CORONERS COURT OF SOUTH AUSTRALIA

INQUEST INTO THE DEATH OF COLIN ANTHONY ROSENZWEIG

[2026] SACC 27

Inquest Findings of her Honour Deputy State Coroner Roper

14 July 2026

CORONIAL INQUEST

Examination of the cause and circumstances of the death of a man with a mechanical aortic valve who experienced a cerebrovascular accident while his anticoagulation was subtherapeutic following a colonoscopy. The inquest explored the management of his anticoagulation during the perioperative period, and whether his death could have been prevented.

Held:

1. Colin Anthony Rosenzweig, aged 61 years of Vine Vale, died at the Royal Adelaide Hospital on 14 November 2020 as a result of bronchopneumonia complicating cerebrovascular accident due to cardioembolism on a background of mechanical aortic valve.

2. Circumstances of death as set out in these findings.

Recommendations made.

Counsel Assisting: MS R SCHELL

Family: MRS D ROSENZWEIG
Counsel: MR R KATSAMBIS - Solicitor: LINDBLOM LAWYERS

Witness: DR P WORLEY
Counsel: MR A HARRIS - Solicitor: WALLMANS LAWYERS

Witness: DR T VAUGHAN
Counsel: MR M TILLEY - Solicitor: HWLE LAWYERS

Hearing Date/s: 18/11/2025-20/11/2025, 25/11/2025, 27/11/2025, 15/12/2025

Inquest No: 36/2025 **File No/s: 2329/2020**

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1 Mr Colin Anthony Rosenzweig was 61 years old when he died on 14 November 2020 at the Royal Adelaide Hospital following a stroke. Ten days earlier, he had undergone a colonoscopy and polypectomy. This necessitated temporary cessation of his anticoagulant medication, warfarin, which had been prescribed following insertion of a mechanical aortic valve in 2014. Ceasing warfarin increased Mr Rosenzweig's risk of a cardioembolic stroke, which subsequently occurred and resulted in his death.

2 The primary issue explored during the inquest was whether Mr Rosenzweig's anticoagulation regimen was appropriately managed perioperatively, and whether his death could have been prevented.

Background

3 Mr Rosenzweig is survived by his wife, Mrs Debra Rosenzweig, their three children, and two grandchildren. Mr Rosenzweig had worked as a specification consultant for Dulux until 2014, when he underwent cardiac surgery and experienced a postoperative stroke which prevented him from returning to work.

4 Mrs Rosenzweig and members of the Rosenzweig family attended each day of the inquest and were represented by counsel, Mr Katsambis. Mr Rosenzweig was plainly a much-loved husband, father and grandfather and it is clear he is dearly missed.

Circumstances leading to the death of Mr Rosenzweig

5 On the evening of 11 November 2020, Mrs Rosenzweig heard her husband collapse and found him on the floor, bleeding from his head and shaking as if having a seizure. She called for an ambulance.

6 Ambulance officers arrived promptly and examined Mr Rosenzweig. They observed weakness on the left side of his body, and a right-sided facial droop. Mr Rosenzweig was conveyed to Angaston Hospital where coagulation testing was performed. In patients on warfarin, this involves a blood test which measures how long the blood takes to clot, expressed as the International Normalised Ratio (INR). Mr Rosenzweig's target INR was between 2.5 and 3.5. When he arrived at the Angaston Hospital however, his INR was 1.3, indicating that he was not anticoagulated. This was an unexpected result as Mr Rosenzweig was believed to have recommenced warfarin therapy on 5 November 2020, the day following his colonoscopy and polypectomy.

7 Mr Rosenzweig was retrieved from the Angaston Hospital by MedSTAR and airlifted to the Royal Adelaide Hospital, where he arrived at about 2:07 am on 12 November 2020. His INR was recorded as 1.4 at that time.

8 A CT angiogram was performed which revealed occlusion of the distal right internal carotid artery and middle cerebral artery. In other words, Mr Rosenzweig had suffered a severe stroke. It was thought that the cause of the stroke was cardioembolic, meaning that a blood clot had formed on the heart and travelled to the brain, preventing adequate blood flow. This is known as a cardioembolism, or thromboembolic event.

9 As a result of the stroke, there was minimal salvageable brain tissue, and it was determined that active treatment would be futile. Mr Rosenzweig's condition deteriorated, and he was transitioned to end of life care. He passed away at 5:35 pm on 14 November 2020 in the presence of his family.

10 Mr Rosenzweig had undergone a colonoscopy and polypectomy on 4 November 2020, performed by general surgeon Dr Philip Worley. While the procedure itself was uneventful, it required Mr Rosenzweig to cease taking warfarin four days prior to surgery, with a plan to recommence at his usual dose the day after surgery.

11 Mr Rosenzweig had a follow-up appointment scheduled with Dr Worley for 13 November 2020. There was no monitoring of his coagulation planned during the intervening period of nine days. The inquest examined whether that was an appropriate course of action, and whether a more assertive postoperative anticoagulation regime could have prevented the death of Mr Rosenzweig. The failure to monitor Mr Rosenzweig's INR following surgery was the major concern expressed by Mrs Rosenzweig following the death of her husband. It was also the primary focus of the inquest.

Evidence at inquest

12 The following witnesses were called to give evidence at the inquest:

- Dr Philip Worley – a general surgeon who performed a diagnostic colonoscopy on Mr Rosenzweig on 15 July 2020, followed by a second colonoscopy with polypectomy on 4 November 2020. Dr Worley divides his time equally between public and private practice, and currently holds the position of Senior Staff Specialist, General Surgery, at Flinders Medical Centre and Noarlunga Hospital.
- Dr Daniel Cehic – Mr Rosenzweig's treating cardiologist since 2008. He currently holds the position of Chief Medical Officer of Cardiology for Advara HeartCare. During his early career, Dr Cehic spent approximately seven years at the Calvary Hospital managing the postoperative care of patients who had undergone cardiac surgery. His last consultation with Mr Rosenzweig was on 2 December 2019.
- Dr Martin Sterck – a general practitioner who consulted with Mr Rosenzweig at the Angaston Medical Centre on several occasions throughout 2020. Dr Sterck and other practitioners at the Angaston Medical Centre managed Mr Rosenzweig's anticoagulation for several years. Dr Sterck's last consultation with Mr Rosenzweig was on 28 October 2020.
- Dr Brian Norcock OAM – a rural general practitioner who has been practising in Naracoorte since 1983. He was awarded a Medal of the Order of Australia in 2015 for his service to rural medicine and to the community of Naracoorte. Dr Norcock was commissioned by the Court to provide independent expert evidence in relation to the perioperative management of Mr Rosenzweig's anticoagulation, from the perspective of a general practitioner.
- Professor Robert Padbury – a general surgeon with a doctorate in neuroscience. He held the position of Clinical Director of Surgery and Perioperative Medicine at the Flinders Medical Centre from 2001 to 2024. Presently he is the Executive Director of Continuous Improvement at the Southern Adelaide Local Health Network. Professor Padbury was commissioned by Dr Worley's legal representatives to provide an expert report in relation to Dr Worley's management

of Mr Rosenzweig's perioperative anticoagulation. Professor Padbury is an experienced expert witness in the coronial jurisdiction.

- Professor John Cade – an intensive care specialist and emeritus consultant within the Intensive Care Unit of the Royal Melbourne Hospital. He is the former Director of Intensive Care at the Royal Melbourne Hospital, a position he held for over 30 years. Professor Cade was commissioned by the Court to provide independent expert evidence in relation to the perioperative management of Mr Rosenzweig. Professor Cade is also an experienced expert witness in the coronial jurisdiction.

13 Expert reports were received from the following witnesses who did not give oral evidence during the inquest:

- Dr David Walsh – a general surgeon. I admitted Dr Walsh's report only as part of the material considered by and commented upon by Professor Cade and Professor Padbury. I do not treat the report of Dr Walsh as expert opinion evidence upon which I can independently rely, as his opinions were not subject to cross-examination.
- Dr Peter Joyner OAM – an experienced rural general practitioner and expert witness in the coronial jurisdiction. Dr Joyner was unavailable to attend court to provide oral evidence. Accordingly, Dr Norcock was commissioned to provide a report in his stead. I received Dr Joyner's report into evidence only insofar as it formed part of the material provided to and commented upon by other witnesses, particularly Dr Norcock. I do not treat the report of Dr Joyner as expert opinion evidence upon which I can independently rely.

14 Dr Tom Vaughan, the anaesthetist for Mr Rosenzweig's two colonoscopies, was initially summoned to attend court. However, he was released from his summons following the evidence of Dr Worley, at which time it became apparent that there was no dispute in relation to the content of their conversations regarding Mr Rosenzweig's management.

15 Numerous documents were also received into evidence, including journal articles published in the academic literature pertaining to the perioperative management of anticoagulation. I have considered all the evidence before me, including evidence not specifically referred to. I will refer only to the aspects of the evidence necessary to explain the findings reached and in the interests of narrative clarity.

Standard of proof and hindsight bias

16 The civil standard of proof, the balance of probabilities, applies to the making of coronial findings. If findings which imply or express criticism of individuals are considered, I shall not make such a finding unless the evidence leads me to a comfortable level of satisfaction that the finding should be made. In this regard, I am guided by the principles enunciated in *Briginshaw v Briginshaw*.[1] The effect of this and similar authorities is that coroners should exercise caution when considering adverse findings against, or comments about, individuals or entities. Proof of facts underpinning a finding that would, or may, have an extremely deleterious effect on a party's character, reputation or employment prospects

[1] (1938) 60 CLR 336

demands a weight of evidence commensurate with the gravity of the facts sought to be proved.

17 I have also remained cognisant of the potential intrusion of hindsight bias. A description of 'hindsight bias' is given in *The Australasian Coroner's Manual*, namely:

> The tendency after the event to assume that events are more predictable or foreseeable than they really were. What is clear in hindsight is rarely as clear before the fact. If it were, there would be far fewer mistakes made. It is an obvious point, but one that nonetheless bears repeating, particularly when coroners are considering assigning blame or making adverse comments that might damage a person's reputation…
>
> Hindsight, of course, is a very useful tool for learning lessons from an unfortunate event. It is not useful for understanding how the involved people comprehended the situation as it developed. The distinction needs to be understood and rigorously applied.[2]

18 Throughout my consideration of the evidence, I have remained mindful of this risk and of the need to assess events by reference to information available at the time.

What was the cause of Mr Rosenzweig's death?

19 An examination of Mr Rosenzweig's longitudinal medical history was performed by Dr Erin O'Connor, a medical practitioner experienced in providing opinions as to cause of death. The resulting pathology review provided a suggested cause of death as cerebral infarction secondary to middle cerebral artery and internal carotid artery thrombosis. Dr O'Connor observed that although the cause of death was apparent due to the right middle cerebral artery stroke, it was unclear whether the source of the thrombus was embolic. A post-mortem examination with neuropathology was recommended and subsequently directed.

20 The post-mortem examination was performed by senior forensic pathologist, Dr Karen Heath. She found patchy but widespread bronchopneumonia, which she opined was likely agonal and the product of Mr Rosenzweig's decreased conscious state due to his stroke. I agree and find that bronchopneumonia was a terminal event caused by the stroke.

21 Dr Heath referred Mr Rosenzweig's brain for specialist neuropathological examination. This examination was performed by Professor Peter Blumbergs, who produced a report. Professor Blumbergs found evidence of a previous haemorrhagic infarct in the left basal ganglia in the middle cerebral artery territory. He considered that this was an old infarct which had been present for years. The medical records confirm that Mr Rosenzweig suffered a left middle cerebral artery ischaemic embolic stroke on 18 December 2014. I am satisfied that the old infarct observed by Professor Blumbergs was the result of Mr Rosenzweig's embolic stroke in 2014.

22 Professor Blumbergs also found evidence of a recent haemorrhagic infarct in the right middle cerebral artery vascular territory involving head of caudate, internal capsule and lateral putamen. He opined that the features were consistent with cerebral embolism although no emboli were demonstrated in the material examined. He did not propose any alternative cause of the infarct or express a view regarding the source of the emboli.

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[2] Dillon H & Hadley M (2015), p10

23 Dr Heath considered the report of Professor Blumbergs, in combination with her own findings, and suggested that the cause of death was bronchopneumonia complicating cerebrovascular accident (stroke). Dr Heath's conclusion is well-supported and I accept it.

24 However, it is necessary to determine, insofar as possible, the cause of the stroke. The records from the Royal Adelaide Hospital assist in this regard. The discharge summary included the following opinion:

> His final diagnosis was a right severe ischaemic stroke affecting the M1 and carotid terminus location, likely cardioembolic in the setting of subtherapeutic INR on warfarin.[3]

25 In essence, the clinical opinion was that Mr Rosenzweig suffered an ischaemic stroke caused by a blood clot that most likely travelled from his heart to his brain while his anticoagulation was below the therapeutic range (a thromboembolic event). This was also the view of Professor Cade, who considered that the embolic stroke was likely related to the cessation of anticoagulation.

26 I note that a cardioembolic event was the precise risk against which anticoagulation with warfarin was intended to prevent, and there is no evidence identifying a more probable embolic source.

27 I am satisfied, on the balance of probabilities, that the cause of Mr Rosenzweig's death was: bronchopneumonia complicating cerebrovascular accident due to cardioembolism on a background of mechanical aortic valve.

Why did Mr Rosenzweig experience a thromboembolic event?

28 On 18 December 2014, Mr Rosenzweig underwent a mechanical aortic valve replacement due to severe aortic stenosis.[4] His aortic valve was excised and replaced with an On-X mechanical valve. This increased his risk of a thromboembolic event.

29 Dr Cehic provided a clear explanation as to why this risk was increased, namely that there is a tendency for blood to clot on mechanical valves as they are a foreign body. If a blood clot (thrombus) forms, it may dislodge and travel through the blood stream as an embolus. If the embolus lodges in a vessel in the brain, it is referred to as a cerebral embolus and may block blood flow, causing an ischaemic stroke.

30 Accordingly, anticoagulation is prescribed to thin the blood and thereby prevent blood from clotting on the valve, reducing the thromboembolic risk. Warfarin is the only medication effective in reducing the thromboembolic risk for patients with a mechanical aortic valve. Mr Rosenzweig was required to cease warfarin therapy prior to his second colonoscopy, increasing his risk of thromboembolism.

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[3] Exhibit C9, p128

[4] A condition in which the aortic valve is significantly narrowed and stiffened, restricting blood flow from the heart to the body and increasing the workload on the heart

What was Mr Rosenzweig's baseline risk of thromboembolism prior to ceasing warfarin therapy?

31 There was consensus among the expert witnesses that anticoagulation reduces, but does not eliminate, the risk of an embolic stroke in patients with a mechanical aortic valve. Professor Cade opined that the risk is generally regarded to be less than 1% a year if a patient is adequately anticoagulated. Professor Padbury agreed. This statistic is based upon published data of relevant clinical trials.

32 There are factors which are known to increase the thromboembolic risk above 1% per annum, including previous thromboembolism.

33 Mr Rosenzweig had experienced a left middle cerebral artery ischaemic embolic stroke on 18 December 2014 within six hours of his cardiac surgery, which required emergency clot retrieval surgery. It was therefore necessary to consider whether this previous stroke increased his baseline thromboembolic risk.

34 Dr Worley did not consider that it did. He opined that due to the temporal relationship between the stroke and the cardiac bypass procedure, it was reasonable to assume that Mr Rosenzweig did not have an increased predisposition for stroke.

35 Dr Cehic considered it to be debatable as to whether the previous stroke should be regarded as a risk factor. He explained that a cautious approach could be taken, and you could assume that Mr Rosenzweig was a 'bit of a higher risk'.[5] However, as the previous stroke occurred in the context of a major operation, Dr Cehic did not consider that Mr Rosenzweig had a genetic predisposition towards clotting.

36 Professor Padbury was unable to locate any literature which would support a periprocedural stroke presenting as an increased risk factor.

37 Professor Cade opined that Mr Rosenzweig's postoperative stroke in the setting of aortic surgery would have little bearing on the future thromboembolic risk.

38 Accordingly, I accept that it was reasonable for Dr Worley to proceed on the basis that the prior stroke in 2014 did not increase Mr Rosenzweig's risk of a subsequent stroke. I am satisfied that Mr Rosenzweig's thromboembolic risk was about 1% a year while adequately anticoagulated.

Was Mr Rosenzweig adequately anticoagulated, prior to ceasing warfarin for the second colonoscopy?

39 Mr Rosenzweig's anticoagulation prior to ceasing warfarin was managed by the Angaston Medical Centre, where he had been a patient since May 2017 and attended regularly, usually consulting with Dr Ben Baker.

40 Mr Rosenzweig attended the practice for INR testing monthly until January 2018, when a decision was made that six-weekly testing would suffice. More frequent testing and dose adjustments were required in April and June 2018 due to an unexplained increase in Mr Rosenzweig's INR.

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[5] T204

41 On 28 June 2018, Dr Baker noted that Mr Rosenzweig had missed one warfarin dose due to an upcoming operation on his knee. Dr Baker recorded that they had discussed the risk of stroke with a mechanical aortic valve if perioperative anticoagulation was not managed properly, and that Mr Rosenzweig would call if he had any questions or uncertainty about his perioperative anticoagulation regimen.

42 On 17 July 2018, Dr Peter Lewis operated upon Mr Rosenzweig's right knee. That same day, he wrote to Dr Baker to inform him of the results of the surgery, and to advise that he would ask for some assistance managing Mr Rosenzweig's anticoagulation while in hospital. Dr Lewis provided Dr Baker with a further update on 31 August 2018, advising that Mr Rosenzweig seemed to have come through the surgery quite well, despite his stroke concerns and preoperative issues.

43 From August 2018, Mr Rosenzweig's INR results and dosages were recorded in a booklet. On 11 September 2018, Mr Rosenzweig's INR had risen to 4.3. Dr Baker held Mr Rosenzweig's warfarin and measured again the following day, when the result had reduced to 3.6. Further INR tests were performed on 19 and 27 September 2018, until Mr Rosenzweig's INR stabilised. Monthly testing resumed until February 2019, when more regular testing was considered necessary due to fluctuating INR results. From August 2019, Mr Rosenzweig's INR stabilised, and he resumed his six-weekly reviews.

44 From 12 February 2020 Mr Rosenzweig consulted with Dr Sterck at the Angaston Medical Centre, who continued to manage his six-weekly reviews. Mr Rosenzweig's INR remained within his therapeutic range up until 24 June 2020, when it climbed to 3.7. Dr Sterck instructed Mr Rosenzweig to decrease his warfarin dose.

45 On 5 August 2020, Mr Rosenzweig's INR result fell to 1.9. Dr Sterck explained during his evidence that he did not reach any conclusion about why this occurred, as dramatic changes in INR results without an obvious cause are not uncommon. He instructed Mr Rosenzweig to increase his warfarin dose and return for a repeat INR. Following the increased dose, Mr Rosenzweig's INR returned to, and remained within, his therapeutic limits prior to ceasing anticoagulation in preparation for the November 2020 colonoscopy.

46 Dr Norcock opined that the management of Mr Rosenzweig's anticoagulation by the Angaston Medical Centre was exemplary, and a demonstration of how well warfarin care can be managed in a general practice setting. He observed that Mr Rosenzweig's INR was mostly in the therapeutic range, and when it was not, adjustments were made and follow-up occurred.

47 Dr Cehic described the regulation of warfarin as 'a nightmare', as a patient's response to it can be unpredictable. He considered this to be a reason why more frequent INR monitoring should be done. Informed decisions could then be made about what action, if any, should be taken in relation to a subtherapeutic INR. This was plainly the approach taken by the Angaston Medical Centre.

48 I find that Mr Rosenzweig was adequately anticoagulated prior to ceasing warfarin. Therefore, his annual risk of stroke at that time was in the order of 1%. I agree with Dr Norcock that the anticoagulation management by the Angaston Medical Centre was exemplary.

What was Mr Rosenzweig's thromboembolic risk following cessation of warfarin in preparation for surgery?

49 As set out above, the thromboembolic risk in mechanical aortic valve recipients adequately anticoagulated is in the order of 1% a year. The thromboembolic risk in mechanical aortic valve recipients who are not anticoagulated or are subtherapeutic is higher. The risk increases the longer a patient remains subtherapeutic and the further the INR falls below the therapeutic range.[6] Quantification of the increased risk of thromboembolism also depends upon factors individual to the patient.

50 In Mr Rosenzweig's case there was no dispute that the second colonoscopy and polypectomy were medically necessary, and that it would be unsafe for those procedures to be undertaken while he was anticoagulated. The decision to undergo these procedures therefore required Mr Rosenzweig to accept an increased risk of thromboembolism for a period of time. The issue is the magnitude and duration of that risk, and whether the risk could have been ameliorated.

51 There are established clinical guidelines available to practitioners to assist them to assess a patient's thromboembolic risk and their bleeding risk, and to balance these competing risks.

52 Professor Cade explained that the management of perioperative anticoagulation is a well-documented area of clinical practice, with published and widely available guidelines having been in circulation for over 40 years.

53 Several guidelines were referred to during the inquest, including:

- Guidelines on periprocedural management of anticoagulant and antiplatelet agents (March 2025), published by the Clinical Excellence Commission of New South Wales
- Guidelines on perioperative management of anticoagulant and antiplatelet agents (December 2018), published by the Clinical Excellence Commission of New South Wales
- Guideline for the management of patients with valvular heart disease (June 2017), published by the American Heart Association/American College of Cardiology
- Guidelines on non-cardiac surgery: cardiovascular assessment and management (August 2014), published by the European Society of Cardiology and the European Society of Anaesthesiology

54 Professor Cade explained the evidentiary basis for the various guidelines as follows:

> So, the guidelines are developed in a very structured process, by examining the literature, putting all the studies together, assessing their weight as to how valuable they are, statistically, and whether there's bias and their numbers and particular values. And then pooling them and then also taking into account patient values, practicability, cost, and not just the statistical science. So, it's a consensus building process of the value of a particular process or procedure.

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[6] T208, T397

55 The Court also received into evidence various published journal articles which underpin the guidelines, including, but not limited to:

- Douketis JD et al. (2015) 'Perioperative Bridging Anticoagulation in Patients with Atrial Fibrillation'[7] (the Bridge trial)
- Kovacs MJ et al. (2021) 'Postoperative low molecular weight heparin bridging treatment for patients at high risk of arterial thromboembolism (PERIOP2): double blind randomised controlled trial'[8] (the PERIOP2 trial)
- Tan CW et al. (2019) 'How to bridge? Management of anticoagulation in patients with mechanical heart valves undergoing noncardiac surgical procedures'[9] (How to Bridge)
- Bungard TJ et al. (2017) 'A randomized trial of restarting warfarin at maintenance versus loading doses following an elective procedure'[10] (the Bungard study)
- Shah S et al. (2013) 'Perioperative Management of Vitamin K Antagonists and Direct Oral Anticoagulants'[11]

56 Dr Worley explained that, while there are multiple guidelines, they all involve working out whether the patient is low, moderate or high risk of bleeding and thromboembolism respectively. Dr Worley considered Mr Rosenzweig to be a moderate to high risk of bleeding and a low, at most moderate, risk of thromboembolism.[12] He did not identify a specific guideline that he utilised to categorise the risks and he was not familiar with either of the Clinical Excellence Commission guidelines.

57 Professor Cade opined that Mr Rosenzweig was properly categorised as being at moderate risk of thromboembolism without anticoagulation, corresponding to an estimated annual risk of between 2% and 10%. He estimated that Mr Rosenzweig's risk would likely fall in the middle, at 6% per year. Professor Cade agreed with Dr Worley that the bleeding risk was moderate to high.

58 Professor Padbury considered it to be unclear as to whether Mr Rosenzweig would fall into the low or moderate thromboembolic risk category. However, he referred to literature which estimated the thromboembolic risk to be 5.2% annually for patients with mechanical aortic valves who had ceased their anticoagulation. He explained that this

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[7] Douketis JD, Spyropoulos AC, Kaatz S, Becker RC, Caprini JA, Dunn AS, Garcia DA, Jacobson A, Jaffer AK, Kong DF, Schulman S, Turpie AG, Hasselblad V, Ortel TL, & BRIDGE Investigators (2015) 'Perioperative Bridging Anticoagulation in Patients with Atrial Fibrillation', *The New England Journal of Medicine*, 373(9), 823–833 https://doi.org/10.1056/NEJMoa1501035

[8] Kovacs MJ, Wells PS, Anderson DR, Lazo-Langner A, Kearon C, Bates SM, Blostein M, Kahn SR, Schulman S, Sabri E, Solymoss S, Ramsay T, Yeo E, Rodger MA, & PERIOP2 Investigators (2021) 'Postoperative low molecular weight heparin bridging treatment for patients at high risk of arterial thromboembolism (PERIOP2): double blind randomised controlled trial', *BMJ (Clinical research ed.)*, *373*, n1205, https://doi.org/10.1136/bmj.n1205

[9] Tan CW, Wall M, Rosengart TK, & Ghanta RK (2019) 'How to bridge? Management of anticoagulation in patients with mechanical heart valves undergoing noncardiac surgical procedures', *The Journal of Thoracic and Cardiovascular Surgery*, *158*(1), 200–203, https://doi.org/10.1016/j.jtcvs.2018.06.089

[10] Bungard TJ, Mutch J, Ritchie B (2017) 'A randomized trial of restarting warfarin at maintenance versus loading doses following an elective procedure', *J Thromb Thrombolysis,* Nov;44(4):507-515. doi: 10.1007/s11239-017-1553-6

[11] Shah S, Nayfeh T, Hasan B, Urtecho M, Firwana M, Saadi S, Abd-Rabu R, Nanaa A, Flynn DN, Rajjoub NS, Hazem W, Seisa MO, Hassett LC, Spyropoulos AC, Douketis JD, Murad MH (2013) 'Perioperative Management of Vitamin K Antagonists and Direct Oral Anticoagulants: A Systematic Review and Meta-analysis', *Chest,* May;163(5):1245-1257 doi: 10.1016/j.chest.2022.11.032

[12] Exhibit C14, p6 at [11]; T54

means that for every day of low or no anticoagulation, the chance of suffering a thromboembolic event is somewhere between 1:5000 and 1:7000.

59 As Professor Cade also calculated the daily risk as 1:5000, I do not consider there to be any substantial divergence of opinion between his opinion and Professor Padbury's opinion on this topic. In any event, the recommended management for a patient with a moderate to high bleeding risk is the same regardless of whether the thromboembolic risk is assessed as medium or low.

60 Accordingly, I accept the evidence of Professors Cade and Padbury that the risk of Mr Rosenzweig experiencing a thromboembolic event in the setting of subtherapeutic anticoagulation was approximately 6% per year, acknowledging that this figure is approximate but represents the best available evidence.

Were there additional factors which increased the thromboembolic risk above 6% per annum?

61 Mr Katsambis submitted that the assessment of Mr Rosenzweig's thromboembolic risk should have taken into account his previous difficulty returning to therapeutic levels of anticoagulation following a partial knee replacement in July 2018. Following that procedure, Mr Rosenzweig received Clexane in addition to warfarin until he returned to therapeutic anticoagulation.[13]

62 Mr Katsambis argued that this was a key risk factor which was not considered by Dr Worley.

63 Counsel for Dr Worley, Mr Harris, submitted that there was no expert evidence to support a finding that Mr Rosenzweig's previous difficulty returning to therapeutic anticoagulation was a risk factor which increased his chances of a thromboembolic event. This was not a variable considered in the academic literature.

64 While I accept the logic of the submission that past events may be predictive of future events, I am not satisfied that such an inference can be properly drawn in this case. The cause of Mr Rosenzweig's difficulty returning to therapeutic anticoagulation in 2018 is unknown. It may have been unique to the procedure, as was the case with the periprocedural stroke in 2014, or it may have been related to the postoperative management of his anticoagulation.

65 I do not accept that Dr Worley ought to have considered Mr Rosenzweig's thromboembolic risk to be elevated above low to moderate due to his previous difficulty returning to therapeutic anticoagulation.

What was Dr Worley's management plan regarding Mr Rosenzweig's perioperative anticoagulation?

66 Dr Worley told the Court that his initial perioperative management plan for Mr Rosenzweig was as follows:

1. Cease taking warfarin four days prior to the procedure;
2. Recommence warfarin at the usual dose the day after surgery;

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[13] Exhibit C7, p271

3. Advise Mr Rosenzweig to have his INR checked on the day of his postoperative appointment, scheduled for 13 November 2020, and then return his anticoagulation management to his general practitioner.

67 It was common ground at the inquest that Dr Worley advised Mr Rosenzweig to cease warfarin four days before the second colonoscopy and that this advice was clinically appropriate. It was also agreed that Dr Worley's instruction to recommence warfarin at the usual dose the day after surgery was appropriate.[14] The contentious aspect of the plan was the postoperative monitoring.

68 Dr Worley's documentation of his management plan was limited to the Day Surgery Discharge Form on which he had handwritten the words 'may recommence warfarin 5/11', and an information form sent to Mr Rosenzweig dated 7 September 2020 that included the following information in bold type:

> Dr Worley has recommended that you take 100mgs aspirin during the 4 days you have ceased your warfarin.

69 Dr Cehic initially queried whether this plan provided Mr Rosenzweig with a sufficient level of instruction, given that there was no reference to the dosage of warfarin to recommence on 5 November 2020, or of who would be monitoring his INR to ensure return to therapeutic anticoagulation.

70 However, in oral evidence, and in his affidavit, Dr Worley explained that these documents were supplemented by comprehensive oral instructions to Mr Rosenzweig, which included information regarding the dosage. His evidence on this topic was unchallenged.

71 Debra Rosenzweig provided an affidavit to the Court. She stated that her husband stopped taking warfarin four days prior to his procedure. This was in accordance with the instructions Dr Worley told the Court that he provided. Mrs Rosenzweig also stated that her husband was told to take aspirin while off warfarin. She presumed that Dr Worley had instructed him to take the aspirin.

72 I find that Dr Worley's advice to cease warfarin four days prior to the second colonoscopy and to recommence at the usual dose the following day was adequately conveyed to Mr Rosenzweig. I am satisfied that the plan for cessation and resumption of warfarin was explained to Mr Rosenzweig and that he understood it.

73 However, there was limited contemporaneous documentation of this plan, and it was not communicated to Mr Rosenzweig's general practice, his cardiologist, or his wife. The plan was also silent as to monitoring Mr Rosenzweig's return to therapeutic anticoagulation, aside from the notation 'may recommence warfarin 5/11'.

74 There was no evidence that the monitoring of Mr Rosenzweig's return to therapeutic anticoagulation formed part of Dr Worley's discussions with Mr Rosenzweig at any stage. Mrs Rosenzweig stated in her affidavit that there was no appointment that she was aware of for her husband to attend the Angaston Medical Centre to have his INR checked. She

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[14] I note that the Bungard study found that warfarin resumption with loading doses shortened the time to achieve a therapeutic INR by a median of 1.2 days. However, the Clinical Excellence Commission Guidelines recommend recommencement of warfarin at the usual dose. There is no evidence justifying a criticism of Dr Worley for advising recommencement at the usual dose. See also T308 (Padbury).

believed that she would have been aware of an appointment as they shared use of one vehicle. This is consistent with the evidence of Dr Worley that he intended to tell Mr Rosenzweig at his postoperative appointment that he should have an INR test that day.

75 Dr Worley considered that Mr Rosenzweig, as his patient, was entitled to be at the centre of decision-making in relation to his treatment.[15] I agree. However, it does not appear that Mr Rosenzweig was consulted in relation to the monitoring of his return to therapeutic anticoagulation, or informed regarding the detail of the plan. Mrs Rosenzweig stated that she was not advised as to next steps.

76 It may be that this aspect of the plan was not at the forefront of Dr Worley's mind at the time of his consultations with Mr Rosenzweig, or when he reviewed him following the second colonoscopy.

77 However, it does illustrate the problem described by Professor Cade as 'bundling apples and oranges'. Professor Cade explained that in his view, bundling up the INR testing with the other duties of the proceduralist was not the best way to deal with the management of Mr Rosenzweig's return to anticoagulation. While an appointment at the nine-day mark was perfectly appropriate for the other postoperative issues, it was 'probably a little bit late' to test the INR. I will return to this issue later in these findings.

78 Further, Mr Rosenzweig was known to Dr Worley to be particularly concerned about ceasing warfarin due to his prior experience. The decision to endure two colonoscopies rather than one was made to avoid interrupting his warfarin therapy. In my view, Mr Rosenzweig's monitoring plan should have taken into account his heightened level of concern about returning to therapeutic anticoagulation. Unfortunately, however, the monitoring plan was, in practical terms, absent; it amounted to an unrecorded intention regarding a future INR test date. There was no documented schedule for INR testing, and no communication of the plan to the Angaston General Practice.

Aspirin

79 Dr Worley did not consider the advice to replace warfarin with aspirin to be part of his initial management plan. He explained that Mr Rosenzweig telephoned his rooms on 26 August 2020 and spoke with his receptionist, Annette Lawrence. Ms Lawrence provided an affidavit in which she annexed her note of this conversation, and Dr Worley's reply. Her note read:

> … He's asked if he needs to replace the warfarin with anything else ?aspirin.

80 Dr Worley replied:

> Yes, he could go onto 100 mg aspirin daily whilst he is off warfarin.

81 While Dr Worley's language was permissive rather than prescriptive, this was also the case with the instruction he wrote on the discharge form 'may recommence warfarin 5/11'. Given that the advice regarding aspirin was repeated in the information form dated

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[15] T55

7 September 2020, I consider that it was part of Dr Worley's management plan, irrespective of the source of the initial suggestion.

82 There is a note contained within the Royal Adelaide Hospital records which suggests that Dr Worley prescribed aspirin as a form of 'bridging' therapy.[16] This note, written by an intern of the Acute Stroke Unit, is a record of collateral information apparently provided by Dr Worley via telephone following Mr Rosenzweig's fatal stroke. The intern recorded that Dr Worley stated that he had discussed bridging therapy with anaesthetist Dr Tom Vaughan who advised that, for aortic valves, aspirin was the preferred 'bridging agent' if bridging was felt necessary and that enoxaparin and heparin would not be required.

83 Dr Worley disputed the accuracy of this note in several respects and stated that he did not discuss the use of aspirin with Dr Vaughan, and he did not consider it to be an effective bridging medication. Dr Worley's evidence on this topic was unchallenged. All counsel submitted that the note contained numerous inaccuracies, and I accept that it cannot be considered a reliable record of the conversation.

84 I find that Dr Worley did not discuss the use of aspirin with Dr Vaughan. Aspirin is not an effective bridging medication. It is improbable that both Dr Worley and Dr Vaughan did not appreciate that fact. I also find that Dr Worley did not prescribe aspirin as a bridging medication.

85 There is no clear explanation, however, for why Dr Worley responded to the query from Mr Rosenzweig in the way that he did. Mr Rosenzweig did not call Dr Worley's rooms seeking pain relief. Rather, he queried whether he needed to *replace* the warfarin with anything else. The response was yes; he could take aspirin. It may be that Dr Worley suggested aspirin as a placebo. In any event, it is unnecessary for me to make a finding on this issue as I accept the evidence of Professor Cade that the use of aspirin did not contribute to the adverse outcome for Mr Rosenzweig.

To bridge or not to bridge?

86 As explained by Professor Cade, perioperative management of anticoagulation interruption for an operative procedure comprises three parts: timing of preoperative cessation, bridging regimen, and program for resumption postoperatively. The term 'bridging' is commonly used to refer to the use of low molecular weight heparin, usually Clexane, to 'bridge' the gap between the interruption of warfarin and the resumption of warfarin.[17] This is the definition of bridging that I adopt for the purposes of this finding. The planned use of Clexane after resumption of warfarin will be referred to as 'postoperative bridging'.

87 In relation to the preoperative phase, there was unanimous support for the approach taken by Dr Worley to cease warfarin four days prior to surgery. I find that this decision was appropriate and in accord with best practice.

88 In relation to the second phase, Dr Worley did not prescribe a bridging regimen. He explained that he assessed Mr Rosenzweig's bleeding risk to be moderate to high, and his

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[16] Bridging therapy is the administration of a therapeutic dose of a short-acting anticoagulant, typically low molecular weight heparin, during the interruption of warfarin

[17] T396

thromboembolic risk to be low, at most, moderate. Accordingly, bridging medication was not recommended by him.

89 Professor Cade opined that Dr Worley's decision to not implement a bridging regimen was appropriate in Mr Rosenzweig's circumstances and in accord with best practice.[18] Professor Padbury concurred with this opinion, citing the 2025 Clinical Excellence Commission guidelines. He opined that, considering the results of the PERIOP2 trial, Mr Rosenzweig's outcome could have been exactly the same even if he had been bridged with low molecular weight heparin.

90 Dr Cehic explained that new evidence indicates that bridging does not necessarily reduce the stroke risk for the bridging period and this has influenced a trend away from bridging. Dr Norcock also agreed in his oral evidence that bridging in Mr Rosenzweig's case was not indicated.

91 The current Clinical Excellence Commission guideline recommends bridging therapy only for patients who are assessed to have a high risk of thromboembolism.

92 However, Professor Cade explained that current research data no longer supports bridging therapy, even with high-risk patients. The PERIOP2 trial is the most recent study evaluating the efficacy of postoperative bridging in reducing the rate of arterial thromboembolism. This study was a double-blind, randomised, placebo-controlled trial with a sample size of 1471. The trial participants comprised patients with atrial fibrillation or mechanical heart valves who required warfarin interruption for a surgical procedure. All participants received preoperative bridging anticoagulation and were then randomised to receive either postoperative bridging anticoagulation or placebo. The trial therefore examined the benefit of postoperative bridging in addition to preoperative bridging. No significant reduction in major thromboembolic events was demonstrated with postoperative bridging, suggesting that postoperative bridging is not beneficial in preventing major thromboembolism in the studied population.

93 As acknowledged by the authors of the article, the PERIOP2 trial has limitations. The full sample size was not achieved, the number of patients with mechanical heart valves was relatively low, the rate of thromboembolism in the mechanical heart valve group was very low, and all parties received preprocedural bridging with low weight molecular heparin, in this case dalteparin. These limitations were highlighted by Mr Katsambis, who also noted that postoperatively, loading doses of warfarin were administered where required. Mr Katsambis submitted that the low rates of thromboembolism could be explained by the preprocedural bridging. Professor Cade agreed that this was a possible explanation for the low rate of thromboembolism in the studied population.[19]

94 Professor Padbury considered that despite the limitations of this study, its publication in the highest-ranking medical journal is testament to its scientific quality and clinical significance. While the authors of the article considered their sample size to be a limitation, Professor Padbury noted that there were 300 aortic valve participants in the study. Having had regard to the study limitations, Professor Padbury considered that this trial represented the best available scientific evidence, and that an inference can be drawn

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[18] T382; Exhibit C20(a), p2
[19] T418

that postoperative bridging is not beneficial in preventing major thromboembolism in patients with mechanical heart valves.

95 The results of the PERIOP2 trial were consistent with the results of the Bridge trial. The Bridge trial was a randomised, double-blind, placebo-controlled trial published in a highly regarded medical journal. This study found that bridging with low molecular weight heparin did not significantly reduce the rate of major arterial thromboembolism in patients with atrial fibrillation who had warfarin treatment interrupted for elective surgery. While patients with mechanical heart valves were excluded from this study, I consider that it forms part of the general evidence base in relation to the efficacy of bridging medication.

96 The treating and independent medical practitioners unanimously concluded that the decision not to prescribe bridging medication was appropriate. Notwithstanding that unanimity, Mr Katsambis persevered with his exploration of this issue and an in-depth analysis of the academic literature occurred during his cross-examination of the expert witnesses. During closing submissions, however, it became apparent that Mr Katsambis did not criticise Dr Worley's decision not to initiate bridging therapy while Mr Rosenzweig was off warfarin.

97 Rather, he ultimately submitted that following warfarin resumption, once it ought to have become apparent that the INR was not increasing at the desired rate, then it would have been appropriate to use Clexane to return Mr Rosenzweig to therapeutic anticoagulation.[20] His criticism was therefore directed towards the monitoring of Mr Rosenzweig's warfarin resumption, i.e. the third stage of the perioperative management plan, and what could, or should, have been done in response to a recognised subtherapeutic INR.

98 I accept the unanimous expert evidence that bridging with a low molecular weight heparin, such as Clexane, does not provide a significant reduction in the risk of a thromboembolic event for patients with a mechanical aortic valve with no atrial fibrillation and no prior venous thromboembolism. In the studies considered by the Court, there were examples of participants undergoing bridging therapy who experienced major thromboembolic events,[21] which demonstrates that bridging with low molecular weight heparin does not eliminate this risk.

99 I find that Dr Worley's decision not to institute bridging medication was appropriate in the circumstances and represented an appropriate exercise of clinical judgment, having regard to the competing risks of thromboembolism and bleeding.

Dr Walsh

100 It was suggested by Mr Katsambis that Professor Cade and Professor Padbury were influenced by the report of Dr Walsh and essentially adopted his interpretation of the literature. Neither witness was provided with the opportunity to respond to this suggestion. It is not clear to me why either Professor Padbury or Professor Cade would be influenced by opinions expressed by Dr Walsh to the extent that they would not independently assess the literature and form their own views in accordance with their duty

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[20] T506

[21] In the PERIOP2 trial, the rate of major thromboembolism in the bridging group at 90 days was 1% (8 events in 820 patients), compared with 1.2% in the placebo group (8 events in 650 patients). One participant in the mechanical valve group experienced a major thromboembolism, and that patient was in the bridging group.

to assist the Court. They were content to disagree with each other, respectfully, where there was a divergence of opinion.

101 Professor Cade had not been provided with the report of Dr Walsh prior to providing his initial opinion on 30 July 2025. In that report, Professor Cade wrote that bridging anticoagulation with heparin in patients with mechanical heart valves, but no atrial fibrillation or prior venous thromboembolism, had a non-significant incident rate ratio for clinically relevant major bleeding and transient ischaemic attack (minor stroke) with low or very low certainty of evidence. He opined that, therefore, the recommendation for bridging with heparin was not necessary, and the absence of bridging was in accord with best practice.

102 Professor Cade was subsequently provided with the report of Dr Walsh. Professor Cade adhered to the view expressed in his initial report and agreed with Dr Walsh's view that bridging was not indicated in Mr Rosenzweig's case.

103 It is of note that the report of Dr Walsh did not refer to the Bridge trial or the PERIOP2 trial. In terms of the academic literature, Dr Walsh's primary focus was Tan et al's 'How to Bridge'.[22]

104 In Professor Padbury's report, he attested to compliance with the Uniform Civil Rules 2020, particularly Chapter 7, Part 14. Pursuant to rule 74.5, it is the duty of the expert to assist the Court impartially on matters relevant to the area of expertise of the witnesses. Professor Padbury referred to the PERIOP2 trial in his initial report and did not directly comment upon the report of Dr Walsh, which he had received.

105 I do not accept that either Professor Cade or Professor Padbury were unduly influenced by the views of Dr Walsh. They were impressive witnesses, both in terms of their experience and qualifications, and as expert witnesses more generally.

106 In conclusion on this topic, I accept the consistent expert evidence that Dr Worley's decision not to prescribe bridging medication while Mr Rosenzweig was off warfarin, accorded with best practice. The key issue, as identified by Mrs Rosenzweig at the outset, was the monitoring of Mr Rosenzweig's anticoagulation resumption.

Was Dr Worley's postoperative monitoring of Mr Rosenzweig's anticoagulation appropriate?

Dr Worley's rationale

107 Dr Worley arranged to see Mr Rosenzweig for a postoperative review on 13 November 2020 via telehealth, nine days following surgery. He stated that he would have advised Mr Rosenzweig to have his INR taken on the day of the appointment, emailed a blood test referral form to Mr Rosenzweig or a pathology service that day, and asked that the result be copied to the general practitioner. Dr Worley told the Court that he would have instructed Mr Rosenzweig to ask for an urgent result, which he would expect to see that afternoon.

[22] Exhibit C18d

108 In his affidavit, Dr Worley stated that it was not, and is not, his practice to measure a patient's INR in the immediate postoperative period if they were previously on an established dose of warfarin and they were assessed as low to moderate risk of thromboembolism. He expected that it would take Mr Rosenzweig about a week or more to return to therapeutic level after resuming warfarin.[23]

109 Dr Worley did not concede that he should have checked Mr Rosenzweig's INR at a time prior to 13 November 2020 and considered the difference between checking at 'day 7' (11 November 2020) versus 'day 9' (13 November 2020) to be 'negligible'.[24] He stated that if he thought that this appointment was too late, he would have organised to see Mr Rosenzweig earlier. Moreover, Dr Worley considered there to be risks associated with checking an INR too early. He believed that a lower-than-expected INR might lead to an increased dose of warfarin, which could have the effect of over-anticoagulating the patient and increasing the bleeding risk. He also referred to the cost and inconvenience to the patient of additional INR testing, the risk of the patient losing the referral form, and the undesirability of overburdening the patient with too many things to remember following surgery.

110 Dr Worley believed that his approach to postoperative monitoring of anticoagulation would be consistent with the approach of other surgeons. Professor Padbury agreed and expressed the view that INR testing nine days postoperatively would have been a very common practice among surgeons in 2020.[25]

Opinions regarding Dr Worley's postoperative monitoring plan

111 During the inquest, several witnesses were asked to comment upon Dr Worley's postoperative monitoring plan. These witnesses represented a range of specialties and possessed differing levels of experience in the practical management of warfarin resumption.

Dr Cehic

112 Dr Cehic was asked by Mr Harris for his opinion regarding Dr Worley's plan to arrange for Mr Rosenzweig's INR to be tested on 13 November 2020. It did not appear that Dr Cehic had considered this prior to being asked, as he then undertook a calculation to ascertain how many days that would be. Once he arrived at the figure of eight days following warfarin resumption, he stated that he would be 'a little bit nervous about leaving it for eight days' and that he would 'like to see that INR climbing up'. While he agreed that opinions may differ, he considered eight days to be a long time and stated that he would not leave it that long for one of his patients. Rather, he would check the INR two or three days after warfarin resumption.[26]

113 Dr Cehic explained that while there are no guidelines outlining when testing should occur, he would test two or three days after warfarin resumption to see which direction the INR

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[23] I note that views on this topic varied: Professor Padbury opined the fastest possible time that the INR could have returned to the therapeutic range would be around day four to five based on the Bungard study, with a median of 7.8 days with loading doses and nine days with the usual preprocedural dose: T308. Dr Norcock agreed with the average of seven days and the possibility for a longer return: T242. Prof Cade would have expected a return to therapeutic level for Mr Rosenzweig specifically to be four to five days: T407.

[24] T76

[25] T312

[26] T207

was heading. His rationale was that if you do not check, you do not know, and he considered it preferable to 'make decisions based on the information rather than assumptions'.[27]

Professor Cade

114 Professor Cade considered that Mr Rosenzweig's INR should have been checked two to three days after the procedure, to confirm that the warfarin was starting to take effect. The timing of subsequent INR testing would then depend upon the initial progress of re-anticoagulation. He considered that waiting for nine days was a 'very long time under these sorts of circumstances', and described it as 'unfortunately long'.[28]

Professor Padbury

115 Professor Padbury considered that an INR test on day 9 was at the 'outer end of reasonable'.[29] In oral evidence, he elaborated upon this opinion and stated that testing at day 9 was 'okay but that was really the outer limit'.[30]

Dr Sterck

116 Dr Sterck stated that if he were tasked with the anticoagulation management of a patient who had been discharged from hospital, he would want to see the patient for daily INR checks initially. Dr Sterck also stated that he would ordinarily utilise Clexane up until the INR had returned to the therapeutic range.

Dr Norcock

117 Dr Norcock stated in his oral evidence that he considered Dr Worley's plan to arrange for the initial INR at day 9 to be 'totally appropriate'.[31]

118 In his written report, Dr Norcock was critical of Dr Worley's postoperative management plan, stating that in his view, there was a lack of clear instruction regarding the recommencement of warfarin **and the monitoring** (emphasis added). He retracted the first criticism, appropriately, upon receipt of further information regarding Dr Worley's conversations with Mr Rosenzweig. In relation to the second criticism, he explained that he had since learned that Mr Rosenzweig was not assessed as being at high risk of a thromboembolism, and therefore, his previously recommended regimen of rapid stabilisation with daily INR testing was not relevant.

119 Dr Norcock initially agreed with the following opinion of Dr Joyner, describing it as very harsh but very reasonable:

> There was a lack of correct, informed and competent management over restarting protocol covering the warfarin use.

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[27] T200
[28] T390
[29] Exhibit C19, p4
[30] T347
[31] T239; T243

120 However, he explained that his initial criticism softened with time, the provision of further information, and following conversations with a cardiologist and gastroenterologist, who considered Dr Worley's management to be appropriate and in keeping with his peers. [32]

121 Included within the body of Dr Norcock's report were extracts of conversations between himself and other medical professionals, including Professor Padbury, as set out below:

> Professor Padbury's comments as follows:
>
> No particular benefit to bridging identified pre or intraoperatively but post op bridging recommended in patients at higher risk of embolism. The question remains whether this man's thromboembolic event at the time of aortic valve insertion constitutes elevation to higher risk status.
>
> In summary my conclusion remains the same. Preop management was fine, as was the decision to do the first procedure on warfarin. The problem was the post op with lack of clear instruction regarding the recommencement of warfarin and the monitoring.
>
> Rob

122 Professor Padbury was subsequently commissioned by counsel for Dr Worley to provide an expert report. He did not adhere to the view expressed to Dr Norcock that the monitoring of Mr Rosenzweig's anticoagulation was problematic.

123 Professor Padbury explained that he had been asked a question in generality by Dr Norcock, which he answered having been provided with a precis of the case.[33]

124 Dr Norcock's inclusion of Professor Padbury's opinion in his report illustrates the dangers of relying upon untested hearsay opinion evidence when the facts underpinning it are unknown. I disregard the summary of Professor Padbury's opinion as contained within the report of Dr Norcock. Fortunately, the Court had the benefit of Professor Padbury's considered opinion, the foundation of which was able to be tested.

125 In relation to the opinions of the other colleagues Dr Norcock consulted with, I consider that these opinions are relevant to explain the foundation upon which Dr Norcock formed his own view. I do not consider these extracts to be expert opinions upon which I can rely. It is of note that Dr Norcock's own practice is to return the patient to the therapeutic window as soon as possible and that he is proactive in doing so 'because the one-percenters happen one percent of the time'.[34] Dr Norcock's approach in this regard is consistent with the approach of Dr Sterck.

Analysis

126 The evidence demonstrates that the approach taken to monitoring warfarin resumption could vary from daily INR testing with Clexane coverage, to nine days without a single INR test. The approach taken may, to some extent, be influenced by a practitioner's specialty and relative degree of clinical conservatism. There are no clinical guidelines in place to assist practitioners to determine when a patient's INR ought to be checked

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[32] T247
[33] T353
[34] T242

following resumption of warfarin.[35] In my view, this has contributed to the lack of a standardised approach to re-warfarinisation.

127 Professor Padbury suggested that study protocols adopted in the Bridge trial, the PERIOP2 trial and the Bungard study (the study protocols) represent the closest available proxy to formal guidelines in the literature. He explained that these protocols would have been reviewed by an ethics review board prior to approval, who would have required satisfaction that the protocols were the safest possible. Professor Padbury considered these protocols to be the only current guide to best practice, in the absence of trial evidence.[36] He observed that each of these protocols involved INR testing no later than four days, and then again at day 7 to day 9 after recommencing warfarin.[37]

128 The Bridge trial protocol specified INR testing at least twice during the period between day 2 and day 10. Additional testing beyond this was at the discretion of the site investigator. Of note, all participants in this study had at least one scheduled hospital, or clinic visit, or telephone contact by day 7.

129 The PERIOP2 trial protocol involved testing at day 2 and day 4. Professor Padbury opined that the INR on day 4 was the INR upon which decisions would be made regarding anticoagulation management. The Bungard study protocol required INR testing on day 3 or day 4.

130 I accept the evidence of Professor Padbury that the study protocols represent the only current guide to best practice monitoring of patients recommencing warfarin therapy. The study protocols align with the evidence of Professor Cade and Dr Cehic to the effect that a patient's INR should be checked two or three days after resumption of warfarin.

131 I am satisfied that the safest method of monitoring the effectiveness of a patient's return to therapeutic anticoagulation is to ensure that INR testing occurs no later than four days after warfarin resumption. This enables the clinician to determine whether any adjustments to the warfarin dosage are necessary, or whether other action should be taken, such as further testing. As observed by Dr Cehic, if you do not check, you do not know, and it is preferable to 'make decisions based on the information than on assumptions'.[38]

132 There is a tension between the evidence of Professor Padbury and Dr Norcock on one hand, and the evidence of Professor Cade and Dr Cehic on the other. While Professor Padbury and Dr Norcock opined that Dr Worley's plan was acceptable, Professor Cade and Dr Cehic did not share this view. In assessing the weight to be attributed to their respective evidence, I have considered each witness's practical and theoretical expertise in anticoagulation management.

133 Dr Cehic was not engaged as an expert witness. However, he was asked to provide his opinion on this topic, which was clearly within his area of expertise, and he did so in a manner which was balanced and helpful. I note that Dr Cehic spent the early part of his career managing cardiac patients following surgery, which included their return to

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[35] T312
[36] T358
[37] T312; followed by biweekly to twice weekly thereafter until stable
[38] T200

therapeutic anticoagulation.[39] In addition to this practical experience, Dr Cehic also demonstrated an in-depth knowledge of the literature in this area.

134 Professor Cade's experience in anticoagulation, and thromboembolism in particular, is extensive. He was the head of the anticoagulation clinic within the Royal Melbourne Hospital for several years, his thesis was on coagulation, and for four years, he was Associate Professor at McMaster University (Canada) in pathology and medicine in the thromboembolism program. He has published extensively over the years in the field of thromboembolism and has been a member of the International Society on Thrombosis and Haemostasis for about 40 years. Professor Cade's expertise in this area is academic and practical. I found his evidence to be measured and logical.

135 Professor Padbury frequently dealt with issues of perioperative anticoagulation in his private and public practice as a general surgeon with over 40 years' experience. He told the Court that it was his usual practice to rapidly return the patient to their general practitioner after surgery for anticoagulation management, due to their experience monitoring the patient's INR.[40] His day-to-day experience in monitoring warfarin resumption postoperatively is therefore necessarily limited by this practice. While he acknowledged that balancing warfarin dose and INR results does not fall within his primary area of expertise, his evidence demonstrated an impressive depth of engagement with the academic literature, informed by his considerable expertise.

136 Dr Norcock routinely manages anticoagulated patients as a general practitioner and, accordingly, his evidence was principally grounded in regular practical clinical experience. Dr Norcock's level of support for the approach taken by Dr Worley was unmatched by any other practitioner who expressed a view on the subject. Aside from Dr Norcock, the highest level of support for his approach came from Professor Padbury, who regarded testing at day 9 to be at the outer limit of what he considered reasonable.

137 While I found Dr Norcock's evidence to be honestly given, I was unable to discern the degree to which his opinions were based on his own considerable experience, as opposed to his canvassing of the views of others. Although I accept his evidence that Dr Worley's management may have accorded with the practice of other medical practitioners at that time, I do not consider that this rendered it appropriate. I do not agree that testing at day 9 was 'totally acceptable'. If I were to consider an initial INR test at day 9 to be within the acceptable range, it would certainly be at the very outer limit of the tolerable range.

138 In resolving the tension in the evidence, I have had regard to the study protocols provided by Professor Padbury. With the exception of the Bridge trial, which stipulated testing twice between day 2 and day 10, these protocols involved initial INR testing no later than day 4. I have considered the evidence of the expert witnesses within this context.

139 I have also taken into account the concerns expressed by Dr Worley regarding the dangers of early INR testing. If he is correct that earlier testing creates a risk that a practitioner may prematurely increase a patient's warfarin dose, resulting in an INR above the therapeutic range and an increased risk of postoperative bleeding, that is a serious

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[39] T176
[40] T359

concern. I note the evidence of Dr Cehic and Dr Worley that an internal bleed following polypectomy could itself be fatal.

140 While I accept the sincerity of Dr Worley's concern about 'overshooting' the target INR range, I am not persuaded that it is justified. Dr Worley presumed that a low INR might result in an increase of the patient's warfarin dose, and that this increase would be erroneous. However, an unexpectedly low INR may necessitate an increase in warfarin dosage, just as an unexpectedly high INR may necessitate a decrease.

141 I prefer the reasoning of Dr Cehic that it is preferable to make clinical decisions based on a known INR rather than assumptions. Further, I note that the study protocols, which Professor Padbury considered the best available guide to postoperative warfarin management, involved early testing and adjustments to warfarin doses as required. This is also an approach which finds support in the literature. In particular, the Bungard study concluded with the following recommendation:

> Clinicians are encouraged to be responsive to INRs and dose escalate beyond prior maintenance dosing, as required following an elective procedure, with prompt INR assessment.[41]

142 Having considered the evidence as a whole, I find that the decision to defer initial INR testing until day 9 was an excessively long interval.

143 Professor Padbury opined that there is a significant variation in how medical practitioners approach the monitoring of a patient's return to therapeutic anticoagulation. He expressed the view that this was not a surprise in the absence of defined best practice guidance.[42]

144 I accept that the approach taken by Dr Worley to the monitoring of Mr Rosenzweig's warfarin resumption would have been considered appropriate amongst many of his peers at that time. Rather than demonstrating the correctness of his approach, however, I consider that this demonstrates the need for evidence-based best practice guidelines in this area.

Did Mr Rosenzweig follow Dr Worley's management plan? If so, why was his INR 1.3 on 12 November 2020?

145 Professor Padbury stated that he would have expected Mr Rosenzweig's INR to be higher than 1.3 at day 7 and expressed that he was surprised by that number and could not explain it. Professor Cade was also surprised by that INR result and opined that something had gone 'gravely wrong', to the point that he queried whether Mr Rosenzweig had been taking his warfarin.[43]

146 Mrs Rosenzweig stated in her affidavit that her husband recommenced warfarin the day after the procedure. As far as she knew, he took his usual dose. She believed that she would have seen him take his warfarin in the kitchen in accordance with his usual practice.

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[41] Exhibit C19 at p513
[42] T358
[43] T409

147 Mrs Rosenzweig stated that her husband was always concerned about his warfarin levels. She recalled that after his knee surgery it took some time for his warfarin levels to return to the 'safe window', and that her husband found this worrying and frustrating. This was the reason for him electing to undergo an exploratory colonoscopy on warfarin in the first instance. She stated that her husband 'did not want to play around with the warfarin unless he really had to', and that he was 'pedantic' about taking his medication and she never had to oversee it.

148 Dr Sterck told the Court that Mr Rosenzweig was one of the most compliant patients he had ever seen, and that he believed Mr Rosenzweig would follow every instruction that was given to him by any doctor in relation to any medication.

149 Dr Cehic observed that preparing for a colonoscopy is unpleasant, and some patients may choose to accept the risk of thromboembolism rather than potentially undergo two colonoscopies. Mr Rosenzweig was prepared to endure a second colonoscopy rather than run the increased risk of a thromboembolic event. He had already experienced one stroke, which put an early end to his working life.

150 Mr Rosenzweig also took the initiative to call Dr Worley's rooms on 26 August 2020 to ask whether he needed to replace his warfarin with any other medication. This indicates that he was proactive in relation to his anticoagulation regimen.

151 Mr Katsambis submitted that Mr Rosenzweig preferred the medication Clexane, as demonstrated by the postoperative management of his 2018 partial knee replacement, which involved attendance at the Angaston Medical Centre each day for a period of five consecutive days for the administration of Clexane.

152 I am unable to draw the inference that because Mr Rosenzweig used Clexane in the past that it was his preferred method of managing his anticoagulation following surgery. Mr Rosenzweig experienced difficulties returning to therapeutic anticoagulation following his knee surgery. The use of Clexane was not the panacea to these difficulties and its efficacy in producing an immediate effect following the 2020 procedure cannot be assumed.

153 The note of the intern referred to at [82] suggests that Mr Rosenzweig recommenced warfarin two days prior to his hospitalisation on 12 November 2020, i.e. 10 November 2020 instead of 5 November 2020. Dr Worley denied providing this information to the intern. It is also inconsistent with the recollection of Debra Rosenzweig and with the handwritten instruction to recommence warfarin on '5/11'.

154 To my mind, it is implausible that Mr Rosenzweig would have taken it upon himself to set his own timetable for recommencing warfarin, adjust his dose, or otherwise fail to comply with Dr Worley's advice.

155 I have considered the hypothesis of Professor Padbury that Mr Rosenzweig's unexpectedly low INR could be explained by a failure to recommence warfarin as instructed. However, as Professor Padbury acknowledged, there are many variables that

can influence warfarin effectiveness, and that this theory is an explanation, but not necessarily the explanation.[44]

156 Dr Cehic told the Court that Mr Rosenzweig's INR of 1.3 at the Angaston Hospital indicated that he was clearly not responding to his usual warfarin dose and that something should be done, either an increase in the dose, or even administering Clexane, following consultation with the surgeon. He considered the INR of 1.3 at day 7 to be 'very odd'.[45]

157 Dr Cehic explained that various factors influence the way in which warfarin is metabolised, including other medications, and fruits and vegetables. I also note that on 5 August 2020, Mr Rosenzweig's INR was only 1.9. Dr Sterck noted that there was no apparent underlying cause for this, and that things can change dramatically without any obvious reason.

158 Mr Rosenzweig was acutely aware of the risk of a stroke while his anticoagulation was subtherapeutic and of its potential consequences, which to my mind would have heightened his awareness of the importance of adhering to his anticoagulation management plan. I do not accept that Mr Rosenzweig would have knowingly failed to follow instructions provided to him to recommence warfarin on 5 November 2020.

159 In conclusion, I am satisfied that Mr Rosenzweig followed Dr Worley's management plan and resumed his usual dose of warfarin on 5 November 2020, the day after his second colonoscopy. I am unable to determine why Mr Rosenzweig did not respond as expected to resumption of warfarin.

If Mr Rosenzweig's subtherapeutic INR had been identified at an earlier time, could therapeutic anticoagulation have been restored prior to his stroke?

160 To determine whether the death of Mr Rosenzweig was preventable, it is necessary to consider what measures could have been taken to return him to therapeutic anticoagulation prior to his thromboembolic event on 11 November 2020, seven days after surgery (day 7).

161 Mr Katsambis submitted that Clexane administration on day 7 would have led to full anticoagulation immediately, thereby preventing the death. In support of this submission, Mr Katsambis relied upon the evidence of Dr Worley that he would not have administered Clexane on 12 November 2020 as that 'would have led to full anticoagulation immediately, which would have increased his risk of bleeding'.[46]

162 In my view, it is not open to construe Dr Worley's answer as expert evidence that Clexane immediately restores therapeutic anticoagulation, or that it would do so to a degree or for a period sufficient to prevent the stroke. The evidence of Professor Cade was to the effect that Clexane is fast acting, which I accept.[47] The efficacy of Clexane in preventing thromboembolic events cannot, however, be assumed. For the reasons detailed above, the evidence does not support a finding that postoperative bridging with Clexane would have

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[44] T361
[45] T209
[46] T75
[47] T387

significantly reduced the likelihood of Mr Rosenzweig experiencing a thromboembolic event.[48]

163 I note that Clexane may be administered as part of a perioperative bridging strategy, or in response to a subtherapeutic INR. The efficacy of Clexane for the latter purpose was not explored during the inquest. I accept that Clexane was a treatment option which could have been considered if Mr Rosenzweig's INR was identified as being subtherapeutic prior to his stroke. The evidence does not allow a finding to be made as to its efficacy relative to other measures, such as increasing the dose of warfarin.

Opinions regarding management of subtherapeutic INR

164 I am mindful of the evidence regarding the variable nature of an individual's response to warfarin, and of the various factors, known and unknown, that influence this response. However, an INR of 1.3 is a normal result to be expected of a patient who is not taking warfarin. It is not a result to be expected of a patient who has been taking warfarin for seven days at a dosage which previously resulted in an INR in the range of 2.5 to 3.5 (noting some fluctuations above and below this range). Accordingly, I am persuaded by the evidence of Professor Cade, Dr Cehic and Professor Padbury that an INR of 1.3 on day 7 was a highly unexpected result in the circumstances, which, if identified, warranted action. The general consensus was that the appropriate action would be to increase the dose of warfarin and re-test the INR.

165 Dr Worley told the Court what he thought he would have done if he had checked Mr Rosenzweig's INR at day 7, and the result was 1.3. He said that he would have called Mr Rosenzweig's general practitioner and suggested that his warfarin be gently increased, and Mr Rosenzweig return in two days for a re-measure.[49] He stated that he would not have recommended administration of Clexane at that point, as it would have increased the risk of bleeding.[50]

166 Dr Cehic opined that an INR of 1.3 seven days later would warrant an increased dose of warfarin or, if you were 'starting to get really nervous', Clexane could be considered.[51]

167 Dr Padbury told the Court that he would have responded to an INR of 1.3 at day 7 by considering an increase in the warfarin dose. He believed a low INR at day 4 would also have led him to increase the warfarin dose and re-measure after two days.[52]

168 Dr Sterck believed that he would have administered Clexane if Mr Rosenzweig presented at day 7 with an INR of 1.3.[53]

169 Professor Cade stated that if Mr Rosenzweig attended him on day 5 with an INR of 1.4, he would have considered increasing the dose of warfarin and re-testing.[54]

170 Mr Katsambis submitted that I ought to reject the evidence of Dr Worley that he would not have done anything differently if he had arranged for an INR test on day 7 and the

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[48] See also T328 (Professor Padbury)
[49] T69; T169
[50] T75
[51] T209
[52] T331
[53] T265
[54] T391-2

result was 1.3.[55] However, that was not the evidence of Dr Worley. As set out above, Dr Worley told the Court that he would have suggested that Mr Rosenzweig's warfarin dose be increased, and that he reattend for testing.

171 I accept the evidence of Dr Worley that if he had arranged for an INR test on day 7, instead of day 9, and the result was 1.3, it is probable that he would have advised Dr Sterck, or another practitioner at the Angaston Medical Centre, to increase Mr Rosenzweig's warfarin dose. Had the latter occurred, it is highly likely that the general practitioner would have increased Mr Rosenzweig's warfarin dose as recommended, tested his INR the following day, and monitored him until his INR was back in the therapeutic range. This approach would be consistent with the documented history of how the practice responded to an INR which was outside of the therapeutic range. Alternatively, Clexane may have been administered in conjunction with warfarin therapy until therapeutic anticoagulation had been achieved. While the use of Clexane was not considered appropriate by Dr Worley, it was the approach Dr Sterck believed that he would have taken if responsibility had been delegated to him (which it was not).

172 It does not necessarily follow that if Mr Rosenzweig's INR had been measured on day 7, and found to be 1.3, that his stroke could have been prevented. A reasonable response to an INR of 1.3 on day 7 would have been to increase the dose of warfarin and test the INR the following day. There is no evidence that an increase in warfarin dosage at some stage on day 7 would have prevented the thromboembolic event which occurred that evening at around 10:45 pm. It is a possibility. Although an increased dose of warfarin would be expected to raise the INR towards the therapeutic range, it may not have done so to a sufficient degree by 10:45 pm to prevent the stroke.

173 Mr Katsambis submitted that if Mr Rosenzweig's INR had been tested on day 3 or day 4, this would have allowed for adequate time to achieve an INR at or near the therapeutic range before the evening of day 7 when he suffered the stroke. As set out above, it is my view that the initial INR reading ought to have been taken no later than day 4.

174 I note that Dr Worley told the Court that if INR testing had taken place at day 3 or day 4, the result may not have changed his management as he would expect Mr Rosenzweig's INR to slowly approach the therapeutic level.[56] However, I have no doubt that Dr Worley would have discussed management options with Mr Rosenzweig in those circumstances. This would have provided Mr Rosenzweig with the opportunity to make an informed decision regarding his treatment. Considering Mr Rosenzweig's heightened level of concern regarding stroke, it is implausible that he would have elected to take no action in response to an INR of 1.3 at day 3 or day 4.

175 I also note that Professor Padbury told the Court that a low INR at day 4 would have led him to increase warfarin dosage and re-test. Professor Cade was asked to assume a low INR result at day 5 and opined that he would increase the dose of warfarin and re-test.

176 I find that an INR test on day 3 or day 4 would have returned a subtherapeutic result which would have, on balance, resulted in an increased dosage of warfarin, and further testing.

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[55] T500
[56] T76

177 It is difficult to determine the length of time it would have taken to return Mr Rosenzweig to therapeutic anticoagulation. The evidence as to the expected timeframe was not uniform, and the question is further complicated by the fact that Mr Rosenzweig did not respond as expected to warfarin resumption. There were also four participants in the Bungard study who did not return to therapeutic anticoagulation until their dose of warfarin was increased from their prior maintenance dose. These four participants did not achieve an INR of 2 until day 20-28.[57] The authors commented that this reinforces the need to dose-escalate warfarin in response to the INR and not simply rely on the prior maintenance dosing. In my view, this case places that recommendation into sharp focus.

178 I find that closer monitoring of Mr Rosenzweig's return to therapeutic anticoagulation, including INR testing on day 3 or day 4 after warfarin resumption, would have provided Dr Worley with the opportunity to make an informed decision, in consultation with Mr Rosenzweig, as to his management. If Mr Rosenzweig's failure to respond to warfarin had been recognised by day 3 or day 4, action would have been taken which may have returned him to therapeutic anticoagulation, whether by increasing his warfarin dose, or potentially utilising Clexane.

179 If Mr Rosenzweig's INR had been tested on day 4 and found to be 1.3, clinicians would have had approximately three days to intervene prior to the thromboembolic event. The evidence of the witnesses was that an increased dose of warfarin may take approximately 48 hours to produce an increase in the INR.[58] Accordingly, an increased dose of warfarin on day 4 would be expected to affect the INR within two days, i.e. by day 6. This suggests that if Mr Rosenzweig's subtherapeutic INR had been detected on day 4 and his warfarin dose increased, his INR would have been higher than 1.3 on day 6. This would have reduced his risk of a thromboembolic event.

180 However, the evidence does not enable a reliable assessment of the extent to which Mr Rosenzweig's INR would have increased had the warfarin dose been increased on day 4. Nor does it enable a reliable assessment as to whether any such increase would have been sufficient to prevent the thromboembolic event. While it is possible that intervention on day 4 may have restored the INR to the therapeutic range in time to prevent the event, I am not satisfied, on the balance of probabilities, that it would have done so.

What was the effect of extended subtherapeutic anticoagulation on Mr Rosenzweig's thromboembolic risk?

181 Mr Rosenzweig ceased taking warfarin on 31 October 2020 and underwent surgery on 4 November 2020. His INR would have been 1.5 or below on the day of surgery.[59] At 12:22 am on 12 November 2020, his INR was measured at 1.3. Accordingly, he remained subtherapeutic for a minimum of eight days, and possibly longer depending on when his INR fell below 2.5 in the period leading up to surgery. As set out earlier in these findings, this period was significantly longer than anticipated. Mr Rosenzweig's risk of thromboembolism increased with each day of subtherapeutic anticoagulation. The risk occasioned by the procedure was therefore higher than anticipated.

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[57] Exhibit C19, p513
[58] T407-8, T199, T68
[59] T316 (Padbury)

182 Professor Cade, during his oral evidence, undertook a calculation to determine Mr Rosenzweig's increased statistical risk of thromboembolism caused by his extended period of subtherapeutic anticoagulation. He estimated that Mr Rosenzweig was subtherapeutic for two or three times as long as he should have been, and that he should have been approaching the therapeutic level four to five days after resuming warfarin. He explained that he would expect the length of time it took Mr Rosenzweig to transition from an anticoagulated state to a coagulable state (INR of 1) to be similar to the length of time it would take him to return to therapeutic anticoagulation, i.e. four days.[60] The thromboembolic risk of coming off anticoagulation for four days is approximately 1 in 1500 (rounded).

183 However, Mr Rosenzweig's anticoagulation was subtherapeutic for at least eight days. According to Professor Cade, eight days of subtherapeutic anticoagulation doubled the thromboembolic risk of the anticipated four days.[61] The statistical thromboembolic risk of eight days without adequate anticoagulation increased to 1 in 650.

184 Professor Padbury, on the other hand, calculated the additional days at risk as two to three, assuming that the quickest possible time that the INR could be returned to therapeutic was around day 4 to day 5, noting the median time of 7.8 days in the Bungard study. Based on the daily risk of 1:5000, he deduced that the thromboembolic risk for Mr Rosenzweig would have been 2-3:5000 or 1:1667-2500. Assuming a re-anticoagulation period of five days, two additional days increased the risk from 1:1000 to 1:714. Three additional days would increase the estimated risk to 1:625.

185 Professor Cade did not consider the timeframe of warfarin offset and onset to be material to his calculations. He explained that regardless of the pharmacodynamics of warfarin, the fact is that Mr Rosenzweig was still subtherapeutic 12 days after ceasing warfarin. At the very least, Professor Cade opined that Mr Rosenzweig must have been sub-therapeutic from the time of the procedure on 4 November 2020 until his admission to the Royal Adelaide Hospital on 12 November 2020, i.e. eight days. I agree.

186 Professor Cade considered the expected timeframe for Mr Rosenzweig to be subtherapeutic was four days. It was on this basis that he considered Mr Rosenzweig's actual thromboembolic risk to have been at least double the anticipated risk. Professor Padbury, on the other hand, considered four to five days to be the fastest possible timeframe to return to therapeutic anticoagulation. He did not consider that the actual thromboembolic risk was double the expected risk.

187 I consider that the expected timeframe for Mr Rosenzweig's anticoagulation to be subtherapeutic after surgery was a minimum of four days, but more likely a week. Accordingly, I am unable to find that Mr Rosenzweig's actual thromboembolic risk doubled in comparison with the expected risk.

188 I also note that the thromboembolic risk is known to increase not merely as a function of the time spent outside of the thromboembolic range, but also according to the degree to which the anticoagulation was subtherapeutic. Mr Rosenzweig was not approaching therapeutic anticoagulation as expected on 12 November 2020. His coagulation was

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[60] T407-8
[61] Exhibit C20e

normal. Mr Rosenzweig was not following the expected trajectory and, accordingly, his risk was elevated by the degree to which his anticoagulation was subtherapeutic by day 7.

189 I consider the statistical evidence to be of limited utility in illustrating the relative increase in thromboembolic risk associated with subtherapeutic anticoagulation in this case. These figures are necessarily approximate and population-based, rather than precise predictors of an individual's level of risk. I therefore find, in more general terms, that Mr Rosenzweig was unknowingly exposed to a materially increased thromboembolic risk.

Was the death of Mr Rosenzweig preventable?

190 Professor Cade opined that it is more probable than not that Mr Rosenzweig's death could have been prevented by more timely attention to his anticoagulation regimen. While the risk that eventuated was low, it was materially higher than it needed to be.

191 Special Counsel assisting the Court, Ms Schell, relied principally upon Professor Cade's evidence in support of a submission that timely attention to Mr Rosenzweig's anticoagulation program in the postoperative period would have prevented his death on the balance of probabilities.

192 Mr Katsambis submitted that Mr Rosenzweig's death was 'entirely preventable'. He emphasised Mr Rosenzweig's low thromboembolic risk while appropriately anticoagulated and argued that early identification of his failure to respond as expected to warfarin would have enabled him to be restored to therapeutic anticoagulation, which therefore would have prevented the stroke.

193 Mr Harris submitted that a finding cannot be made that the death was preventable. Mr Harris highlighted the difficulty in determining whether the stroke occurred because of the extended period of subtherapeutic anticoagulation, or due to Mr Rosenzweig's baseline thromboembolic risk. Further, considering the evidence that it may take seven to nine days to return to therapeutic anticoagulation, Mr Harris argued that the failure to perform an INR test at day 3 or day 4 is irrelevant, as it would be reasonable to expect that the INR would be subtherapeutic at that time. Mr Harris also submitted that it would have been appropriate to manage a subtherapeutic INR at day 7 with an increased dose of warfarin, and that it cannot be assumed that this would have taken effect such that the stroke on day 7 would not have occurred.

194 I do not consider that Mr Rosenzweig's baseline thromboembolic risk precludes a finding that his death was preventable on the balance of probabilities. The relevant issue is the extent to which the risk materially increased during the period of subtherapeutic anticoagulation, and whether it is more likely than not that the increased risk, if known, could have been ameliorated to the necessary degree to prevent the stroke.

195 I agree with the submission of Mr Harris that INR testing at day 7 and subsequent adjustment to Mr Rosenzweig's warfarin dose is unlikely to have prevented the stroke. While I am satisfied that INR testing at day 4 represented the most appropriate course in the circumstances, I cannot find that, on the balance of probabilities, this would have allowed sufficient time to restore Mr Rosenzweig to therapeutic anticoagulation, given his failure to respond as expected to resumption of warfarin.

196 However, I am satisfied that there was a missed opportunity to identify and correct Mr Rosenzweig's failure to respond to warfarin. Closer monitoring of his postoperative anticoagulation regimen by INR testing no later than day 4 would have identified this issue and potentially afforded sufficient opportunity for successful intervention.

197 I find that the death of Mr Rosenzweig was potentially preventable.

Was Dr Worley's decision not to notify the Angaston Medical Centre of the management plan a missed opportunity to prevent the death of Mr Rosenzweig?

198 Dr Sterck did not believe that he knew of the second colonoscopy on 4 November 2020. Dr Sterck last saw Mr Rosenzweig on 28 October 2020. It was his evidence that if he had been informed of the upcoming procedure during this consult, he would have corresponded with the proceduralist, arranged for bridging anticoagulation, and monitored Mr Rosenzweig's anticoagulation until he had returned to his therapeutic level. He was adamant that he would not have made Mr Rosenzweig an appointment for six weeks' time if he had known that he would be ceasing and recommencing warfarin in the intervening period.

199 It is unnecessary for me to make a finding as to whether Mr Rosenzweig would have been administered Clexane by Dr Sterck if he had known of the second procedure, considering the finding above in relation to the utility of this course in reducing thromboembolic risk. However, if I accept Dr Sterck would have undertaken prompt and regular INR monitoring following the procedure if he had known of it, the failure to communicate that information amounted to a missed opportunity to identify and potentially correct Mr Rosenzweig's subtherapeutic anticoagulation, which may have prevented his death.

200 Dr Worley accepted that he did not notify the Angaston Medical Centre of the need for a second colonoscopy off warfarin once that need became apparent. He did not write to the practice following the second colonoscopy or otherwise advise of his plan for monitoring Mr Rosenzweig's INR. He explained that he intended to do so after the follow-up appointment, which was his usual practice at that time.

201 Dr Worley did arrange for the pathology results of the second procedure to be copied into Dr Nunis at the Angaston Medical Centre. However, the histopathology results were not reported by the laboratory until 7 November 2020 and therefore could not have been seen by Dr Sterck during the 28 October 2020 consult, or in preparation for it.

202 Concerningly, the electronic medical record system used by the general practice at that time required the practitioner, to whom the correspondence was directed, to 'action' the document before it would become accessible on the patient's medical file. Accordingly, if Dr Nunis did not open the histopathology results and 'action' them, they would not have been available to any other practitioner at the Angaston Medical Centre. It is not clear what procedures were in place to manage this risk, for example, during a practitioner's extended absence. The records do not indicate when Mr Rosenzweig's histopathology results were opened, or by who. Dr Sterck told the Court he was not aware of these results until after the death of Mr Rosenzweig.

203 Dr Worley expressed surprise bordering on disbelief that Dr Sterck was not informed of the second colonoscopy by Mr Rosenzweig during his appointment on 28 October 2020, given that Mr Rosenzweig was due to cease his warfarin two days later. Further, the consult was long and included an INR check.

204 Dr Sterck did not record a note of the upcoming procedure, which he believed he would have done if informed of it. He believed that he was only aware of the second appointment following the passing of Mr Rosenzweig.

205 Mr Katsambis and Ms Schell submitted that I ought to accept the evidence of Dr Sterck that he did not know of Mr Rosenzweig's second colonoscopy off warfarin. Mr Katsambis relied in part upon Dr Sterck's response and demeanour when confronted with the proposition that he must have been told by Mr Rosenzweig during his appointment on 28 October 2020.

206 Mr Harris, on the other hand, submitted that Dr Sterck must have known of the second colonoscopy and that it should not be inferred that the absence of a note of the date suggests otherwise. Mr Harris directed me to the notes of a consult on 5 August 2020, which did not refer to a discussion about the first colonoscopy, a conversation which Dr Sterck agreed likely occurred. Mr Harris did not attack the credibility of Dr Sterck; rather, he suggested that his memory let him down.

207 Having observed Dr Sterck giving evidence, I found him to be a conscientious witness who gave his evidence in a straightforward manner. His previous management of Mr Rosenzweig's anticoagulation was proactive. I have no doubt, based on his evidence and past practice, that if Dr Sterck had known of this procedure, he would have made an appointment to see Mr Rosenzweig shortly afterwards.

208 Accordingly, I find that Dr Worley's failure to notify the Angaston Medical Centre of the second procedure off warfarin was a missed opportunity to attempt to restore Mr Rosenzweig to therapeutic anticoagulation prior to the stroke.

209 Dr Cehic told the Court that, in his view, communication between clinicians is the Achilles heel of medicine. He stated:

> I think communication amongst treating practitioners is essential and I think it is one of the Achilles heel of modern medicine in that - you know not in this case in particular - but in general, it's becoming increasingly frustrating that often we receive no communication from other specialists and with this trend towards general practitioners giving indefinite referrals, an increasing trend that we hear nothing from GPs either which I think is not actually good patient care.[62]

210 The present case demonstrates the need for communication between treating clinicians.

211 Dr Worley has made several changes to his practice since the death of Mr Rosenzweig. Significantly, he is now more inclined to involve general practitioners at every step, notwithstanding that he still maintains responsibility for the perioperative anticoagulation regime.

212 Dr Worley stated that he now engages with the patient's cardiologist for approval or input in relation to the anticoagulation program for a cardiac patient requiring non-cardiac surgery. Dr Cehic considered this to be a good conservative approach but observed it is not one that he often sees unless it is relating to a patient at extraordinarily high risk.

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[62] T179

213 Professor Cade opined that a cardiologist or cardiac team should always be involved in non-cardiac surgery in a cardiac patient.

214 Dr Worley did not, however, concede that he should have checked Mr Rosenzweig's INR at an earlier time, nor did he refer to any changes in his practice in relation to INR monitoring. Dr Worley was a witness who made appropriate concessions, other than in this important respect. This illustrates the need for guidelines in this area to inform experienced and competent practitioners such as Dr Worley as to the importance of early INR testing.

Recommendations

215 Pursuant to section 25(2) of the Coroner's 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.

216 The expert witnesses were asked to consider the need for development of guidelines for the postoperative monitoring of a patient's INR following interruption of warfarin therapy for surgery. Professor Padbury suggested adopting a best practice approach extrapolated from methodology used in the study protocols.[63] This methodology involved an INR test at day 3 or 4, repeated at days 7 to 9.

217 Professor Cade agreed that the study protocols represent the only current guide to best practice monitoring of patients recommencing warfarin therapy. He agreed that these protocols are a good starting point for best practice guidelines and described Professor Padbury's suggestion as wise. I agree.

218 Ms Schell submitted that I consider making a recommendation to the Minister for Health for the formulation of a working group including representatives from the appropriate fields of anticoagulation management to develop guidelines for the postoperative management of anticoagulated patients.

219 Mr Katsambis supported this recommendation, however suggested that the Clinical Excellence Commission in New South Wales could be invited to supplement their existing guideline with a component relating to postoperative INR monitoring.

220 I make the following recommendation directed to the Minister for Health and Wellbeing:

One That best practice guidelines concerning the postoperative monitoring of patients requiring interruption of warfarin therapy are created and implemented across all Local Health Networks.

221 I consider it to be a matter for the Minister to determine how these guidelines are developed and whether he adopts the suggestion of Special Counsel regarding the formulation of a working group or the suggestion by Mr Katsambis of consultation with the Clinical Excellence Commission in New South Wales.[64]

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[63] T360

[64] I note that an Anticoagulant Medicines Working Party contributed to the development of the Clinical Excellence Guidelines, which includes a pro forma document which can be used to record a patient's anticoagulation management plan

222 It is respectfully suggested that the following professional bodies be consulted in the development of these guidelines:

- The Haematology Society of Australia and New Zealand
- The Thrombosis & Haemostasis Society of Australia and New Zealand
- The Royal Australasian College of Surgeons
- The Royal Australian College of General Practitioners
- The Royal Australasian College of Physicians
- The Cardiac Society of Australia and New Zealand

223 I make the following recommendation directed to the Haematology Society of Australia and New Zealand, the Royal Australasian College of Surgeons, Royal Australasian College of General Practitioners, Royal Australasian College of Physicians, and the Cardiac Society of Australia and New Zealand:

Two That a Bulletin be circulated promoting the importance of early monitoring of a patient's anticoagulation following interruption of warfarin therapy, including a recommendation that the initial INR test is performed no later than four days after surgery, and that the monitoring plan is clearly documented and a copy is provided to the patient and their general practitioner.

Three That the circumstances of Mr Rosenzweig's death are circulated as a case study for educational purposes.

Conclusion

224 It is acknowledged that the risk of a thromboembolic event for a patient with a mechanical aortic valve is low. However, the potential consequences can be fatal. It is for this reason that lifelong anticoagulation is recommended, together with regular monitoring and dose adjustments as necessary. The relatively low statistical risk of thromboembolism does not diminish the importance of assertive postoperative monitoring for patients required to interrupt anticoagulation therapy.

225 I express my condolences to Debra Rosenzweig and to the family and friends of Mr Rosenzweig.

226 I also recognise the expression of condolences made by Dr Worley to the family of Mr Rosenzweig. His expression of grief appeared sincere, and I acknowledge the courage it took to voice those sentiments in open court. As Dr Worley observed, his own grief does not compare to, nor diminish, the depth of loss experienced by the family of Mr Rosenzweig.

227 I thank Special Counsel, Mr Harris, and Mr Katsambis for their assistance in this inquest.

Keywords: *Stroke; Thromboembolism; Anticoagulation; Postoperative Monitoring*