a femoral angiogram. During the course of this Inquest there was some debate concerning the site of the bleeding which led to Ms Krivitch developing hypovolaemic shock. I deal with that issue within these findings but I indicate here that I have concluded that the site of the bleeding that led to Ms Krivitch experiencing hypovolaemic shock was indeed the site of her femoral angiogram. In these findings I will also deal with the circumstances in which Ms Krivitch came to undergo a femoral angiogram as well the manner in which bleeding from that site led to Ms Krivitch's ultimate demise.

1.4. In this Inquest I also examined how it came to pass that Ms Krivitch became over-anticoagulated following her femoral angiogram and how bleeding from the original puncture site became so significant as to lead to hypovolaemia and the resultant shock.

2. The circumstances of Ms Krivitch's admission to the RAH

2.1. Ms Krivitch was admitted to the RAH on Sunday 22 January 2006. For the previous few days she had been experiencing symptoms that included a cold and painful right foot. Ms Krivitch had a past medical history of hypertension (high blood pressure), hypercholesterolaemia (high cholesterol), hypothyroidism and ischaemic heart disease. On admission to hospital she was diagnosed as having an ischaemic right

[2] Exhibit C9

foot, which essentially means that the lower part of her right leg was receiving a significantly diminished blood supply. This was diagnosed by way of an angiogram that was administered in the Department of Radiology at the RAH. The angiogram revealed that Ms Krivitch had at least one blood clot within one of the arteries in her leg and possibly others as well. Ms Krivitch immediately underwent thrombolytic therapy for the clot or clots. Unfortunately this procedure was unsuccessful in her case. The angiogram and the thrombolysis were both administered by Dr David Taylor who was the Director of Radiology at the RAH. He administered the procedure during the course of that Sunday afternoon.

2.2. The angiogram and thrombolytic therapy required Dr Taylor firstly to administer dye through a catheter inserted into the femoral artery above the knee and then administer the thrombolytic therapy, consisting of the drug Urokinase, through another catheter inserted into the femoral artery. The entire procedure involved two punctures of the femoral artery, one of 1mm in diameter and the other 1.5mm in diameter. It is said that the bleeding episode, which later resulted in Ms Krivitch developing hypovolaemic shock and persistent coagulopathy, was the site or sites of the femoral arterial punctures. Indeed, in due course Ms Krivitch was to undergo surgery for the oversewing of the femoral artery puncture site in order to stem further loss of blood into the surrounding tissues. Unfortunately the surgery could not reverse or arrest what by that time had been a significant decline in Ms Krivitch's wellbeing.

2.3. One issue that arose for consideration at the Inquest can be disposed of quite readily. Insofar as it has been suggested that the site of the femoral angiogram ought to have been the subject of a vascular plug so as to prevent bleeding from the site, I would not accept that as a valid criticism for the reasons advanced by Dr Taylor in his evidence before me. Dr Taylor explained that he would only utilise such a plug if there were two of three factors present: the patient is extremely obese; the patient is anticoagulated; or the puncture site is greater than 3mm. As we know, Ms Krivitch was to be anticoagulated but she was neither obese nor had a puncture site greater than 3mm. Dr Taylor further explained that in order to insert a plug he would have needed to expand the existing puncture site of 1.5mm to 3mm and he believed that this would have been contraindicated in all of Ms Krivitch's circumstances. It is impossible to say that Dr Taylor's approach was anything other than reasonable.

2.4. When Dr Taylor performed the femoral angiogram and administered the thrombolytic therapy, he administered 200,000 units of Urokinase which is a drug that is designed to dissolve clots. He also administered 5,000 units of Heparin which in this context assists to prevent the formation of thrombus or clotting within the catheter during the thrombolytic procedure. Heparin is an anticoagulant.

2.5. The reasons why the procedure was unsuccessful in Ms Krivitch's case do not need to be explained in any detail save and except to say that X-rays revealed that the therapy had made no impression upon the clots whatsoever despite competent administration. A decision was taken that no further attempt would be made to dissolve the clot or clots. There is no suggestion that this decision was unreasonable.

3. Ms Krivitch is admitted to the Vascular Surgery Unit of the RAH – The plan

3.1. Following Ms Krivitch's unsuccessful procedure she was admitted to the Vascular Surgery Unit of the RAH, which is one of three units within the Cardiovascular Services Unit at the hospital. At that time Mr Laurence Ferguson was the Director of Vascular Surgery and therefore the head of the unit to which Ms Krivitch was admitted on 22 January 2006. Mr Ferguson is a vascular surgeon whose medical degrees were conferred in Scotland in 1971. Mr Ferguson has Fellowships of the Royal College of Surgeons in both Edinburgh and Glasgow and is a Fellow of the Royal Australasian College of Surgeons. Mr Ferguson gave evidence in the Inquest.

3.2. Mr Ferguson explained to the Court the plan of treatment for Ms Krivitch in the light of the lack of success in dissolving the clot or clots within her right leg. He explained that Ms Krivitch was at all material times in jeopardy of losing her leg to amputation. The primary focus of her treatment plan was to avoid that outcome. He told me that it was very unlikely that the clot would spontaneously dissolve or otherwise remove itself[3]. Moreover, there was no surgical option available to achieve revascularization of the leg. As part of Ms Krivitch's treatment plan, the origin of her leg clotting needed to be identified in order to prevent further embolism. Mr Ferguson explained that there were two possible mechanisms by which leg clots might be established. Firstly, the patient might have diseased arteries such that over a period of years the surface of the artery has roughened and at a particular point in time a clot is formed. The other scenario is that the arteries might be viable but that the clot has come from

[3] Transcript, page 123

the heart. This possibility would have required an evaluation of Ms Krivitch's heart function and this was part of the plan that was developed for her. One possible complication of an embolism that has a cardiac origin is that it might cause a stroke. The possibility of such as outcome needed to be taken into account in Ms Krivitch's case. It was planned that Ms Krivitch would undergo an echocardiogram in order to identify the origin of her embolus. The suspicion that her clotting may have had a cardiac origin was somewhat heightened by Ms Krivitch's statements that she had experienced intermittent palpitations over the past 3 to 6 months. This raised a possible diagnosis of atrial fibrillation, a well understood precipitator of clotting.

3.3. As far as Ms Krivitch's existing difficulty with her right leg was concerned, Mr Ferguson explained that the ultimate goal was to ensure adequate circulation within the leg for Ms Krivitch to survive without losing her leg. There was a possibility that it might recover over a period of time. In order to promote the viability of her leg it was decided that Ms Krivitch should be placed on an anticoagulation regime. This would not dissolve the clots that she had in her leg, but it would prevent clotting in other smaller blood vessels that were keeping the leg alive by way of marginal circulation. Mr Ferguson regarded all of this as standard treatment in a patient with a leg such as that of Ms Krivitch. If everything went according to plan Ms Krivitch would ultimately have been discharged and probably would have been placed on an ongoing anticoagulant such as Warfarin. In the meantime it was necessary for Ms Krivitch, while in the RAH, to be commenced on an anticoagulation regime that consisted of the anticoagulant drug Heparin. It will be remembered that in the course of her unsuccessful thrombolytic therapy Ms Krivitch had already been administered Heparin in order to prevent the development of clots during that therapy. Heparin, like other anticoagulant drugs, can be dangerous if not administered correctly. In order to avoid the complications of excessive anticoagulation, such as copious bleeding, protocols for the administration of Heparin have been developed. The RAH had such a protocol.

3.4. There is no criticism of Mrs Krivitch's plan of treatment, but as will be seen the execution of the plan left something to be desired.

4. Staffing arrangements within the Vascular Surgery Unit of the RAH

4.1. Notwithstanding the fact that Mr Ferguson was the Director of Vascular Surgery and therefore head of the unit, he attended at the RAH on a part-time basis only in order to perform examinations and carry out surgery. This arrangement was conducted by way of a roster. The roster involved Mr Ferguson himself performing 4 half days per week, being Monday afternoons, all day Tuesday and Thursday mornings. In addition, he was available 1 day in 4 for 24 hour emergency calls. There were three other vascular surgeons attached to the unit who covered other days of the week. The week commencing Sunday 22 January 2006 contained the Australia Day public holiday, Thursday 26 January 2006. This meant that Mr Ferguson was not on duty during the course of that Thursday morning. I did not understand that any other consultant was scheduled to perform duties within the unit on that Thursday.

4.2. Normally there would have been two Registrars attached to the Vascular Surgery Unit. Generally speaking, Registrars are trainee medical or surgical specialists who are experienced practitioners and enjoy a measure of autonomy within a unit of a public hospital. They are capable of making, and are expected to make, medical decisions regarding the treatment of a patient. They also have a supervisory capacity with respect to junior medical practitioners. However, during the week in question in January 2006 the unit did not have any Registrars on its staff. Mr Ferguson explained that the unit was expecting a Registrar to come from Melbourne who was to perform the duties of the vascular trainee within the unit, but his arrival was not expected for some weeks. The unit was also expecting the imminent arrival of a very senior overseas practitioner who would perform the duties of the second Registrar. However, this practitioner was required to undergo an English examination notwithstanding that, according to Mr Ferguson, his English was perfect and obviously so. The fact that the practitioner had yet to undergo the exam was delaying his arrival in the unit.

4.3. As well as the two Registrars, a full compliment of medical staff in the unit would also comprise two interns. Interns are medical practitioners who for the most part have only recently finished their basic medical degree and are performing duties within the wards of a public hospital. An internship is generally a year in duration.

4.4. To cover for the fact that the two Registrars were absent, two resident medical officers who are also relatively junior in rank were employed temporarily within the unit. One of those resident medical officers, Dr Nam Viet Pham, gave evidence in the Inquest. Dr Pham had completed his one year of internship at the RAH early in January 2006. It was only by virtue of the fact that he was asked to return to the RAH for a short period in January 2006 that he came to be working in the Vascular Surgery Unit that month. In the event, he worked there for approximately 2 weeks. He and a Dr Mauro, who worked in the unit in the same capacity as Dr Pham, both commenced work in the unit on Wednesday 25 January 2006. Dr Pham did not work on Thursday 26 January 2006 but Dr Mauro did. As I understood the evidence, prior to Drs Pham and Mauro commencing work in the unit on the Wednesday, there had previously been two other junior medical practitioners who had been working there in the same capacity. Dr Hajir Nabi was working within the unit on Sunday 22 January 2006 when Ms Krivitch was admitted and in fact it was Dr Nabi who had commenced Ms Krivitch on the Heparin regime that evening in accordance with the plan that had been devised. Dr Nabi himself had been an intern during the year 2005. Dr Nabi did not have anything further to do with Ms Krivitch's treatment following the commencement of her Heparin regime.

4.5. Dr Pham told me that he and Dr Mauro were essentially 'relievers'[4]. They were there to augment the staff having regard to the fact that there were no Registrars working within the unit at that time. I took Dr Pham to mean that he and Dr Mauro were essentially performing the duties and functions normally performed by Registrars. I am uncertain as to what the precise staffing arrangements had been between 22 and 25 January 2006 when Drs Pham and Mauro commenced. In particular, the evidence as to when Dr Nabi finished his duties within the unit was not clear.

4.6. Dr Nabi gave two statements to the police[5]. He refers to the RAH Heparin Protocol which he says he followed. He said that during his period of internship at the RAH he had received training in the administration of the Heparin Protocol. The Heparin Protocol is designed to quickly identify and establish two basic things, firstly the patient's therapeutic anticoagulation level and also the appropriate therapeutic dosage of Heparin in order to maintain the therapeutic level.

[4] Transcript, page 68
[5] Exhibits C4a and C4b

4.7. Dr Pham, who started on the Wednesday, told me that the four medical practitioners on the ward, consisting at that time of two resident medical officers freshly out of their internship and two interns, were all present on the ward between 8am and 5pm daily. One of the practitioners would also cover the period from 5pm to 8am on an on-call basis. The person on-call would normally be a Registrar and not an intern, but in this case the on-call practitioner had to be one of the residents.

4.8. Dr Pham told me that he assumed that prior to his commencement day there would have been a Registrar working within the unit in addition to Dr Nabi. However, it appears from the evidence of Mr Ferguson that there had been no such Registrar because of a lack of availability of the same at that time of the year. As far as one can tell, for the entirety of Mrs Krivitch's admission from Sunday 22 January 2006 there were no Registrars on the unit and as a matter of certainty there were no Registrars on duty on 25, 26 and 27 January 2006.

4.9. As of Wednesday 25 January 2006 Dr Pham and Dr Mauro were responsible for Ms Krivitch's care[6]. Dr Pham was not rostered to work on Thursday 26 January 2006, but Dr Mauro was on duty that day. Their patient care, of course, was subject to overarching supervision by the consultants attached to the ward, but as described earlier, the consultants were not necessarily present. As will be seen there was, on Friday 27 January 2006, which was to be the critical day as far as Ms Krivitch's chances of survival were concerned, considerable difficulty involved in reaching any of the consultants.

4.10. To summarise, at the time with which this Inquest was concerned there had been two relatively inexperienced resident medical officers in place at the time of Ms Krivitch's admission to the RAH and there was no continuity as far as the identities of the resident medical officers were concerned. There were no experienced Registrars on the ward at any material time and the consultants were not necessarily available at short notice to assist the junior practitioners actually working in the ward. It is hoped that such an unsatisfactory scenario will never be repeated in a major public hospital. The net result of this state of affairs was that Ms Krivitch's anticoagulation status was only brought under control after an extended period of time, and significant and life threatening blood loss was not identified and monitored in an appropriate way. Had there been the appropriate level of expertise deployed within the unit, namely that

[6] Transcript, page 86

provided by experienced Registrars, it is difficult to believe that this scenario would have prevailed. It is an inescapable conclusion in my view that the unsatisfactory level of staffing within the unit had a direct bearing on the unfortunate events that were to transpire in respect of Ms Krivitch's wellbeing.

5. The course of Ms Krivitch's treatment and decline

5.1. I have already referred to the involvement in this Inquest of Professor Cade. Professor Cade is currently the Principal Specialist in Intensive Care at the Royal Melbourne Hospital. He was the Director of Intensive Care at that hospital at one point in time. He has been with the same Department for over 30 years. The Royal Melbourne Hospital is one of the major public academic university hospitals in Victoria. Professor Cade graduated with his basic medical degrees from the University of Melbourne. He subsequently obtained an MD and a PhD both from the University of Melbourne. He is a Fellow or member of a number of learned colleges and institutions nationally and internationally including the Royal Australasian College of Physicians, the Australian and New Zealand College of Anaesthetists, the Joint Faculty of Intensive Care Medicine and the College of Chest Physicians which is an American institution, and the Royal Society of Medicine which is an British institution. He is a Professorial Fellow at the University of Melbourne. Professor Cade has wide practical and academic experience in the sphere of anticoagulation. The topic of his first doctoral thesis was anticoagulation. He was for many years the head of the Anticoagulant Clinic at the Royal Melbourne Hospital and he wrote the original Heparin Protocol. He has published widely over the years in peer-reviewed journals in relation to thromboembolism and anticoagulation. I regarded Professor Cade as an expert in the field of anticoagulation and, in particular, Heparin administration.

5.2. Professor Cade provided a number of reports to the Inquest[7]. He also gave evidence which was unchallenged by counsel acting for and on behalf of the RAH, Mr Bonig.

5.3. Professor Cade's evidence related to two broad areas involved in Ms Krivitch's anticoagulation Heparin regime. Firstly, Professor Cade was of the view that the anticoagulation regime was not stabilised in a timely manner. He believed that her infusion dose was not stabilised quickly enough with the result that she was probably

[7] Exhibits C9, C9a and C9b

over-anticoagulated for some period of time. Secondly, significant bleeding from the site of Ms Krivitch's original thrombolytic therapy was not identified in a timely manner with the result that Ms Krivitch experienced severe blood loss, hypovolaemia and significant coagulopathy from which she simply did not recover.

5.4. As to the first of those issues, namely anticoagulation, Professor Cade expressed the belief that in the light of Ms Krivitch's original thrombolytic therapy, the starting dose of her Heparin anticoagulation infusion, which commenced on the evening of 22 January 2006, was excessive. He was also of the view that the regime of necessary monitoring was, to begin with, not instituted in an appropriate and timely manner for the most part, such that her APTT level, which is the measure of anticoagulation, reached a figure that was too high. While high APTT levels are not unheard of during the course of a Heparin infusion, it is nevertheless important that they be brought under control by means of regular monitoring and adjustment of dosages and rates of infusion. There was some debate in the Inquest as to whether the RAH protocol had in fact been adhered to, and indeed whether the RAH protocol was appropriate in any event, but there was agreement that a legitimate example of what Professor Cade was saying was afforded by the fact that the result of the first APTT test was not acted upon until a period of about 11.5 hours since the commencement of the infusion had expired and that this was by any measure an excessive and unacceptable interval.

5.5. It has not in my view been necessary for the Court to identify the person or persons who were responsible for the monitoring of Ms Krivitch's Heparin infusion and for making the necessary adjustments to it as it appears that all of the medical practitioners involved were at a very junior level. It is sufficient to say that I agree with Professor Cade's unchallenged evidence that it took an excessive period of time for the therapeutic anticoagulation range to be reached and an excessive period of time for a stable Heparin dosage to be established.

5.6. Professor Cade points out that it took 38 hours for the therapeutic anticoagulation range to be reached and about 49 hours for a stable Heparin dosage to be established. He states in his report[8] that those times are considerably longer than would have been likely if the recommended protocol had been followed more closely. As a result of all of this Ms Krivitch was exposed to a significantly increased risk of haemorrhage because of the much higher than necessary doses of Heparin during this period. I

[8] Exhibit C9

agree with those observations and I think it is fair to say that Mr Ferguson did not disagree with those observations either. Mr Ferguson told me, however, that consultants at his level would not be directly involved in the administration of the Heparin regime and the checking of ongoing results unless there was a concern raised by the junior staff. This naturally pre-supposes that junior staff would recognise an issue of concern when they see one. It also somewhat pre-supposes and assumes that there is a level of expertise available within the ward that would have the necessary aptitude to monitor and adjust APTT levels by way of dosage adjustments. It will be remembered that there were no experienced Registrars on the ward at this time.

5.7. As far as the belated recognition of bleeding was concerned, the first evidence of significant bleeding occurred within approximately 24 hours of the commencement of the Heparin infusion when it was recognised that Ms Krivitch was unable to move her left arm. The diagnosis for this only became clear on 24 January 2006 when aspiration of her shoulder showed a haemarthrosis. This evidenced bleeding within Ms Krivitch's shoulder which was of a non-traumatic nature. In other words, it was spontaneous bleeding that was probably the product of the anticoagulation regime. There was really no other sensible conclusion available.

5.8. A descending haemoglobin level may afford some evidence of bleeding into the tissues. In addition to Ms Krivitch's APTT levels being monitored, her haemoglobin levels were also monitored on 22, 23 and 24 January 2006. On 22 January 2006, the day of Ms Krivitch's admission, her haemoglobin level was 119 which is acceptable. On 23 January 2006 it had descended to 100 and on 24 January 2006 it had descended to 98. Those latter two levels are below normal but they would not necessarily warrant a blood transfusion. Naturally one would have to examine the clinical presentation of the patient in conjunction with such abnormal levels. However, it will be remembered that 24 January 2006, the day that the haemoglobin level of 98 was identified, was the day that the non-traumatic bleeding into Ms Krivitch's shoulder was diagnosed. The Heparin infusion was not discontinued on that day notwithstanding. I return to the explanation for that in a moment.

5.9. Whatever action was required on 24 January 2006 in the light of the findings of that day is perhaps not the issue because it is in any event common ground that what was plainly required on 25 and 26 January 2006 was further monitoring of Ms Krivitch's haemoglobin levels. In the event, by the morning of 27 January 2006 Ms Krivitch's

haemoglobin level was to descend to 54 which is an alarming figure that ought to have dictated a cross-matched blood transfusion at the first available opportunity. The drop in haemoglobin was reflective of an undetected copious bleed from the puncture site of Ms Krivitch's original femoral angiography into the surrounding tissues of her right leg.

5.10. An explanation as to why Ms Krivitch's haemoglobin levels were not monitored on 25 and 26 January 2006 is not readily discernible except by reference to the inexperience of the medical staff on the ward. The omission is remarkable when regard is had to the fact that an established pattern of descending haemoglobin levels consistent with a bleeding episode had already been identified as had clinical evidence of such bleeding. Ms Kereru, counsel assisting, asked Mr Ferguson this question:

> 'Q. But in conjunction with these results, we have Mrs Krivitch whose Heparin infusion has been stopped on three occasions by this time, by 24 January because it is too high and we also have a bleed being discovered from a non traumatic site in her shoulder, looking at that in conjunction with these tests, is that alarming to you.
>
> A. Not particularly that level but certainly the overall picture is concerning in that that should have been monitored, the haemoglobin should have been monitored. I would want to know what that was each day, you know.' [9]

Mr Ferguson told me that he himself would not be expected to order the haemoglobin tests but that the junior staff would have been responsible for daily checking of haemoglobin levels and even the intern staff would have had the necessary expertise and knowledge to be able to do that. Again, it just seems obvious to me that the shortcomings relating to haemoglobin monitoring on 25 and 26 January 2006 were occasioned by a lack of experienced staff. The failure to conduct haemoglobin monitoring is all the more perplexing when it is recognised that on 25 and 26 January 2006 APTT levels were established through blood sampling on those days and that it would have been a simple matter to ensure that the necessary sampling for haemoglobin testing was taken as well.

5.11. One matter that I should refer to in this context is the fact that on 24 January 2006 Mr Ferguson himself became aware of Ms Krivitch's spontaneous bleeding into her shoulder. Mr Ferguson told me that he had made the decision that the Heparin infusion should be continued notwithstanding. Mr Ferguson admitted in his evidence that he had made that decision and he was quite happy to defend it on the basis that a

[9] Transcript, page 151

discontinuation of the Heparin infusion would probably have resulted in the amputation of Ms Krivitch's leg which he was very keen to avoid. But Mr Ferguson readily admitted that the continuation of the Heparin infusion would have required appropriate monitoring and indeed that had been his expectation. Mr Ferguson did not give any instructions for haemoglobin level tests to take place after 24 January 2006 as he had expected that they would be done at the initiative of junior staff[10]. It is clear from a Heparin schedule that was tendered in evidence[11] that Ms Krivitch's APTT levels continued to fluctuate, even with the lowering of dosages. In the event the infusion continued to be administered until Friday 27 January 2006 when it was stopped as a result of Ms Krivitch's clear clinical deterioration by then. In the meantime staff of the ward were required to maintain stabilisation of Ms Krivitch's Heparin regime as well as keep an eye on her bleeding status all of which was fundamental to her continued wellbeing. This was a lot to ask of junior staff in the absence of close supervision.

5.12. Mr Ferguson was not to see Ms Krivitch again until 27 January 2006. In the intervening period her Heparin infusion was in place but her haemoglobin levels were not being monitored. Ms Krivitch's next haemoglobin level monitoring occurred on the morning of Friday 27 January 2006. It had descended by that time to the startling level of 54 that I have mentioned and this would have called for immediate rectification by way of a cross-matched blood transfusion. Having regard to the fact that it is highly unlikely that Ms Krivitch's haemoglobin level descended from 98 on 24 January to 54 on 27 January 2006 all at once, it is probable that between 24 and 27 January 2006 her haemoglobin would have been at levels somewhere between 98 and 54 and very likely at times in the 70s and 80s. I was told during the course of the evidence, and there is no dispute about this, that one would be contemplating administering a blood transfusion where a person's haemoglobin level had reached as low as the 70s or 80s and certainly in the 50s. In the event, no transfusion was to be given until the evening of 27 January 2006. In the meantime, Ms Krivitch continued to lose blood volume at a dangerous rate and to a dangerous level. The failure to identify her blood loss had three effects. It meant that her anticoagulation regime was not ceased or modified, it meant that she did not benefit from an early and potentially

[10] Transcript, page 153
[11] Exhibit C11

life saving blood transfusion and it also meant that there was no timely identification and repair of the site of her blood loss.

5.13. In my view it is more probable than not that the descent of Ms Krivitch's haemoglobin levels to the dangerous level of 54 had a substantial role to play in her deterioration, which I will describe presently, and death. Because of the lack of monitoring of the haemoglobin levels on 25 and 26 January 2006, Ms Krivitch was deprived of a chance at survival by having her anticoagulation status revisited and having a blood transfusion administered in that period. She may ultimately have lost her leg, but she may not have died. Indeed, Professor Cade told me that in his view Ms Krivitch's life could have been saved as late as the afternoon of 27 January 2006 if the appropriate action had been taken in a timely manner. I return to that aspect of the matter in a moment.

5.14. I turn to the events of Friday 27 January 2006. Both Dr Pham and Dr Mauro were on duty on that day but there does not appear to have been any consultant on the ward that day. The haemoglobin level of 54 that was revealed on that day is evidenced by a document at page 10 of the clinical record, Exhibit C6. The document is an IMVS Enquiry Result Chart that establishes that on 27 January 2006 a blood sample collected at 10:55am was analysed and gave rise to the result to which I have referred. The result in fact not only reveals a haemoglobin level of 54, but also reveals an APTT anticoagulation level of 109 which is elevated. Dr Pham reviewed Ms Krivitch having regard to that result. In Dr Pham's witness statement[12] he states that in the light of the fact that Ms Krivitch was receiving Heparin, the lower haemoglobin level was indicative of her having experienced a significant haemorrhage. However, as there was no obvious site of bleeding at that point in time, a CT scan was requested in order to identify the site of a possible intra abdominal haemorrhage.

5.15. It is at this point worthwhile observing that on the evening of Wednesday 26 January 2006, Ms Krivitch had been noted by the nursing staff as having continued to complain of nausea and was vomiting small amounts of sputum. She had complained of feeling constipated and had requested an enema. Her vital signs were nevertheless stable. On the morning of 27 February 2006 at 6am Ms Krivitch is noted as having been unable to sleep overnight and was unable to open her bowels. She is also noted, at an unspecified time, as feeling sick and constipated and complaining of pain in her

[12] Exhibit C10

shoulder. There is another nursing note apparently timed at 2:10pm that day which refers to various aspects of Ms Krivitch's care but does not make any reference to an abnormal haemoglobin level. Ms Krivitch was clinically deteriorating. It was also revealed that by that morning Ms Krivitch's renal function had significantly deteriorated since last tested 3 days earlier when it was normal.

5.16. In his evidence Dr Pham elaborated upon his witness statement and said that his recollection was that he was alerted to Ms Krivitch's predicament when either himself or Dr Mauro had been called by an intern who had been advised of the haemoglobin level of 54 and who had shown understandable concern. Moreover, the intern had been unable to obtain any further blood samples for a grouping and cross-matching and needed assistance with that. As a result of this Dr Pham and Dr Mauro examined Ms Krivitch but at that point in time that they were unable to identify any active bleeding. Dr Pham himself obtained blood for cross-matching, the intern having been unable to do that. This was done with a view to administering a blood transfusion. Dr Pham told me that he thought that he conducted this examination at about 3pm because he took further samples at that time as evidenced by the IMVS Enquiry Result Chart[13]. It is not entirely certain at what time the haemoglobin level result of 54 had become available or at what time it had been seen within the unit for the first time. The intern in question does not appear to have made any note in the clinical record of his discoveries and attempts to obtain blood from Ms Krivitch.

5.17. The appointment with the Department of Radiology to enable the CT scan of the abdomen to be conducted was made for 5:15pm.

5.18. Dr Pham appears to have arranged other measures as well. At approximately 5pm he discussed the need for either High Dependency Unit or Intensive Care Unit to be contacted in relation to Ms Krivitch's condition and he also considered and discussed the need for her to be reviewed. In addition, not unnaturally he had also attempted to contact Mr Ferguson on his mobile phone via the hospital switchboard but had been unsuccessful. He also attempted to contact a Mr Berce, the vascular surgeon on-call, with similar results. He left a message for either Mr Ferguson, Mr Berce or both and in the message outlined his concerns about Ms Krivitch's deteriorating condition. He informed the consultant with whom he left the message of the current plan for a CT

[13] Exhibit C6, page 10

scan of Ms Krivitch's abdomen and pelvis and also mentioned the plan for a blood transfusion. Dr Pham went off duty at about 5:30pm but Dr Mauro remained.

5.19. On 27 January 2006 Ms Krivitch's Heparin infusion was ceased and this decision appears to have been implemented some time during the course of that afternoon. It was established on the afternoon of 27 January 2006 through the further blood sampling that Ms Krivitch's haemoglobin level had descended from 54 to 53 thus confirming the alarming picture as to blood loss. Her AAPT had also ascended to 160.

5.20. Dr Pham in his evidence suggested that he probably did not learn of the haemoglobin level of 54 until about 2:30pm or sometime shortly before 3pm when further bloods were taken which confirmed the low haemoglobin levels. This level had been learnt through an intern. An inference is available that the intern had not learnt of the level until shortly before it was conveyed to Dr Pham. Dr Pham suggested that the earlier sample taken at 10:55am would have been taken by the blood sisters doing their ward rounds at that time. Generally speaking if the blood form stipulates 'urgent' then the blood results would be available within approximately 1 hour. If it is not stipulated as being urgent there can be a greater time lag and it is not uncommon for blood tests to be available only after 3 to 4 hours. However, a result as dramatic as the haemoglobin level of 54 would in any case immediately be called through by the laboratory to the relevant doctor on the ward. Dr Pham suggested that it probably would have been called through to the intern who subsequently called Dr Mauro and himself. Unfortunately, as I say, there are no records of when it was that the haemoglobin level was reported to anyone, save and except for Dr Pham who believes he learnt of it shortly before 3pm. It goes without saying that such a delay in the circumstances was not acceptable and no-one really suggests otherwise.

5.21. There was further delay occasioned before anything meaningful took place that might have precipitated any kind of recovery for Ms Krivitch. Dr Pham left the ward at about 5:30pm by which time no blood transfusion had commenced.

5.22. Professor Cade was of the very firm view that a number of things urgently should have followed from the discovery that Ms Krivitch's haemoglobin level was 54. Professor Cade said:

> 'I think a number of things urgently followed. That is a red flag that needs immediate attention, it needs a number of actions to follow. Firstly it needs to be checked that it is correct; it may have been an erroneous sample, had tissue in it for example, a diluted sample. Then you need to urgently look at the patient, see if they are pale, see if they have got some bleeding and see what their general state is like. If it was believed on all those grounds that the reading is correct then two things have to happen from that: one is urgent repair of the haemoglobin with a blood transfusion; and secondly urgent investigation of the site. Where is all the haemoglobin gone? Where is all the bleeding?'[14]

Professor Cade was also of the view that while the arrangement for scans to identify the source of Ms Krivitch's bleeding was important, it was not a priority. He said that the scan had nothing to do with the need for a blood transfusion. The scan was necessary in order to find the source of the bleeding so as to identify the method by which it should be stopped, but it had nothing to do with the transfusion which had to be instigated in its own right and administered according to the time limits required by the patient's condition. The transfusion was the priority. In this case Professor Cade was of the opinion that the transfusion should have been prompt, should have preceded the CT scan and in any event could have been run during the administration of the scan.

5.23. As things transpired, the commencement of a blood transfusion did not occur until 8:20pm that evening, which was at least 5 hours after the large haemoglobin level drop had been recognised and checked by Dr Pham. The transfusion could plainly have taken place earlier that day and if an urgent request had been made for the results of the haemoglobin level test, or they had been reported and understood more urgently, it is conceivable that the transfusion could have taken place late morning or very early in the afternoon.

5.24. In the event, the CT scan revealed a massive haematoma of the right thigh which was clearly the source of the blood loss. Mr Ferguson was eventually contacted and that evening Ms Krivitch was taken to the operating theatre where the source of the bleeding was oversewn by Mr Ferguson. The surgical procedure took place in the early hours of the morning of Saturday 28 January 2006. Mr Ferguson has specifically recorded in the clinical record[15] that he oversewed the bleeding point in the right femoral artery.[16] Having reviewed X-rays of the general area of bleeding, Dr

[14] Transcript, page 197
[15] Exhibit C6, page 77
[16] Exhibit C6, page 77

Taylor, the practitioner who had originally conducted the femoral angiogram, expressed the view that the bleeding was not sourced from the site of his thrombolytic therapy. However, Mr Ferguson has identified that the source of the bleeding was a puncture of the femoral artery which he oversewed. In my view it is highly unlikely that what Mr Ferguson surgically repaired was anything other than one of the puncture sites that had been administered by Dr Taylor in the first instance. There is no evidence that there was any other trauma to that site. Nor is there reason to suppose that there could have been spontaneous bleeding from the site. Mr Ferguson thought it an unlikely scenario[17]. I find that the source of blood loss was one of the original puncture sites. This is no criticism of Dr Taylor because he was not to know that Ms Krivitch would be over-anticoagulated and not monitored for signs of bleeding. However, the fact that there was a potential site for blood loss that had a traumatic or surgical origin was even more reason for clinical staff in the Vascular Surgery Unit to remain vigilant for bleeding, especially after there had been the non-traumatic bleed in Ms Krivitch's shoulder.

5.25. As far as clinical signs of bleeding were concerned, no person seems to have noticed, or at least noted, whether by 27 January 2006 obvious bleeding was taking place specifically in Ms Krivitch's right thigh. There are nursing notes up to a certain point which suggest that there was nothing clinically visible as far as her right thigh was concerned, but Mr Ferguson told me that by the time he came to examine Ms Krivitch on the evening of 27 January 2006 her leg was more swollen than it had been and was bruised. There is no evidence as to when that would have been obvious within the ward for the first time.

5.26. Mr Ferguson agreed that the matter had been handled poorly within his own unit. He believed that the lack of more senior staff on the ward had contributed to this[18]. He agreed there were a number of delays on 27 January 2006 that '*should have been circumvented*'[19]. He said that IMVS would have regarded the haemoglobin level of 54 as alarming and they probably would have been proactive and have conveyed that to the ward. If this was in fact the case, the failure to act upon that information before about 2:30pm remains puzzling. Mr Ferguson agreed that although the Heparin infusion had come down to a reasonable level in due course, the damage may have already been done in the meantime.

[17] Transcript, page 132
[18] Transcript, page 146
[19] Transcript, page 158

5.27. Mr Ferguson's belief that the unsatisfactory staffing arrangements had contributed to this outcome were reflected in the fact that shortly after these events he decided to close the unit except for emergencies until they secured the services of a Registrar. He told the Court '*I just felt that it was not right that these junior staff were put in these positions*'[20]. It is difficult to disagree.

5.28. In his evidence Professor Cade expressed an opinion that was not challenged that Ms Krivitch would have survived if appropriate interventions including an early identification of her haemoglobin levels on the morning of 27 January 2006 and a blood transfusion had been put in place earlier on 27 January 2006. He said as follows:

> 'I think up until that afternoon any appropriate set of interventions would have lead to her survival. I am not sure what would have happened to her leg, I think its viability was always going to be a little suspect, and an ischaemic leg in a patient of this age is a major problem in its own right. But from the point of view of the information that we have, she would have survived.' [21]

Mr Ferguson himself was not so certain as to the issue of possible survival, but agreed that Ms Krivitch's chances of survival would have been greater if she had received more timely intervention. I accept the unchallenged and better considered evidence of Professor Cade on the issue.

5.29. However, although the bleeding had been controlled following Ms Krivitch's surgery, her chances of surviving had by then diminished given how unwell she had become. She was admitted to the Intensive Care Unit at the RAH, but by that time Ms Krivitch had developed respiratory failure, renal failure, ischaemic hepatitis, metabolic acidosis and an acute myocardial infarction. She deteriorated over the next 24 hours and eventually treatment was withdrawn as it was considered futile. She died at 9:30am on 29 January 2006. Professor Cade has expressed the opinion that following the confirmation of Ms Krivitch's major haemorrhage, the *'rescue process'* cannot be criticised. In his view she had received appropriate surgical repair of the bleeding artery as well as sophisticated and comprehensive intensive care postoperatively. I accept Professor Cade's opinions in this regard. Unfortunately, major organ damage had already occurred and this was not reversible.

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[20] Transcript, page 106
[21] Transcript, page 198

6. Conclusions

6.1. Following unsuccessful angiography and thrombolytic therapy, Ms Krivitch was appropriately placed on a Heparin anticoagulation infusion. However, it took an extended and sub-optimal period of time for a therapeutic anticoagulation range to be reached and for a stable Heparin dosage to be established.

6.2. On 24 January 2006 Ms Krivitch suffered a bleeding episode into her shoulder that had a non-traumatic origin. It is more probable than not that this bleeding episode was the product of Ms Krivitch's Heparin anticoagulation infusion. A decision was nevertheless made to maintain Ms Krivitch on the Heparin infusion in an attempt to save Ms Krivitch's leg and to prevent further clotting.

6.3. However, given the bleeding episode involving Ms Krivitch's shoulder and the fact that the Heparin infusion was continued, it was a fundamental requirement that her haemoglobin levels, which might provide evidence of further bleeding or otherwise, be monitored and evaluated on a daily basis. On 24 January 2006 Ms Krivitch's haemoglobin level was 98. The fact that Ms Krivitch's haemoglobin level had descended from 119 on 22 January 2006 to 98 on 24 January 2006 magnified the importance of the haemoglobin levels being monitored over successive days. There is no evidence that any specific instruction to that effect was given to the clinical staff of the unit as it was expected that staff would appreciate the fundamental necessity to perform such monitoring and to take the initiative in that regard. In the event, Ms Krivitch's haemoglobin levels were not monitored on 25 or 26 January 2006. This was a significant oversight that was to have a direct bearing on Ms Krivitch's decline and death.

6.4. The fact that no specific instruction was given to junior staff to monitor Ms Krivitch's haemoglobin levels on 25 and 26 January 2006 was to my mind a misjudgement given that there was already in existence evidence of spontaneous bleeding that was the product of over-anticoagulation.

6.5. Ms Krivitch's haemoglobin level was not again monitored until the morning of 27 January 2006. On that day the level was determined to be 54. On the afternoon of that day the level was rechecked and was found to be 53. These levels I find were reflective of the fact that Ms Krivitch had been experiencing ongoing and undetected bleeding into the tissues of her right leg from the site of her femoral angiogram. It is

obvious that between 24 January 2006 and 27 January 2006 Ms Krivitch's haemoglobin levels at some point in time would have descended to levels in the 70 and 80 range and that if levels of this kind had been identified as they should have been, this in itself would have invited serious consideration of the administration of a blood transfusion either on 25 or 26 January 2006. It is likely in my view that a blood transfusion would have occurred on either day and that the appropriate clinical and surgical action would have been taken to identify and rectify the source of the bleeding.

6.6. I find that there was a significant and unacceptable delay in appropriate action being taken after Ms Krivitch's haemoglobin level was revealed to be 54. The haemoglobin test sampling was undertaken at 10:55am on 27 January 2006. There is no evidence as to when the result of that test would have been available for the first time, but it was not reported to Dr Pham, one of the two resident medical officers on the ward, until shortly before 3pm that day. Even then there was a considerable delay in administering the necessary blood transfusion. Ms Krivitch's clinical presentation had significantly deteriorated because of the continuing blood loss into the tissues surrounding the site of her femoral angiogram, in particular from a puncture that had been administered in respect of the same. The very low haemoglobin levels in the 50s that were detected on 27 January 2006 were a reflection of all of this.

6.7. Surgery that was conducted in the early hours of the morning of 28 January 2006 involved an oversewing of the puncture site to which I have referred. While this may have resulted in the bleeding into the surrounding tissues being stemmed, it was administered too late to prevent Ms Krivitch's eventual death by way of hypovolaemic shock as a result of the bleeding.

6.8. I find that if Ms Krivitch had been administered a blood transfusion either on 25 or 26 January or the early afternoon of 27 January 2006, and that the source of the bleeding had been identified and stemmed as it could have been, she probably would have survived.

6.9. I find that Ms Krivitch died of hypovolaemic shock following bleeding from the site of a femoral angiogram.

6.10. I conclude that the shortcomings in Ms Krivitch's treatment at the RAH were due to the inexperience of junior practitioners who were staffing the Vascular Surgery Unit

at that time and a lack of supervision of those staff members. It is hard to imagine experienced Registrars, had they been employed within the unit at the time, not ensuring that appropriate monitoring was in place, particularly in relation to Ms Krivitch's haemoglobin levels. Similarly, in my view the delays experienced during the afternoon of 27 January 2006 could have been avoided if more experienced and senior medical staff had been on hand. If appropriate medical expertise had been available within the Vascular Surgery Unit at the relevant times, I find on the balance of probabilities that Ms Krivitch's outcome could have been avoided. Having regard to the inexperience of the medical staff who were employed at the Vascular Surgery unit from 22 to 27 January 2006, there is no evidence to warrant the criticism of any individual junior medical practitioner. They should have been more closely supervised and in particular, it should have been made clear to them that there was a fundamental need for Ms Krivitch's haemoglobin levels to be closely monitored and evaluated on 25 and 26 January 2006.

6.11. To my mind the expectation that the junior staff would of their own initiative conduct the necessary monitoring of Ms Krivitch's haemoglobin levels was misplaced. In my view it is wrong to assume that junior practitioners are all of the same aptitude and temperament, are all at the same level of professional development and all have the ability to act independently of close supervision and at their own initiative.

7. Recommendations

7.1. Pursuant to Section 25(2) of the Coroners Act 2003 I am empowered to make recommendations that in the opinion of the Court might prevent, or reduce the likelihood of, a recurrence of an event similar to the event that was the subject of the Inquest.

7.2. I have already referred to the evidence of Professor Cade in general terms relating to the RAH Heparin Protocol. Professor Cade has expressed the opinion that the protocol requires modification. Specifically, he is of the opinion that the protocol commencement dose of 1400 units per hour, for a person of Ms Krivitch's size was excessive particularly having regard to the fact that Ms Krivitch had already received a Heparin dosage during the course of her anti-thrombolytic therapy. This was to be contrasted with the Royal Melbourne Hospital protocol where a person such as Ms Krivitch would be commenced on a lesser dosage of 1000 units per hour if

thrombolytic therapy has already been utilised. There was also evidence given in the Inquest as to the appropriateness of intervals for the checking of APTT levels. There is some variation between the RAH and the Royal Melbourne Hospital protocols in this regard. The divergence of opinion on technical issues such as those was not capable of resolution in the context of my inquiry. Mr Bonig, counsel on behalf of the RAH, correctly points out that the resolution of these kinds of issues could only be achieved having regard to expert haematological evidence. There was also the complicating factor that the effect of Ms Krivitch Heparin dose that was administered in the course of her anti-thrombolytic therapy may in any case have dissipated before the commencement of her infusion such that it might not have been necessary for the original dose to have been taken into account. In my view it would be inappropriate for this Court to make any specific recommendation about starting dosages and monitoring intervals without such expert evidence. All I can say is that the matter is the subject of legitimate debate and that the RAH should consider the evidence in this case in the light of Ms Krivitch's over anticoagulation and act accordingly.

7.3. There was discussion during the Inquest as to the desirability of a Heparin Protocol making specific reference to the need to consult haematological expertise where the APTT therapeutic range is not established in a timely manner. The RAH Heparin Protocol as it existed at the time with which this Inquest was concerned made no reference to this issue, whereas the Royal Melbourne Hospital protocol contains a stipulation to the effect that if the APTT level is not established within a therapeutic range for 3 consecutive measurements, clinicians should consult with the hospital's department of diagnostic haematology. As Professor Cade pointed out, in this case the therapeutic range for Ms Krivitch was not established until 38 hours had transpired and not before a number of APTT measurements had taken place. There was also the question of delay of the establishment of the therapeutic dosage in her case. Professor Cade pointed out that in Ms Krivitch's case the starting dose was significantly in excess of what her underlying needs turned out to be and it took some considerable time to achieve an appropriate level, thus exposing her to over anticoagulation in the interim. Professor Cade said that ideally a patient should be brought within the therapeutic range within the first 1 or 2 tests and is in any event problematic if it is not brought within the correct range within the first 24 hours. There was also mention of the undesirability of Heparin dosages reaching a certain cumulative level before stability is established. There was no evidence that the junior

staff within the RAH Vascular Surgery Unit consulted haematological expertise at any time despite the difficulty in establishing therapeutic range and dosage. However, a more up to date version of the RAH protocol was tendered before me[22] that does contain a reference to the need to '*contact haematology for advice*' if the patient were to require greater than 40,000 units of Heparin or 24 hours to achieve a therapeutic APTT. It would seem to the Court that such an approach ought to be considered for the inclusion in the Heparin protocols of all major public hospitals.

7.4. The remaining matter as far as the Heparin Protocol is concerned is that Professor Cade has suggested that a copy of the protocol always be attached to the patient's Heparin charts for the ready reference of the clinical staff. I see no reason why such a measure should not be considered desirable if not mandatory.

7.5. There was evidence that some of the delay in the eventual administration of Ms Krivitch's blood transfusion was caused by the need to cross-match her blood, in other words to identify the appropriate blood group for the purposes of transfusion. Mr Ferguson suggested that a more efficient way of going about things would be to cross-match a patient's blood from the outset and at a time before anticoagulation issues, or the need for a transfusion, ever materialise.

7.6. Finally this. This case has in my view illustrated the undesirability of leaving what can only be described as life and death tasks to unsupervised junior medical practitioners some of whom must have been fresh out of medical school. While it is difficult to craft any specific and universally applicable recommendation that would prevent the recurrence of such a serious deficiency in the level of expertise in a ward of a major public hospital, I do intend to make the general recommendation that is reproduced below. As well, the Court expresses the hope and expectation that a set of circumstances such as those that existed here will not be repeated. I direct that observation to the attention of the Minister for Health.

[22] Exhibit C11b

7.7. I make the following recommendations:

a) That the Minister for Health draw these findings to the attention of the Chief Executive Officers of all public hospitals in South Australia with a view to appropriate consideration being given to the observations made by Professor Cade as discussed in paragraphs 7.2, 7.3 and 7.4 herein as to the need to amend the hospitals' Heparin Protocols and as to the desirability of the practice of including a copy of the protocol in the patient's infusion chart;

b) That the Minister for Health draw to the attention of the Chief Executive Officers of all public hospitals the desirability of identifying in advance of the commencement of anticoagulation therapy, the relevant blood grouping of the patient so as to facilitate the more timely delivery of a blood transfusion should the necessity for the same arise;

c) That the Minister for Health draw my findings in respect of the necessity to monitor haemoglobin levels in circumstances such as those that pertained to Ms Krivitch, to the attention of the relevant person at all medical schools in South Australia;

d) That the Minister for Health take the necessary steps to ensure that wards in all public hospitals are at all times appropriately staffed.

Key Words: Anticoagulation Therapy, Heparin Protocol, Haemoglobin Monitoring.

In witness whereof the said Coroner has hereunto set and subscribed his hand and Seal the 4th day of February, 2010.

Deputy State Coroner