



IN THE CORONERS COURT
OF VICTORIA
AT MELBOURNE

Court Reference: COR 2014 1941

FINDING INTO DEATH WITH INQUEST

Form 37 Rule 60(1)

Section 67 of the Coroners Act 2008

Deceased: Sarah Louise Rose

Delivered on: 31 March 2021

Delivered at: Coroners Court of Victoria,
65 Kavanagh Street, Southbank

Hearing dates: Directions Hearing: 9 February 2017
Inquest: 12 April 2019
Written Submissions: May 2019

Findings of: Coroner Paresa Antoniadis Spanos

Counsel assisting the Coroner: Leading Senior Constable Kelly Ramsey from the
Police Coronial Support Unit

Representation: Mr P. Over appeared on behalf of Mr B. Rose
Mr P. Halley appeared on behalf of Dr St John, Dr
Kreltzhaim and Dr Dookhan
Mr A. Pillay appeared on behalf of Eastern Health
Mr J. Arranga appeared on behalf of Dr Nakajima
Ms F. Ellis appeared on behalf of Dr Ratnarajah

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INTRODUCTION¹

1. Sarah Louise Rose was a 45-year old married woman who resided in Vermont with her husband Benjamin Rose and their three children Henry, Stella and Angus. Mrs Rose generally enjoyed good health but struggled with her weight and underwent gastric band surgery in 1998. In 2000, Ms Rose underwent surgery again to replace the gastric band due to an infection. Thereafter, Ms Rose was generally well. She worked full-time as an Investigations Manager at Cignis, Higgins and Shaw, as well as caring for her family.
2. In late 2011, Mrs Rose fell pregnant with their third child Angus, and returned to obstetrician Dr Peter England for management of her antenatal care as she had with her previous pregnancies. Early in the pregnancy, in late 2011, Dr England diagnosed Mrs Rose with Hashimoto's disease² and commenced her on daily thyroxine 100mg. According to her husband, Mrs Rose's health appeared to improve during her pregnancy. Angus was born on 18 July 2012 and Mrs Rose was generally well following the delivery and for some 18 months.

CIRCUMSTANCES IMMEDIATELY PROXIMATE TO DEATH

3. From January 2014, Mrs Rose's health deteriorated with frequent headaches and dizzy spells, episodes of swelling of her hands and feet, confusion and a degree of altered consciousness. Mrs Rose sought medical attention from general practitioners at the Blackburn Medical Clinic and had two admissions to Box Hill Hospital (BHH), the first in February and the second in early April 2014.
4. Despite multiple investigations, treating clinicians were unable to reach a definitive diagnosis. The clinical impression was of stress-related headaches and Mrs Rose was discharged home on 11 April 2014 asymptomatic of headaches.
5. On Saturday 12 April 2014, Mrs Rose had a severe headache and complained of feeling dizzy. Her husband observed possible seizure activity and called 000. Ambulance Victoria paramedics responded a short time later and transported Mrs Rose to the BHH Emergency Department (ED) where she was admitted once again.
6. A CT scan of the brain undertaken at BBH on 12 April 2014 was reported as normal. However, Mrs Rose's conscious state fluctuated, and she remained intubated until 14 April 2014 after which an MRI of the brain was reported as highly suspicious for meningitis and possibly encephalitis.

¹ This is an introductory summary of matters that were uncontentious and is taken largely from the statement of Mr Benjamin Rose at page 281 of the brief.

² This will be discussed in some detail below – paragraphs 22 and following.

7. Mrs Rose was extubated but suffered a cardiopulmonary arrest on 15 April 2014 which required her to be intubated again. A repeat CT scan of the brain showed diffuse brain oedema and probable signs of hypoxic ischaemic injury. Mrs Rose was extubated and passed away later that day.

INVESTIGATION AND SOURCES OF EVIDENCE

8. This finding is based on the totality of the material the product of the coronial investigation of Mrs Rose's death. That is, the brief of evidence compiled by Leading Senior Constable Remo Antolini from the Police Coronial Support Unit that includes statements from Mr Rose, other family members, treating clinicians and several expert reports; and the evidence of the conclave of expert witnesses, both those who provided independent expert reports commissioned by the Court and those who provided medico-legal opinions for the family and other parties; and the final submissions of Counsel provided in written form.
9. All of this material, together with the inquest transcript, will remain on the coronial file.³ In writing this finding, I do not purport to summarise all the material and evidence but will only refer to it in such detail as is warranted by its forensic significance and the interests of narrative clarity.

PURPOSE OF A CORONIAL INVESTIGATION

10. The purpose of a coronial investigation of a *reportable death*⁴ is to ascertain, if possible, the identity of the deceased person, the cause of death and the circumstances in which death occurred.⁵ Mrs Rose's death falls within the definition of a reportable death, specifically section 4(2)(a) of the Act which includes a death that appears to be unexpected.
11. The *cause* of death refers to the *medical* cause of death, incorporating where possible the *mode* or *mechanism* of death. For coronial purposes, the *circumstances* in which death occurred refers to the context or background and surrounding circumstances but is confined to those circumstances sufficiently proximate and causally relevant to the death, and not all those circumstances which might form part of a narrative culminating in death.⁶

³ From the commencement of the *Coroners Act 2008* (the Act), that is 1 November 2009, access to documents held by the Coroners Court of Victoria is governed by section 115 of the Act. Unless otherwise stipulated, all references to legislation that follow are to provisions of the Act.

⁴ The term is exhaustively defined in section 4 of the *Coroners Act 2008* [the Act]. Apart from a jurisdictional nexus with the State of Victoria a reportable death includes deaths that appear to have been unexpected, unnatural or violent or to have resulted, directly or indirectly, from an accident or injury; and, deaths that occur during or following a medical procedure where the death is or may be causally related to the medical procedure and a registered medical practitioner would not, immediately before the procedure, have reasonably expected the death (section 4(2)(a) and (b) of the Act).

⁵ Section 67(1).

⁶ This is the effect of the authorities – see for example *Harmsworth v The State Coroner* [1989] VR 989; *Clancy v West* (Unreported 17/08/1994, Supreme Court of Victoria, Harper J.)

12. The broader purpose of any coronial investigations is to contribute to the reduction of the number of preventable deaths through the findings of the investigation and the making of recommendations by coroners, generally referred to as the *prevention* role.⁷ Coroners are empowered to report to the Attorney-General in relation to a death; to comment on any matter connected with the death they have investigated, including matters of public health or safety and the administration of justice; and to make recommendations to any Minister or public statutory authority on any matter connected with the death, including public health or safety or the administration of justice.⁸ These are effectively the vehicles by which the coroner's prevention role can be advanced.⁹
13. It is important to stress that coroners are not empowered to determine the civil or criminal liability arising from the investigation of a reportable death and are specifically prohibited from including in a finding or comment any statement that a person is, or may be, guilty of an offence.¹⁰

IDENTIFICATION

14. There were no contentious issues surrounding Mrs Rose's identity. Her husband Benjamin Leon Rose signed a formal Statement of Identification dated 15 April 2014 before a member of the clinical staff of Box Hill Hospital.
15. Identification was not in issue and required no further investigation.

THE FOCUS OF THE CORONIAL INVESTIGATION

16. Coronial investigations generally focus on the circumstances in which the death occurred. With deaths such as this one, that occur in a healthcare setting, investigation of the circumstances usually focuses on the adequacy of the clinical management and care provided to the deceased proximate to death.
17. Somewhat unusually, the primary focus of the coronial investigation was on the medical cause of her death which remained unascertained despite a full post-mortem examination (autopsy) and a range of ancillary investigations.

⁷ The 'prevention' role is now explicitly articulated in the Preamble and purposes of the Act, compared with the *Coroners Act 1985* where this role was generally accepted as 'implicit'.

⁸ See sections 72(1), 67(3) and 72(2) regarding reports, comments and recommendations respectively.

⁹ See also sections 73(1) and 72(5) which requires publication of coronial findings, comments and recommendations and responses respectively; section 72(3) and (4) which oblige the recipient of a coronial recommendation to respond within three months, specifying a statement of action which has or will be taken in relation to the recommendation.

¹⁰ Section 69(1). However, a coroner may include a statement relating to a notification to the Director of Public Prosecutions if they believe an indictable offence may have been committed in connection with the death. See sections 69 (2) and 49(1).

18. I took the view that unless and until the medical cause of death could be ascertained or could be elucidated as far as possible, the adequacy of the clinical management and care provided to Mrs Rose from about January 2014 when her health started to deteriorate until her death, could not be meaningfully evaluated.

MEDICAL CAUSE OF DEATH – FORENSIC PATHOLOGIST’S OPINION

19. Dr Gregory Ross Young from the Victorian Institute of Forensic Medicine (VIFM), then a pathology registrar, now a forensic pathologist, reviewed the circumstances of death as reported by police to the coroner (police Form 83), post-mortem CT scanning of the whole body undertaken at VIFM (PMCT) and available medical records and performed an autopsy under the supervision of Professor Noel Woodford, Director of VIFM. Having done so Dr Young provided a seventeen-page written report of his findings and an opinion as to the cause of death.¹¹
20. Dr Young found no post-mortem evidence of any injuries that may have caused or contributed to death.
21. Routine toxicological analysis of ante-mortem and post-mortem samples showed no ethanol (alcohol) or other commonly encountered drugs or poisons apart from 7-aminoclonazepam¹² and lignocaine,¹³ at levels consistent with therapeutic use.
22. Relevantly, Dr Young’s anatomical findings included Hashimoto’s thyroiditis with thyroid function tests showing profound hypothyroidism and a neuropathological finding of diffuse cerebral oedema with rare chromatolysis of neurons in the cerebral cortex and no evidence of encephalitis, vasculitis, vasculopathy or haemorrhage.
23. The neuropathological findings were based on a specialist report from pathologist Associate Professor Penny McElvie at St Vincent’s Pathology, Melbourne, whose conclusion was that “*the aetiology for the cerebral swelling in this patient is not evident*”.¹⁴ Nor did neuropathology of the brain reveal any cause for seizure activity.¹⁵

¹¹ The autopsy was performed on the morning of 17 April 2014. Dr Young’s autopsy report dated 30 October 2014 commences at page 1 of the brief and includes his formal qualifications and experience.

¹² Clonazepam is a benzodiazepine drug related to diazepam possessing sedative and anticonvulsant properties. It is available in Australia as Rivotril and Paxam. Clonazepam is metabolised to 7-aminoclonazepam. This metabolism may occur post-mortem and concentrations can be used as an indicator of clonazepam concentrations.

¹³ Lignocaine is a local anaesthetic often administered to patients prior to surgery or during resuscitation which may also be used as an anti-arrhythmic drug to return the heart to a more regular rhythm.

¹⁴ A/Prof McElvie’s neuropathological examination report is at page 53 of the brief.

¹⁵ Dr Young’s autopsy report at page 16 of the brief.

24. Referring to the testing of serum for thyroid function, Dr Young advised that the results were consistent with primary hypothyroidism, as well as being indicative of inadequate replacement with thyroxine, either due to the dose being too low or the thyroxine not being taken at all.¹⁶
25. Dr Young described Hashimoto's thyroiditis as an autoimmune cause of hypothyroidism, and myxoedema coma (or crisis) as an uncommon but life-threatening form of untreated hypothyroidism with physiological decompensation that usually occurs in patients with long-standing untreated hypothyroidism, usually precipitated by hypothermia, infection, other systemic disease or drug therapy.
26. Dr Young further advised that patients with myxoedema coma have changes in their mental state that can include lethargy, stupor, delirium or coma. Cardiovascular effects include bradycardia and hypotension. Hypothermia, normocytic anaemia and hyponatraemia may also be seen. Metabolic disturbance often leads to weight gain.¹⁷ Dr Young described myxoedema coma as a metabolic and cardiovascular emergency with a high mortality rate if not treated.¹⁸
27. Dr Young also commented about Hashimoto's encephalitis as a diagnosis of potential relevance to Mrs Rose's death. He advised that people with Hashimoto thyroiditis may *very rarely* present with Hashimoto's encephalopathy which is a sub-acute process that responds to immunosuppression (generally with steroids) and not thyroxine replacement. According to Dr Young, while the neurological symptoms in this condition are similar to myxoedema coma, it is usually seen in the euthyroid (normal thyroid) state or after correction of hypothyroidism and is therefore not a diagnosis supported by the thyroid function tests in Mrs Rose's case.¹⁹
28. A number of other possible diagnoses or conditions were considered by Dr Young who noted that there are several causes of sudden unexpected death that leave no signs discernible at post-mortem examination. These include metabolic and biological derangements, cardiac arrhythmias, cardiac channelopathies and seizure disorders.²⁰
29. In conclusion, Dr Young advised that despite the clinical, histological and biochemical findings in keeping with significant hypothyroidism around the time of death, the contribution of this condition to death is not able to be established with any certainty, and the

¹⁶ See comment 6 at page 14 of the brief where the results are documented.

¹⁷ There is evidence that Ms Rose suffered from most, if not all, of these symptoms during the four-month period immediately preceding her death, some evident during her last admission.

¹⁸ See comments 9 and 10 at page 15 of the brief.

¹⁹ See comment 11 at page 15 of the brief.

²⁰ See comments 12, 13, 16, 17 and 18 at pages 15-16 of the brief.

cause of death is best formulated as unascertained, with the possibility of contribution from other unidentifiable natural disease processes unable to be excluded.

OTHER EXPERT OPINIONS

30. In an effort to ascertain a definitive cause of death if possible, three independent expert reports were commissioned by the court. These were the reports of Consultant Physician Dr Peter Greenberg dated 24 May 2015²¹; the report of Consultant Clinical Endocrinologist Professor Duncan Topliss, with a particular expertise in thyroid disease, dated 17 August 2015²²; and the report of Neurologist Dr Jorge Zavala²³ undated, appearing at page 67 of the brief.

31. These reports were provided to Dr Young who provided a Supplementary Report dated 24 March 2017 in which he reiterated his conclusions above and maintained the view that the cause of Mrs Rose's death remains unascertained.²⁴

32. Apart from the independent expert reports commissioned by the court, I was also assisted by an expert report provided by Consultant Endocrinologist Dr John Walsh,²⁵ with a particular interest in thyroid disease, on behalf of Eastern Health; and two expert reports provided by Consultant Physician and Neurologist Professor Bruce Brew, on behalf of Mr Benjamin Rose.²⁶

33. To state the obvious, while the experts shared some common ground, in particular recognising the complexity of Mrs Rose's clinical presentation and the unlikelihood that there was one simple diagnosis or underlying cause of her symptoms that was missed, I was unable to discern sufficient commonality in their reports to support any elucidation of the medical cause of Mrs Rose's death without further input.

34. This was a case which appeared to me likely to benefit from the hearing of concurrent evidence from all the expert witnesses who had already turned their minds to the diagnostic

²¹ Dr Greenberg's report dated 24 May 2015 is at page 59 of the brief.

²² Prof Topliss' report dated 17 August 2015 is at page 74 of the brief. Prof Topliss was subsequently provided with additional material and submitted a second report dated 8 June 2017 at page 69 of the brief. This report was mainly focused on issues of clinical management and care that might have been appropriate had Mrs Rose's hypothyroid state been recognised during her two hospital admissions and whether this would have materially altered the outcome.

²³ Dr Zavala's undated report is at page 67 of the brief.

²⁴ The Supplementary Report is at pages 18-19 of the brief. In this report, Dr Young stated as follows – "*It is clear that the deceased had Hashimoto thyroiditis (seen at autopsy) and biochemical evidence of hypothyroidism (established on analysis of antemortem specimens). What is not clear is whether thyroid disease has contributed to death (Hashimoto encephalopathy and/or hypothyroidism), whether directly or indirectly, and/or in conjunction with any other disease processes.*"

²⁵ Dr Walsh's report dated 26 July 2017 is at page 138 of the brief.

²⁶ Prof Brew's report dated 29 January 2016 at page 99 of the brief was based on Mrs Rose's medical records and radiological imaging and reports from Eastern Health. His second report dated 12 March 2018 at page 80 of the brief takes into account his first report and the reports of all the other experts, as well as Dr Young's autopsy report.

quandary posed by Mrs Rose, including forensic pathologist Dr Young. My hope was that a consensus view might be reached that would further elucidate or refine the medical cause of Mrs Rose's death and provide context for any appraisal of the clinical management and care provided to her by the general practitioners at the Blackburn Clinic and by Eastern Health.

EVIDENCE AT INQUEST - THE CONCLAVE OF EXPERTS

35. The parties were invited to submit questions which could be included in the questions to be asked of the conclave of experts and a hearing was listed. Following some explanatory remarks the conclave of experts was provided with a list of questions settled by me and afforded exclusive use of the courtroom so they could consider the questions, review the coronial brief and medical records and formulate a unanimous response where possible, or absent unanimity, as much of a consensus as possible. The inquest was adjourned to allow them the opportunity to do so.²⁷
36. When the inquest resumed later in the day, each of the experts was sworn or affirmed as a witness, and the questions put to them by my assistant LSC Ramsey. On the whole, Prof Topliss answered as spokesperson conveying the consensus view where one had been reached, and other witnesses offered their own testimony or gloss where appropriate, as is accurately captured in the transcript.²⁸
37. The conclave of experts was unanimous in the opinion that cerebral oedema leading to brain stem compression aptly describe the *mechanism* of Mrs Rose's death, as opposed to the *cause* of death. The focus of the inquest was on the antecedent cause of the cerebral oedema or medical cause of death proper. As a forensic pathologist, Dr Young was well-placed to make this distinction, which was accepted by Prof Topliss in evidence.²⁹
38. It was uncontroversial that Mrs Rose had been diagnosed with Hashimoto's disease or Hashimoto's thyroiditis by her obstetrician Dr England during her last pregnancy. Noting the results of antithyroid peroxide antibodies in December 2011, the consensus view was that this was a sound diagnosis.
39. Prof Topliss explained that Hashimoto's thyroiditis is a pathological condition of the thyroid which can result in impairment of thyroid function. Some people may have Hashimoto's thyroiditis and still have normal thyroid function; some may have impaired thyroid function

²⁷ See transcript pages 3 and following for my introductory remarks.

²⁸ See transcript pages 10 and following.

²⁹ Transcript page 25.

or sub-clinical hypothyroidism; or they may have frank biochemical hypothyroiditis which is clear clinically apparent, albeit with individual variation.³⁰

40. The appropriate treatment for Hashimoto's thyroiditis is daily thyroxine which needs to be taken for life. It was uncontroversial that Mrs Rose was prescribed thyroxine and took it as prescribed for a period. Significantly, the last documented dispensing of thyroxine to Mrs Rose was in April 2012³¹ and thyroid function tests undertaken in September 2012 showed normal results (euthyroid). In the absence of any other thyroxine available to her, say from the dispensing of her previous prescription, Mrs Rose would have had sufficient thyroxine to take her prescribed dose until about October 2012. Relevantly, thyroid function tests in September 2012 indicated she was euthyroid at that time.
41. While post-mortem levels supported the proposition that Mrs Rose had not been taking thyroxine for some time before her death, of the order of months rather than days or even weeks,³² the experts were unable to find any *evidence* that she was hypothyroid as at May 2013 or between 20 May 2013 and 3 February 2014.³³ Nor was there sufficient evidence to say at what time Mrs Rose became clinically hypothyroid.
42. Prof Topliss' evidence was that if Mrs Rose stopped taking her daily prescribed thyroxine in late 2012 or in 2013, she would have progressively developed some signs and symptoms of hypothyroidism but the degree and tempo of that development would be the subject of speculation as it is very individual.³⁴
43. In evidence, Prof Topliss encapsulated the conclave's view as to the causal significance of untreated Hashimoto's disease to Mrs Rose's death when he testified that it was unlikely to have caused the death but was a possible contributing factor.³⁵
44. As regards myxoedema coma or crisis, an extreme manifestation of hypothyroidism³⁶, Prof Topliss testified on the conclave's behalf that it was considered extremely unlikely that this could explain Mrs Rose's illness, the changes in her mental state and the fatal outcome.
There are a series of clinical manifestations of severe hypothyroidism, including hypothermia

³⁰ Transcript page 10.

³¹ Thyroxine prescriptions usually provide for six months' supply. If Mrs Rose had no thyroxine (say) at home, the prescription dispensed in April 2012 would have yielded enough thyroxine for daily dosing until about October 2012, and perhaps later if she had some thyroxine at home. Significantly, Mr Rose's evidence is that after Ms Rose died, he found a quantity of thyroxine still in the fridge at home. Page 283 of the brief.

³² Transcript page 38-39. This was the evidence of Prof Topliss in answer to a question posed by me towards the end of the inquest with no demurrer from any of the other experts.

³³ Transcript page 12.

³⁴ Ibid.

³⁵ Transcript page 19.

³⁶ See Dr Young's explanation at paragraph 25 above.

and hypoventilation and a range of other symptoms, none of which were present apart from obtundation.³⁷

45. The conclave was asked a series of questions pertaining to Hashimoto's encephalopathy (HE) also referred to as steroid responsive encephalopathy associated with auto-immune thyroiditis (SREAT).
46. Prof Topliss testified that Mrs Rose clearly had an encephalopathy, and that her presentation and the progression of her symptoms was compatible with a diagnosis of HE/SREAT. The only identified factor that militated against the diagnosis was the normal electroencephalogram (EEG) undertaken during her illness, while her apparent response to steroid therapy with prednisolone, a glucocorticoid which is the accepted treatment for HE/SREAT, favoured the diagnosis. Moreover, a patient with HE/SREAT could be expected to improve clinically to such an extent that they could be discharged home with prednisolone or another steroid. Premature cessation of steroid therapy would lead to a deterioration.³⁸
47. Despite what more may be inferred from its very name, the expert evidence was that there is an association between Hashimoto's disease and HE/SREAT, rather than a direct causal relationship as, in the majority of reported cases, the patients were euthyroid at the time of presentation with HE/SREAT.
48. According to Prof Brew, although the pathophysiology of the condition is unequivocally unknown, there is "not an insignificant statistical association" between HE/SREAT and Hashimoto's disease, with approximately 25 per cent of patients having overt hypothyroidism. While he could not quantify the increased likelihood, Prof Brew testified that the fact that Mrs Rose had Hashimoto's thyroiditis increased her risk of having HE/SREAT. Although it had to be said that Hashimoto's thyroiditis is quite common in the community, while HE/SREAT is rare.³⁹
49. Prof Topliss testified that one could not say where there was this 'increased likelihood' in Mrs Rose. In his view, HE/SREAT was probably an autoimmune condition but beyond that one could not say that treatment of the thyroid dysfunction would influence the emergence of HE/SREAT.
50. Dr Walsh also offered an opinion on this issue, in stronger terms. His evidence was that thyroid hormone treatment would not alter the likelihood of a patient developing HE/SREAT

³⁷ Transcript page 14. I note that thyroxine (T4) is also the treatment for myxoedema, as well as triiodothyronine (T3) which is often used in myxoedema because of its rapidity of onset of action – see transcript pages 15-16 – and that no thyroxine was administered to Mrs Rose during either the February or the April admissions to BHH.

³⁸ Transcript pages 12-13.

³⁹ My paraphrase - Prof Brew's actual choice of words was "very, very, very uncommon" – transcript page 29.

as it is well recognised that it occurs in patients who have normal thyroid function and its occurrence is thought to be irrelevant to the level of thyroid hormone. Although the pathogenesis of HE/SREAT is not well understood, it is presumed to be an autoimmune process, primarily because it responds to steroid treatment. In his construct, the ‘association’ between Hashimoto’s disease and HE/SREAT was that a patient with one autoimmune condition, Hashimoto’s thyroiditis, develops another autoimmune condition, this time in the brain, because of a disorder of their immune system.⁴⁰

51. As to the causal significance of HE/SREAT in Mrs Rose’s death, the conclave of experts split neatly into two groups. Prof Brew, Dr Walsh and Dr Greenberg were of the view that HE/SREAT was both a probable cause and probable contributing factor in her death, whereas Prof Topliss, Dr Zavala and Dr Young expressed the view that it was (merely) a possible cause and a probable contributor in Mrs Rose’s death.
52. Apart from recognising Hashimoto’s disease and HE/SREAT as potentially causally relevant to Mrs Rose’s death, it is important to note the conclave of experts allowed of the possibility that some other unknown pathological entity was at play. They were however unable to identify what that might be, Prof Topliss saying “*We didn’t identify anything else. We found it difficult to speculate on an unknown, but we found no other specific illness identified...just to clarify. Did we consider there was an unknown illness, the answer was no.*”⁴¹

CLINICAL MANAGEMENT AND CARE

53. Apart from the medical cause of death, the other focus of the coronial investigation of Mrs Rose’s death, as opposed to the inquest, was the adequacy of the clinical management and care provided to her by the various general practitioners with whom she consulted at the Blackburn Medical Clinic and the clinical staff involved in her care during her two admission to Box Hill Hospital.
54. In the broadest terms, the potential for adverse comments or findings against any of the clinicians or institutions involved turned on their management of Mrs Rose’s Hashimoto’s disease in the period of the apparent deterioration in her health during the 18 months or so immediately preceding her death. Relevant statements and medical records were provided but none of this evidence was tested at inquest, consistent with my view that it was premature and/or inappropriate until elucidation of the medical cause of death might establish its forensic relevance.

⁴⁰ Transcript pages 30-31. Prof Topliss agreed with Dr Walsh’s evidence in this regard – transcript page 31.

⁴¹ Transcript pages 18-19.

55. In the circumstances, I do not propose to canvass that body of evidence. However, as a coroner's role includes contributing to a reduction in the number of preventable deaths and the promotion of public health and safety, I do consider it appropriate to observe that:

- a. Optimal management of Mrs Rose's Hashimoto's thyroiditis by the GPs at the Blackburn Medical Clinic, with reinforcement of the need to take thyroxine daily and regular blood tests to review the efficacy of treatment, would likely have improved her overall health.
- b. Similarly, Mrs Rose's two admissions to Box Hill Hospital provided an opportunity for full history-taking which would likely have elicited not only the diagnosis of Hashimoto's disease but importantly, whether or not she was still taking thyroxine and, if not, why not, with the potential for recommencement of thyroid replacement therapy and an improvement in her overall health.

FINDINGS/CONCLUSIONS

56. The standard of proof for coronial findings of fact is the civil standard of proof on the balance of probabilities, with the *Briginshaw* gloss or explications.⁴²

57. Adverse findings or comments against individuals or institutions are not to be made with the benefit of hindsight but only on the basis of what was known or should reasonably have been known or done at the time, and only where the evidence supports a finding that they departed materially from the standards of their profession and in so doing caused or contributed to the death under investigation.

58. Having applied the applicable standard of proof to the available evidence, I find that:

- a. The identity of the deceased is Sarah Louise Rose, born on 6 October 1973, aged 40, late of a Vermont address.
- b. Mrs Rose died at Box Hill Hospital, Nelson Road, Box Hill, on 15 April 2014.
- c. Initially, following full post-mortem examination or autopsy and ancillary investigations, the medical cause of Mrs Rose's death remained *unascertained* but there were findings that pointed to a potential contribution from Hashimoto's disease.
- d. The evidence now available to me does not enable me to determine the primary cause of Mrs Rose's death to the requisite standard, that is the antecedent cause, that without

⁴² *Briginshaw v Briginshaw* (1938) 60 C.L.R. 336 esp at 362-363. "The seriousness of an allegation made, the inherent unlikelihood of an occurrence of a given description, or the gravity of the consequences flowing from a particular finding, are considerations which must affect the answer to the question whether the issues had been proved to the reasonable satisfaction of the tribunal. In such matters "reasonable satisfaction" should not be produced by inexact proofs, indefinite testimony, or indirect inferences..."

which she would not have developed the cerebral oedema to which she ultimately succumbed.

- e. The conclave of experts expressed divided views on whether Hashimoto's encephalopathy was the cause of Mrs Rose's death, with Drs Brew, Walsh and Greenberg considering it a *probable* cause and Drs Topliss, Zavala and Young considering this merely *possible*.
- f. However, the consensus view of the conclave of experts, supports a finding that Hashimoto's encephalopathy was (at least) a *probable* contributor to death.
- g. The consensus view on the role played by Hashimoto's thyroiditis was that it was *unlikely* to have caused death but was a *possible* contributor to death.
- h. The conclave of experts also agreed in allowing of the possibility that another pathological entity had caused Mrs Rose's death, although despite their combined expertise they were unable to suggest what that might be.
- i. The formulation of the cause of Mrs Rose's death that best encapsulates the weight of the available evidence is cerebral oedema of unknown aetiology with contribution from Hashimoto's encephalopathy (or Steroid Responsive Encephalopathy Associated with Auto-immune Thyroiditis) in a woman with untreated Hashimoto's thyroiditis.
- j. As the available evidence does not enable me to determine the primary cause of death, it does not support an adverse comment or finding against any of the medical staff at Eastern Health involved in Mrs Rose's clinical management and care during her February and April admissions.
- k. For the same reason, the weight of the available evidence does not support an adverse comment or finding against any of the general practitioners at the Blackburn Clinic involved in Mrs Rose's clinical management and care following her diagnosis of Hashimoto's disease in late 2011.
- l. For completeness, I find that, for reasons I am unable to determine, Mrs Rose likely ceased taking thyroxine to treat her for Hashimoto's disease at a date I am unable to determine but that is likely to have been at least a number of months before her death.

PUBLICATION OF FINDING

59. Pursuant to section 73(1) of the Act, unless otherwise ordered by the coroner, the findings, comments and recommendations made following an inquest must be published on the internet in accordance with the rules. I make no such order.

DISTRIBUTION OF FINDING

60. I direct that a copy of this finding be provided to:

The family of Sarah Louise Rose c/o Maurice Blackburn

Dr St John, Dr Kreltzhaim, Dr Doukhan c/o Avant Law

Dr Nakajima c/o Ball & Partners

Dr Ratnarajah c/o Perry Maddocks & Trollope

Eastern Health c/o K & L Gates

Signature:



Paresa Antoniadis Spanos

Coroner

Date: 15 March 2021