CORONERS ACT, 2003

SOUTH  *AUSTRALIA*

FINDING OF INQUEST

An Inquest taken on behalf of our Sovereign Lady the Queen at Mount Gambier and Adelaide in the State of South Australia, on the 1st, 2nd, 3rd, 6th, 7th and 8th days of August 2012 and the 8th day of February 2013, by the Coroner's Court of the said State, constituted of Anthony Ernest Schapel, Deputy State Coroner, into the deaths of Angela Catherine Fensom and Andrew Johaanes Taankink.

The said Court finds that Angela Catherine Fensom aged 41 years, late of 15 Lane Street, Tantanoola, South Australia died at the Millicent Hospital, Mount Gambier Road, Millicent, South Australia on the 18th day of August 2009 as a result of influenza A (H1N1 2009, pandemic swine flu).

The said Court finds that Andrew Johaanes Taankink aged 26 years, late of 3/212 Commercial Street East, Mount Gambier, South Australia died at the Flinders Medical Centre, Flinders Drive, Bedford Park, South Australia on the 1st day of October 2009 as a result of hypoxic respiratory failure, H1N1 pneumonitis and acute respiratory distress syndrome.

The said Court finds that the circumstances of their deaths were as follows:

1. Introduction

1.1. These concurrent Inquests relate to the deaths of Angela Catherine Fensom aged 41 years and Andrew Taankink aged 26 years. The two deaths are unrelated except to the extent that both died of complications associated with influenza A H1N1 2009,

pandemic swine flu. The only other feature of commonality was that both Ms Fensom and Mr Taankink resided in the South East of the State. This had the consequence that, in the first instance, they were both admitted to rural hospitals in that region.

1.2. Ms Fensom was hospitalised and treated at the Millicent Hospital where she was declared deceased at 8pm on 18 August 2009. She had been admitted to the Millicent Hospital during the evening of the previous day.

1.3. Mr Taankink had originally been treated at the Mount Gambier Hospital. He had been admitted on 10 August 2009. In the early hours of the morning of 13 August 2009 he was retrieved to the Intensive Care Unit (ICU) of the Flinders Medical Centre (FMC). Mr Taankink would remain at FMC from that day forward. He died there on 1 October 2009.

1.4. Although Ms Fensom died of complications of swine flu, she was at no stage diagnosed with that illness prior to her death. Her cause of death was established at autopsy. Mr Taankink was diagnosed with H1N1 prior to his death, but not until after his eventual retrieval to the FMC ICU.

1.5. In this Inquest the Court examined the circumstances in which both Ms Fensom and Mr Taankink died and, in particular, whether the medical treatment that they received at the Millicent Hospital and the Mount Gambier Hospital respectively was optimal, the central question being whether the death in each case could have been prevented.

2. Influenza A H1N1 – 'swine flu'

2.1. References within these findings to 'H1N1' on the one hand and 'swine flu' on the other should be taken as references to the same viral illness.

2.2. Evidence concerning the incidence of swine flu in this country in 2009 was provided by Professor Anne-Maree Kelly who is Senior Emergency Physician and Academic Head of Emergency Medicine at Western Hospital in Footscray, Victoria. Professor Kelly is an experienced emergency medicine medical practitioner. She is a Fellow of the Australasian College for Emergency Medicine. She has more than 25 years' experience in the practice of emergency medicine and more than 20 years as a medical specialist in South Australia, New Zealand and Victoria. Professor Kelly

provided the Court with an expert overview in relation to the treatment of both Ms Fensom and Mr Taankink. She provided reports in relation to each person[1]. As well, Professor Kelly gave oral evidence in these Inquests.

2.3. Professor Kelly told the Court that the swine flu virus emerged in the world in early 2009. The World Health Organisation announced a health emergency with respect to the virus on 24 April of that year. The first Australian cases started to appear in Victoria at around early May 2009. South Australian cases began to emerge in early June 2009. Most cases in Australia occurred in the period June and July 2009. Professor Kelly told the Court that most of what is currently known about the virus and the illness that it causes has been gleaned from data and information published since the period with which this Inquest is concerned. Thus an element of caution is required when examining the standard of care provided to the two deceased in mid 2009 against what was understood about the virus at that time.

2.4. Professor Kelly explained that certain aspects of a person's general state of health might enhance the risk of the person experiencing life threatening complications of swine flu. Chronic asthma is regarded as such a risk factor. This is of particular relevance to Mr Taankink who was an asthmatic.

2.5. Professor Kelly told the Court that the most common complication of swine flu was severe pneumonia. This was the pathology that ultimately accounted for Mr Taankink's decline and death. It was obvious to clinicians at the Mt Gambier Hospital that Mr Taankink had contracted pneumonia, but it was not obvious that he had done so as a complication of swine flu.

2.6. Myocarditis, a disease of the heart, is also a possible complication, albeit a rare one. It was this complication that was responsible for Ms Fensom's death. I add here that there is no evidence that Ms Fensom had experienced heart disease as part of her previous medical history. Professor Kelly explained that it is quite common for the flu-like or cold-like symptoms of swine flu to have disappeared before cardiac related symptoms emerge. It is thought that it takes some time for the heart to be adversely affected[2]. Symptoms that could be expected in respect of myocarditis include tachycardia, which is a fast heart rate, and low blood pressure. Professor Kelly asserted that low blood pressure takes some time to develop, particularly in young fit

[1] Exhibits C24a (Taankink) and C24b (Fensom)
[2] Transcript, page 465

people who have a lot of reserve, so that even in the early stages of the heart muscle failing, different bodily mechanisms might nevertheless maintain blood pressure such that a significant decrease in blood pressure is a late finding in the illness. Thus persistent low blood pressure would be sign that would give rise to concern. There seems little doubt that myocarditis accounted for Ms Fensom's presentation throughout her admission at the Millicent Hospital. Myocarditis was not diagnosed at any time prior to Ms Fensom's death.

2.7. At the time with which these Inquests were concerned there was in existence an antiviral therapy known as Tamiflu. At the beginning of the swine flu pandemic it was not known with complete certainty how effective Tamiflu might be against the virus, but it had been shown to be effective against other strains of influenza A and was believed to be most effective if given within 48 hours of the onset of symptoms. The official guidance from the National Prescribing Service as at May 2009 was that Tamiflu should be instituted in patients with symptoms within 48 hours. In other situations it might be indicated on a case by case basis. Professor Kelly suggested that a senior clinician would be loathe to withhold the administration of Tamiflu in a severely ill patient merely on the basis that 48 hours had passed since the onset of symptoms. Professor Kelly suggested that it is now known that some patients treated outside the 48 hour window and who had significant disease did gain some benefit from the agent. In a Japanese group, the administration of Tamiflu assisted in the recovery from the complication of myocarditis. The efficacy of Tamiflu is a subject that has not been without its controversy, but the evidence in this Inquest demonstrated that there is wide-spread scientific belief that it does have efficacy in relation to the swine flu illness.

2.8. The provision of the antiviral Tamiflu medication, or more correctly the lack of it, is a pertinent consideration solely in the case of Mr Taankink. There is no real suggestion that in the case of Ms Fensom, and in light of the knowledge about the illness as it existed at that early time in its emergence, swine flu would have been such a readily identifiable differential diagnosis as to have dictated the administration of Tamiflu at any time before her death. The issue in the case of Ms Fensom is whether or not she received adequate clinical support having regard to a proper assessment and interpretation of her vital signs regardless of the true nature of her diagnosis. As will be seen, no positive diagnosis was made in respect of her condition prior to her death

except to the extent that it was believed, erroneously as it now transpires, that she had been suffering from gastroenteritis and, as a consequence, dehydration. Swine flu was only suspected as a possible differential diagnosis very late in the piece, and possibly only as an afterthought, but at no stage prior to her death was it confirmed. And, as alluded to earlier, the principal causative factor in her death, namely myocarditis, was not considered nor diagnosed. The question for consideration in Ms Fensom's case is whether different clinical management and support would have arrested her fatal decline such that an eventual accurate diagnosis of her condition could have been made and her condition effectively treated.

2.9. In the case of Mr Taankink, the question of proper clinical support is also a live one. There was no question but that Mr Taankink had a severe case of pneumonia. This was recognised at the Mt Gambier Hospital. His treatment there with antibiotics in the first instance was based upon a working assumption that the illness was of bacterial origin as opposed to a viral origin such as might be engendered by influenza. Mr Taankink had been prescribed Tamiflu by his general practitioner when seen on 6 August 2009. The medication was prescribed at a time at which he may have derived some therapeutic benefit. For reasons that are not entirely clear Mr Taankink did not fill the prescription. When he presented to the Mt Gambier Hospital later that same day, and indicated to doctors that he had not filled it, he was discharged. At one point a rapid antigen test, designed to detect swine flu but which had limited sensitivity and therefore reliability, was undertaken and it was negative, probably falsely so having regard to the fact that he would later be positively diagnosed with the virus. As with Ms Fensom's case, the principal issue in Mr Taankink's case is not so much related to precision of diagnosis but whether, when he was ultimately admitted to the Mt Gambier Hospital with pneumonia after a number of earlier presentations and discharges, his care had been optimal and specifically whether the ultimately devastating effects of his pneumonia could have been ameliorated by more timely delivery of intensive care thereby avoiding his death.

3. Cause and circumstances of the death of Ms Fensom

3.1. Ms Fensom was a married woman aged 41 years. She lived with her family at Tantanoola in the South East. Ms Fensom had no previous relevant medical history.

3.2. Ms Fensom was subjected to a post-mortem examination which was carried out by Dr John Gilbert, a forensic pathologist at Forensic Science South Australia. Dr Gilbert provided a report concerning his post-mortem examination of Ms Fensom to the Court[3]. In his report Dr Gilbert expresses Ms Fensom's cause of death as influenza A (H1N1 2009, pandemic swine flu). In the body of his report Dr Gilbert explains that a viral PCR study of a specimen of lung tissue sampled at autopsy detected influenza A virus, type H1N1 2009 (pandemic swine flu). Microscopic examination of the heart showed widespread but milder early changes in keeping with viral myocarditis. Dr Gilbert expresses the opinion that this pathology could account for Ms Fensom's terminal cardiac failure at the time of her death. The cut surfaces of both lungs were oedematous and congested which other evidence would suggest was consistent with pulmonary oedema that is in keeping with heart failure at the time of death. To my mind there is little doubt that Ms Fensom's terminal event was cardiac failure due to myocarditis which was a complication of swine flu. Mr Homburg of counsel for Doctor Veemarajah Vernugopolan, who was involved in Ms Fensom's clinical management at the Millicent Hospital, suggests that a more appropriately expressed cause of death would be 'fulminant myocarditis complicating Influenza A (H1N1) 2009'. While agreeing that this would also be an accurate description of the cause of death, in the sense that the ultimate mechanism of death was myocarditis, the underlying cause is nevertheless to be ascribed to swine flu. I see no need to depart from the cause as expressed in the official post mortem report. The cause of death in Ms Fensom's case can be described as influenza A (H1N1 2009, pandemic swine flu) which accords with Dr Gilbert's opinion. I find that to have been the cause of Ms Fensom's death.

3.3. Ms Fensom presented to the Millicent Medical Clinic, a local private medical practice, in the late afternoon of Monday 17 August 2009 which was the day before her death. She there saw Dr Veemarajah Vernugopalan who was one of a number of medical practitioners employed at the clinic. Dr Vernugopolan gave oral evidence in the Inquest. Dr Vernugopalan obtained his primary medical qualifications from the University of Tasmania in 2004. He completed a year of internship in 2005 at the Launceston General Hospital. He was a resident medical officer at Launceston in 2006. He was a hospital medical officer at the Royal Women's Hospital in Melbourne and then at the Mildura Base Hospital in 2007 and 2008. From February

[3] Exhibit C2a

2009 to January 2010 he was a general practitioner registrar at the Millicent Medical Clinic. Dr Vernugopalan was one of a number of medical practitioners in Millicent who took part in the on call roster at the Millicent Hospital. Although Dr Vernugopalan was a registrar, he had admitting rights to the Millicent Hospital and enjoyed virtual complete autonomy in respect of patients admitted under his care. As a reflection of this, Dr Vernugopalan was the only medical practitioner who examined or treated Ms Fensom until the late afternoon of the day of her death at which stage she was seen by another more experienced doctor within the Millicent Medical Clinic practice, a Dr Paul Angus who was a general practitioner anaesthetist. By then Ms Fensom was in extremis.

3.4. Dr Vernugopalan saw Ms Fensom at the Millicent clinic at approximately 6:20pm on 17 August. Ms Fensom described to Dr Vernugopalan a recent history of flu-like symptoms for 4 days together with loss of appetite and lethargy. The previous evening she had begun to experience diarrhoea, dry retching and nausea. Dr Vernugopalan examined Ms Fensom and found her to be pale and flat with a temperature of 38.9ºC, which is elevated (febrile), and to have a heart rate of 121 beats per minute which is also elevated (tachycardic). Dr Vernugopalan performed a capillary return examination that suggested that Ms Fensom was dehydrated and would require rehydration by way of intravenous fluid. This would require Ms Fensom to be admitted to the Millicent Hospital. In his oral evidence before the Court Dr Vernugopalan said that Ms Fensom displayed no respiratory symptoms at the time of his examination, although he has noted in the clinical record that her throat was red. Dr Vernugopalan formed an impression that Ms Fensom was suffering from viral gastroenteritis.

3.5. Ms Fensom made her own way to the nearby Millicent Hospital where she was seen by the nursing staff and where in due course Dr Vernugopalan saw her again. Dr Vernugopalan at that time made notes that included the earlier observations of Ms Fensom that he had made at his clinic. Dr Vernugopalan ordered a blood examination, the results of which would not become available until the following day. However, nothing turns on the results that ultimately would be revealed. As will be seen, there would be other worrying signs about Ms Fensom's well being quite apart from blood results and which ought to have dictated more aggressive clinical management at a time before the blood results became known. Dr Vernugopalan

ordered the administration of intravenous fluid. For this purpose he completed intravenous therapy orders that included the administration of a litre of normal saline to be given immediately over a period of 1 hour, followed by a further litre of normal saline to be administered over a period of 2 hours, to be followed by a litre of Hartmann's solution to be administered over a period of 6 hours and then a further litre of Hartmann's solution to be administered over a period of 8 hours. Upon her admission Ms Fensom's blood pressure was recorded as $^{108}/_{66}$ the systolic component of which for an otherwise healthy 41 year old female was at the lower end of normal. Panadol was given for her raised temperature.

3.6. Dr Vernugopalan again reviewed Ms Fensom briefly at 9:20pm. By then, as is apparent from the intravenous therapy order chart[4], Ms Fensom had received a litre of normal saline. This had been completed at 8:30pm. At the time of Dr Vernugopalan's review, Ms Fensom would have been in the process of receiving the second ordered litre. Dr Vernugopalan told the Court that at 9:20pm Ms Fensom had improved. She had more colour and had she said that she felt better. A note in the clinical record timed at 9:30pm[5] made by Registered Nurse Julie Hateley records that Ms Fensom had said that she was 'a bit better'. A note made by Ms Hateley timed at 10pm suggests that Ms Fensom was then feeling 'much better'. However, it is apparent that a blood pressure reading taken by nursing staff at 10pm was $^{81}/_{53}$ which is very low and would have been difficult to explain in the light of the earlier reading that had approached normality. There is no evidence that Dr Vernugopalan was made aware of this later blood pressure recording. He told the Court that as far as he could recall there had not been any further blood pressure readings taken between Ms Fensom's admission and when he saw her again at 9:20pm.

3.7. Ms Hateley, who also gave evidence in the Inquest, finished her shift later that evening. It had been determined that throughout the night Ms Fensom would be subjected to 4-hourly observations that would include vital signs such as temperature, pulse, respiratory rate and blood pressure. Ms Hateley gave evidence that the low blood pressure reading of $^{81}/_{53}$ had concerned her. She handed Ms Fensom over to the next shift and drew the low BP reading to the attention of the personnel for that shift and suggested that this would need to be watched. However, Ms Fensom, as is recorded, appeared to be feeling better. As well, her tachycardia and temperature had

[4] Exhibit C9, page 34
[5] Exhibit C9, page 9

settled and so looking at the complete picture, Ms Hateley took the view that Ms Fensom was improving despite the unexplained low blood pressure. At that point, as is true, the blood pressure reading was, as far as Ms Hateley was concerned, a '*one-off*'[6] and therefore not alarming in and of itself especially when considered in conjunction with the patient's improved clinical picture. However, other evidence would suggest that in cases such as this, where a blood pressure as low as the one recorded is consistently maintained as it would be throughout the course of the night and ensuing day in Ms Fensom's case, an improved clinical picture might tend to deceive. For example, Professor Kelly suggested that the ability of the patient to converse in a normal fashion and to remain conscious should not be relied upon as a marker of well being when other features of the person's presentation are a matter of concern.

3.8. After his 9:20pm review, Dr Vernugopalan would not see Ms Fensom again until the following morning. However, at approximately 2am he was telephoned by hospital nursing staff. Within the intervening period nursing staff had measured Ms Fensom's blood pressure as $^{75}/_{54}$ at midnight and $^{72}/_{46}$ at 2am. It will be noted that the systolic measurements are very low and lower than what the measurement had been at 10pm the previous evening[7]. A nursing note timed at 5am but which deals with events throughout the course of the night, and which were made at a time before Dr Vernugopalan would return to the hospital later that morning, is in the following terms:

> '18/8/09
> 0500 Nursing note – restless night, remains light-headed, no worse but no better. BP taken 2/24, remains low $^{72}/_{50}$, $^{75}/_{54}$, given a fluid bolus of 500mls saline over 2 hours with minimal results. Has voided on a pan x 2, small amount of diarrhoea, no nausea. Minimal oral intake tolerated.
>
> IVT continues at 166mls/hr
>
> RN Kimber' [8]

The two systolic blood pressure readings that are referred to in this note accord with those that I have already described as having been measured at midnight and 2am. The reference to a fluid bolus of 500mls saline over 2 hours was in accordance with an instruction given by Dr Vernugopalan over the phone during the 2am conversation.

[6] Transcript, page 91
[7] 'The 4-hourly observations contemplated by Ms Hateley had been converted to 2-hourly observations by 2am.'
[8] Exhibit C9, page 10

The description of a bolus of 500mls of saline over a period of hours is something of a contradiction in terms. A bolus of saline fluid is given rapidly rather than over an extended period of time such as 2 hours. In any event that was the order that was given and carried out.

3.9. Dr Vernugopalan gave evidence about the 2am phone conversation. He told the Court that he was advised by the nursing staff of '*a couple of low blood pressures*'[9]. In fact, Dr Vernugopolan acknowledged that he knew that one of the blood pressures was 72/50, which is very low[10]. He asserted in his evidence that he was not told, nor enquired about, the length of time over which these low blood pressures had persisted[11]. He said that he instructed the nursing staff to give Ms Fensom 'boluses'[12] which he regarded as the first measure that should be taken when people have low blood pressure. In his evidence Dr Vernugopalan suggested that the thought process behind the 2am boluses was a desire to see whether they would make an improvement in her blood pressure over time. However, he acknowledged that he did not specifically instruct the nurses to report the effects of the fluid on Ms Fensom's condition. Rather, he indicated that if there were any concerns or changes that they should call him again. He did not instruct the nurses to report back if there had been no change[13].

3.10. There are a number of matters that give rise to some concern in the light of what Dr Vernugopalan planned at 2am. Firstly, despite the fact that by 2am Ms Fensom had already received in excess of 2.5 litres of intravenous fluid, her blood pressure had dropped quite significantly. Secondly, a direction to nursing staff to the effect that he should be notified of a change would seem flawed when a lack of any alteration in Ms Fensom's low blood pressure despite the administration of a fluid 'bolus' would be a more significant and worrying finding. Thirdly, as at 2am Ms Fensom was being administered fluid at a rate of 166mls per hour in any event. Therefore a fluid bolus of 500mls of saline over 2 hours at the rate of 250ml per hour could hardly be characterised as aggressive fluid administration by way of bolus. Nevertheless, Dr Vernugopalan had seemed somewhat reassured by the fact that at 2am the patient was

[9] Transcript, page 33
[10] Transcript page 34
[11] Transcript page 34
[12] Transcript, page 33
[13] Transcript, page 51

still conscious '*which is a major thing*'[14]. In short, Dr Vernugopalan's evidence is that there was no specific concern conveyed to him by nursing staff in the course of this telephone call in the middle of the night and that he saw no other reason to be concerned.

3.11. It is difficult to see how Dr Vernugopalan could be reassured by the simple fact that the patient was still conscious. His assertion in this regard raises a question in the mind of the Court as to what it would have taken, short of the patient becoming unresponsive or otherwise being in extremis, for Dr Vernugopalan's own level of concern to have been excited. The evidence suggests that the blood pressure readings to 2am despite the giving of intravenous fluid were in reality alarming. As a measure of Dr Vernugopalan's inadequate appreciation of that fact and of his general insouciance, he asserted in his evidence that the low blood pressure readings recorded to 2am had by morning been '*corrected*'[15] with the administration of the fluid that he had ordered. In this respect he referred to the fact that when the blood pressure was tested at 6am it was $^{87}/_{53}$. This does represent an increase, but it does not represent an increase to a level that could be regarded in any sense as reassuring. Nor could it be viewed as a correction.

3.12. The nursing note compiled at 5am in respect of the position at 2am states that Ms Fensom remained 'lightheaded'. Dr Vernugopalan asserted in his evidence that he did not remember the nursing staff describing Ms Fensom as feeling lightheaded when they telephoned him. He said:

> 'I don't remember them saying that, no.' [16]

3.13. There was a question raised during the Inquest as to whether or not after the 500ml bolus over 2 hours as ordered by Dr Vernugopalan at 2am had been given, the administration of his original order was resumed. The suggestion, perhaps made only faintly, was that following the administration of the 500ml of fluid that would have concluded at 4:20am, no further fluid was given to Ms Fensom for several hours. I have taken into account other evidence that suggests that later in the morning there remained 500mls of fluid in a container that was not actually being administered and that this may have been the remainder of the 500mls that had been administered to

[14] Transcript, page 59
[15] Transcript, page 64
[16] Transcript, page 64

4:20am and that there was no evidence of any other fluid having been given after, or in addition to, that 500mls. I would reject the suggestion that the administration of fluid was interrupted because the nursing note compiled at 5am makes it very plain that the intravenous therapy continued after the administration of the 500ml bolus. The notes states 'IVT continues at 166mls/hr'. It will be noted that this is the rate over which a litre of fluid would be administered over 6 hours which is in precise accordance with Dr Vernugopalan's original order. I am satisfied from the nursing note and from the intravenous therapy order chart[17] that the first litre of Hartmann's solution was continued after 4:20am and that it concluded at about 6am. There is a further notation in the intravenous therapy chart that the remaining litre of Hartmann's solution that was ordered by Dr Vernugopalan was commenced at 6am.

3.14. The only other blood pressure reading taken during the course of the night occurred at 4am and it was then at $^{75}/_{46}$ which is in keeping with the blood pressure readings seen at midnight and 2am. In other words, the fluid bolus of 500mls had not made any difference at least as far as the patient's blood pressure is concerned. There is no direct evidence as to Ms Fensom's clinical state at 4am, except to say that, as seen, the nursing note indicated that she had remained 'lightheaded' overnight.

3.15. Dr Vernugopalan returned to the Millicent Hospital at about 8:25am the following morning. He made a note in the clinical record timed at 8:25am to the effect that Ms Fensom said that she had a restless night. She had felt better at one stage but now felt worse. She was still experiencing diarrhoea and had night sweats. On this examination Dr Vernugopalan observed that Ms Fensom was still flat. Although her temperature was unremarkable as was her pulse, as alluded to earlier Ms Fensom's blood pressure had been $^{87}/_{53}$ at 6am, the level that Dr Vernugopalan viewed as a correction and was for him only to be regarded as low '*in retrospect*'[18]. Dr Vernugopalan recorded his diagnostic impression as being one of gastro with a plan to continue with the administration of intravenous fluid and among other things to chase up the blood result from the previous evening. Dr Vernugopolan did not measure Ms Fensom's blood pressure at 8:25am which is another matter that limited the weight that should have been accorded to the isolated BP figure of 6am.

[17] Exhibit C9 page 34
[18] Transcript, page 38, line 14 and Transcript, page 39, line 16

3.16. Dr Vernugopalan would not review, see nor hear anything about Ms Fensom again until the late afternoon of that day by which time Ms Fensom was in extremis and dying. No other doctor saw her in that period.

3.17. In his evidence before the Court, Dr Vernugopalan explained his 8:25am plan as involving the administration of fluid and for the nurses to administer boluses of fluid if they felt that the blood pressure was too low, although he did not specify for these purposes what might be viewed as too low. However, given the nurses' level of experience, he expected that they would have either called him or given the fluid boluses at their discretion[19]. It is not precisely clear as to what was given to Ms Fensom by way of fluid administration from that point onwards. There is some suggestion that fluid was not given again until 11:30am when a further litre of saline was commenced. It was the belief of one nurse that there had been a significant interruption in the administration of fluid but I am satisfied that the interruption was not a significant one and one that did not in any way compromise Ms Fensom's management. By the morning of 18 August 2009 it is clear that no amount of fluid was going to alter the outcome or correct her low blood pressure. What Ms Fensom required by that point in time was an escalated level of care. The insignificance of any interruption in the giving of fluid is measured by the fact that the giving of fluid throughout the course of the night had little or no effect except marginally to raise the systolic blood pressure reading. As well, the administration of the litre of saline between 11:30am and 12:15pm, as well as administration of a further litre of saline over 2 hours from 12:15pm to 2:30pm in actual fact saw a decrease in her blood pressure from $^{84}/_{54}$ at 11:30am to $^{80}/_{56}$ at 2:15pm.

3.18. The reality was that despite fluid therapy, Ms Fensom's blood pressure remained significantly low and, worse, was unexplained. None of these developments were conveyed to Dr Vernugopalan or to any other medical practitioner. Dr Vernugopalan would not be contacted until the late afternoon. Manifestly he should have been contacted. The explanation as to why he was not contacted by nursing staff for the most part originates from a belief that Ms Fensom's low blood pressure was the result of dehydration coupled with an absence of any clinically concerning signs until much later in the afternoon. I have given anxious consideration to the role of the nursing staff in these unfortunate events. In viewing their performance one needs to take into

[19] Transcript, page 41

account the fact that Dr Vernugopalan did not identify to them what might have been viewed as a concerning pattern of low blood pressure sufficient to warrant him being contacted. In addition, when Dr Vernugopalan examined Ms Fensom at 8:25am, at which point it was known that Ms Fensom had experienced low blood pressure levels throughout the night, there was no reason to have expected his own low level of concern to have excited any meaningful level of concern on the part of nursing staff. In his own evidence Dr Vernugopalan did agree that in order to determine properly whether there was an improvement due to the giving of fluid, one would need to monitor the blood pressure as well, and that for this purpose he would need to know about further blood pressure results[20]. He also conceded in retrospect that this would mean that the blood pressure would have to be taken on a regular basis and that the result would have to be conveyed to him by nursing staff. Dr Vernugopalan said that he had not explicitly given any such instructions but assumed that if the nursing staff were worried about blood pressure they would call him[21]. It is difficult to see why that Dr Vernugopolan's concession made in hindsight could not have been the subject of sensible deduction and action on the day of these events.

3.19. In the event Dr Vernugopalan was not called until approximately 5pm when nursing staff became concerned about Ms Fensom, not so much based upon measurements of her vital signs, but upon worrying clinical observations that included pallor and sweating as well as difficulty with breathing. She had sounded extremely chesty according to Registered Nurse Jennifer Bertram. The truth is though that the earlier measurements of Ms Fensom's vital signs, specifically her very low blood pressures, were the portent of her clinical decline.

3.20. Ms Bertram who was employed at the Millicent Hospital commenced work at 2:45pm on the afternoon of 18 August 2009. She gave evidence. At the beginning of her shift Ms Bertram believed that Ms Fensom's diagnosis at that point was that she had gastro. Ms Fensom was Ms Bertram's allocated patient for the shift. When she first attended on Ms Fensom she observed that Ms Fensom was extremely flat and very tired. The time at that point was about 3:45pm. Ms Bertram took a faecal sample from Ms Fensom. At 4:45pm Ms Bertram became concerned about Ms Fensom. Her condition had changed and deteriorated. An hour earlier she had been communicating well, but by 4:45pm she was starting to grunt and she could hear crepitations

[20] Transcript, page 71
[21] Transcript, page 72

(crackles) and rattles in the chest. Her concern at that point was that Ms Fensom may have been experiencing fluid overload due to the amount of intravenous fluid that had been administered to her. At that time Ms Fensom's blood pressure was $^{150}/_{128}$ which was now elevated. The last previously recorded blood pressure was that at 2:15pm, namely $^{80}/_{56}$. On the observation record Ms Bertram noted that Ms Fensom was grunting, cold, pale and was clammy. Ms Bertram told me in her evidence before the Court that at no stage had she checked Ms Fensom's medical records prior to 4:45pm and that this had included her blood pressure levels[22]. As a result of her observations Ms Bertram telephoned Dr Vernugopalan who advised Ms Bertram to slow the administration of fluid and said that he would be at the hospital as soon as his clinic was concluded.

3.21. Ms Bertram told the Court that in her telephone conversation with Dr Vernugopalan she told him what Ms Fensom's blood pressure had been at that time. Dr Vernugopalan had asked her to '*keep an eye on it*'[23].

3.22. Dr Vernugopalan gave evidence about the 5pm telephone conversation with Ms Bertram. Dr Vernugopalan stated that, if anything, he derived some reassurance from this conversation to the extent that the blood pressure was now elevated and that Ms Fensom was still conscious and talking to the nurses. I have already commented upon the nonsensical derivation of reassurance from consciousness, having regard to the fact that anything short of that would have been alarming. In any event Dr Vernugopalan agreed that his only instruction was that the rate of fluid should be reduced. He agreed that he had indicated that he would see Ms Fensom at the conclusion of his clinical rounds. By about this time Dr Vernugopalan became aware of the results of the blood tests that had been conducted the previous evening. The results, which do not need to be recited in any detail, were such to make Dr Vernugopalan think that his original working diagnosis of viral gastroenteritis was less likely. He discussed the case with Dr Angus who was in the Clinic at that point and as a result of that Dr Angus went to the hospital to see Ms Fensom.

3.23. Dr Vernugopalan's sense of reassurance at 5pm has to be examined in the light of the fact that he made no enquiry about Ms Fensom's progress over the course of the hours since he had last examined her at 8:25am. In particular he did not ask about, and was

[22] Transcript, page 150
[23] Transcript, page 115

not told about, the blood pressure readings that had been taken during the course of the day which, despite the continued administration of fluid, had not improved and in fact had descended save an except for the sharp increase that had occurred within the last hour. Dr Vernugopalan said that if he had been told all of that, and knowing what he knows now, it would have identified a significant change in Ms Fensom's condition as one that had progressed from a state of hypotension to a state of hypertension. He would have endeavoured to have quickly sought assistance from someone more senior and gone over to the hospital immediately[24]. He also would have considered transfer to a hospital that could have provided greater care[25]. He would concede only in retrospect that by mid afternoon, having regard to the fact that there had been no improvement, would he have deduced that no amount of fluid was going to improve Ms Fensom's blood pressure[26]. In this context Dr Vernugopalan stated that he had no personal experience with the delivery of inotropic support of blood pressure which would have been indicated at that time. However, it is clear that Dr Paul Angus the general practitioner anaesthetist would have been able to handle any inotropic support that would have been required in the first instance and to have commenced its administration at the Millicent Hospital. Of course, Dr Angus had not been made aware of any of these events as they were unfolding until very late in the piece.

3.24. In accordance with Dr Vernugopalan's instructions, Ms Bertram reduced the flow rate of fluid and took further blood pressure readings including one at 5pm that reflected an increase in the systolic reading. By 5:30pm Ms Bertram observed that Ms Fensom had further deteriorated clinically. At that time her blood pressure had started to descend and was $^{126}/_{112}$. At 5:45pm it had further descended to $^{106}/_{76}$. Due to the descent in blood pressure, at that point Ms Bertram decided that there was a need to place Ms Fensom on a heart monitor and for this purpose she was moved into a different room. Dr Vernugopalan had not come to the hospital by then but Dr Angus entered the hospital at about that time.

3.25. Dr Angus gave evidence in the Inquest. Like Dr Vernugopalan, Dr Angus had only limited experience with the H1N1 virus, but after he had examined Ms Fensom, he at least included within her differential diagnosis that it was a possibility in her case. Dr

[24] Transcript, page 75
[25] Transcript, page 75
[26] Transcript, page 76

Angus did not know of any association between H1N1 and myocarditis at that point. This is no criticism of him, or of Dr Vernugopalan whose knowledge must have been the same.

3.26. Dr Angus had provided a statement to the police concerning his examination and management of Ms Fensom[27]. In his oral evidence before the Court Dr Angus was asked certain questions about the proper clinical approach and management that ought to have been instituted throughout the course of Ms Fensom's admission. He also gave evidence about changes in procedures relating to the management of his clinic's patients within the Millicent Hospital. I deal with those more general matters later.

3.27. Dr Angus told the Court that when he walked into Ms Fensom's room he was presented with a picture of shock and what he thought to be respiratory failure, the causative factor of which was unclear. In his witness statement Dr Angus describes Ms Fensom as clearly very unwell. At that point she was alert but agitated and had difficulty breathing. She was also pale and was in respiratory distress with crepitations in both lungs. She was cold and shutdown. Her blood pressure was at that point 70 systolic, taken by palpation. An ECG revealed sinus tachycardia. Her peripheral pulses were difficult to feel. Dr Angus called for immediate assistance from another on-call medical practitioner. Meanwhile he administered intravenous doses and then boluses of adrenalin. This was accompanied by a rise in blood pressure to 90 systolic but with no sustained benefit. An adrenalin infusion was commenced with little improvement. Dr Angus believed that Ms Fensom required intubation and transfer to an Intensive Care Unit in Adelaide. Further and larger adrenaline boluses with an increase in infusion rate did not result in restoring an acceptable blood pressure.

3.28. By this time Ms Fensom's husband had arrived at the hospital and it was explained to him that arrangements should be made for travel to Adelaide. Mr Fensom left the hospital at that point.

3.29. Although Ms Fensom was then intubated, a satisfactory heart rate could not be achieved and a pulse could not be detected. CPR commenced with further administration of adrenaline. However, there was no cardiac output and further

[27] Exhibit C17

resuscitative efforts were unable to prevent Ms Fensom's death. CPR was ceased at 8pm and her life was pronounced extinct.

4. The evidence of Professor Kelly regarding Ms Fensom

4.1. Professor Kelly provided an overview in relation to the management of Ms Fensom[28]. Professor Kelly stated in her oral evidence before the Court that given Ms Fensom's presentation at the Millicent Medical Centre on 17 August 2009 and then at the Millicent hospital, it was reasonable for Dr Vernugopalan not to have considered the H1N1 virus as a diagnosis. Furthermore, although in Professor Kelly's opinion Ms Fensom's presentation and decline, and in particular her persistent low blood pressure was likely due to the ultimate diagnosis of myocarditis, she would not have expected Dr Vernugopalan to have considered that as part of his working diagnosis. However, Professor Kelly expressed concern that Dr Vernugopalan had not adequately appreciated the significance of low blood pressure and of the lack of response to the therapy that he himself had devised overnight. As well, in her opinion blood pressure measurements had been too infrequent in the period during which Ms Fensom's blood pressure had remained at a level within the 70s or 80s systolic. It would be normal practice to take blood pressure readings at least every quarter or half an hour in order to identify whether there was rapid response to therapy[29]. An associated concern in Dr Kelly's opinion was that no specific instruction in respect of frequency of measurement of blood pressure and in relation to levels below which he should be alerted had been given to nursing staff by Dr Vernugopalan.

4.2. In her report regarding Ms Fensom[30], Professor Kelly points out that the persistent hypotension that Ms Fensom had been experiencing was an indicator of severe disease and that, irrespective of the cause, persistent hypotension requires close observation with continuous monitoring as opposed to periodic taking of vital signs as had occurred in this case. As well, an aggressive search for its cause and aggressive intervention to correct the problem was required. Specifically, she opines that more aggressive management should have been instituted from about 10pm onwards on the evening of 17 August 2009 when the blood pressure of 81 systolic was identified, with continuous monitoring, aggressive fluid therapy and onsite attendance by the

[28] Exhibit C24b
[29] Transcript, page 450
[30] Exhibit C24b

treating doctor. Failing more aggressive treatment from late on the evening of 17 August 2009, retrieval to Adelaide should have been initiated.

4.3. As to the mechanics of Ms Fensom's death, the physiological dangers of low blood pressure of these levels were that if the blood pressure remained low for a long period of time, oxygen and nutrients do not get to key organs, in particular the heart, kidneys, the liver and the brain which can result in irreversible damage. Ultimately the rattly chest that Ms Fensom experienced towards the late afternoon of 18 August 2009 was pulmonary oedema, not caused by fluid overload, but occurring as a reflection of the heart not working correctly. Professor Kelly regarded this development, as first observed by the nursing staff and reported at 5pm, as a sudden and serious change in Ms Fensom's condition which ideally should have prompted oxygen saturation measurements which would have confirmed that. This would have been regarded as a measure of how urgent things had become and would have been helpful in communicating appropriately with Dr Vernugopalan.

4.4. Professor Kelly made other observations in her oral evidence including (i) that even during the early stages of Ms Fensom's admission on the night of 17 August 2009 there were signs that she may have been in the early stages of shock at that point[31], (ii) that low blood pressure was quite uncommon in viral gastroenteritis, particularly blood pressures of the kind noted here[32], (iii) that at 2am on the morning of 18 August 2009 Ms Fensom was clearly seriously ill requiring reconsideration of her provisional diagnosis with the expectation that the doctor would attend the hospital to review her personally[33] and (iv) that during the early hours of the morning between 2am and 4am that Ms Fensom was severely hypotensive and that this should have signified to the clinician that Ms Fensom was now seriously ill and required escalation of care[34]. Professor Kelly stated as follows:

> 'I would have expected the doctor to have attended to review the patient, to perhaps have instigated other investigations looking for a cause different from the one that he had initially thought of and in the case of a doctor in a registrar training position, to have escalated the advice to include the senior doctor available to assist with the patient.' [35]

As well, Professor Kelly stated that by 8:30 on the morning of 18 August 2009:

[31] Transcript, page 443
[32] Transcript, page 446
[33] Transcript, page 446
[34] Transcript, page 447
[35] Transcript, page 448

> '… it was clear that she was not responding to the treatment and in fact had a persistent shock state and escalation of care was indicated at that time.' [36]

In cross-examination by Mr Homburg on behalf of Dr Vernugopalan, Professor Kelly expressed the opinion that inotropic therapy should probably have been commenced at about 8:30 on the morning of 18 August 2009, or at least that is when it should have been given consideration, especially given the lack of response to further boluses of fluid[37].

4.5. As to the significance or otherwise of Ms Fensom remaining conscious throughout, Professor Kelly said as follows:

> 'I'm actually quite concerned that there appears to be an assumption that altered conscious state is the marker here. A young person won't alter their conscious state until they are almost dead. It will be a very, very late finding and should not be relied upon in this circumstance, even with a very low blood pressure.' [38]

Similarly, Professor Kelly told the Court that false reassurance is sometimes derived from the fact that a patient does not appear to be clinically deteriorating, but where nonetheless the numbers relating to the patient's vital signs are deteriorating. She said that such a flawed approach '*has actually led to preventable deaths in a number of jurisdictions*'[39].

4.6. I have already referred to the evidence of Dr Angus concerning his examination and management of Ms Fensom on the late afternoon of 18 August 2009. It is fair to say that Dr Angus gave evidence of a general nature in respect of Ms Fensom's earlier treatment that was not inconsistent with that of Professor Kelly insofar as at 2am on 18 August 2009 he probably would have attended the hospital personally and reassessed the patient and that if there had not been any further improvement following further aggressive fluid administration, he would be considering inotropes. This would involve transfer to a more appropriate institution[40], that is to say other than the Millicent Hospital. Furthermore, Dr Angus agreed that there was nothing reassuring about the slight increase in blood pressure recorded at 6am when compared to the previous readings throughout the night[41]. Dr Angus also suggested that at 8:30am, although he would too have instigated further aggressive fluid management,

[36] Transcript, page 453
[37] Transcript, pages 474-475
[38] Transcript, page 479
[39] Transcript, page 481
[40] Transcript, pages 182-183
[41] Transcript, page 190

he would have planned a defined timeframe with a view to reviewing the observations and the response to that administration. We know in Ms Fensom's case that no meaningful plan was derived in that regard and no real account was taken of the lack of any response on Ms Fensom's part other than her clinical deterioration at a stage very late in the afternoon. Finally, Dr Angus suggested that Ms Fensom's was a case that was worthy of priority at the hospital on the morning of 18 August 2009. In this regard the following passage of evidence is pertinent:

> 'Q. But would you regard Ms Fensom's presentation on the morning of the 18th as a case that you would give priority to.
>
> A. Absolutely. I think the low blood pressures overnight and the fact that the blood pressure hasn't really responded to intravenous fluid would attract my attention.' [42]

I accept the evidence of both Professor Kelly and Doctor Angus.

5. Was Ms Fensom's death preventable?

5.1. In her report Professor Kelly states as follows:

> 'My concern regarding this case is the failure to adequately appreciate and respond to Mrs Fensom's persistent hypotension. Although this may not have altered the eventual outcome given the significant mortality of this condition even in specialist centres, it would likely have triggered treatment and referral which might have afforded Mrs Fensom a better chance of recovery.' [43]

5.2. In her oral evidence Professor Kelly elaborated upon this observation. Counsel assisting, Ms Kereru, asked Professor Kelly as to whether if Ms Fensom had been retrieved at some point during 18 August 2009 her prospects of survival would have been greater. In answer Professor Kelly pointed out that in 2009, H1N1 influenza had a significant mortality, particularly where the complication of myocarditis was involved. Nevertheless, she expressed the view that if Ms Fensom had been retrieved during the day of 18 August 2009 her chances of survival would have been higher, but with no guarantee that the outcome would be different[44]. In this regard Professor Kelly told the Court that a Japanese study had revealed that there had been a great number of survivors of myocarditis but they had all been managed within specialist centres with access to specialist ventilation support and external cardiac support. In cross-examination Professor Kelly reiterated that in many cases where myocarditis

[42] Transcript, page 195
[43] Exhibit C24b, page 5
[44] Transcript, page 452

was the complication of swine flu there had been full recovery[45]. When asked specifically as to whether intervention as late as 4:45pm on 18 August 2009 would have saved Ms Fensom, Professor Kelly stated that if she had been intubated and thereby have been properly oxygenated and the heart supported, it is possible that she might have survived, but by that time it was becoming very unlikely[46]. It should be noted here, however, that Professor Kelly was not suggesting that the time of 4:45pm on the 18 August 2009 was the appropriate time at which intervention should have been administered to Ms Fensom. Her evidence was that there were earlier opportunities that should have been taken to readdress Ms Fensom's management as already indicated in these findings.

6. Conclusions regarding the matter of the death of Ms Fensom

6.1. At approximately 8:00pm on 18 August 2009 Ms Angela Fensom, aged 41, died as a result of *influenza A (H1N1 2009, pandemic swine flu)*.

6.2. Neither swine flu nor its lethal complication, myocarditis, was at any stage diagnosed prior to Ms Fensom's death. That she remained so undiagnosed is not the subject of any criticism or due to any lack of clinical care.

6.3. Ms Fensom was admitted to the Millicent Hospital on the evening of 17 August 2009. It was suspected at all material times that Ms Fensom had been suffering from gastroenteritis which had required rehydration by way of the administration of fluid. However, it is clear and I so find that as at the time of Ms Fensom's admission to the Millicent Hospital, she was suffering from the effects of swine flu.

6.4. Ms Fensom continued to experience significant hypotension during the evening of 17 August 2009 and during the day of 18 August 2009. I find that her low blood pressures were reflective of myocarditis affecting her heart. This was not recognised by clinicians. That is not to say that the clinicians are to be criticised for not so recognising this fact. Rather, the persistent low blood pressures were attributed to dehydration.

[45] Transcript, page 464
[46] Transcript, page 469

6.5. Regardless of the perceived cause of Ms Fensom's very low blood pressure, the levels that persisted should have been regarded as alarming by her treating medical practitioner, Dr Vernugopalan. They were not so treated.

6.6. Ms Fensom's management within the Millicent Hospital was sub-optimal in that:

(i) There was inadequate oversight by Ms Fensom's treating medical practitioner, Dr Vernugopalan;

(ii) Dr Vernugopalan failed to recognise the seriousness of Ms Fensom's persistent low blood pressure;

(iii) Ms Fensom's persistent low blood pressure was the subject of inadequate support. Dr Vernugopalan failed to appreciate that the administration of intravenous fluid was having no meaningful therapeutic effect;

(iv) No or no adequate consideration was given to providing Ms Fensom with alternate support such as might have been provided by the administration of inotropes until a time when she was in extremis and in essence dying;

(v) Dr Vernugopalan placed too much reliance on the fact that Ms Fensom remained conscious virtually throughout her admission at the Millicent Hospital. This was a wholly inadequate measure of determining her actual wellbeing when regard was had to the alarmingly low and sustained blood pressures that she exhibited throughout the course of her admission;

(vi) More senior and experienced clinical input such as may have been provided by Dr Angus should have been given to Ms Fensom early on the morning of 18 August 2009.

(vii) No medical practitioner was contacted about or was asked to review Ms Fensom on 18 August 2009 until she was in extremis.

(viii) To his credit Dr Vernugopolan now accepts that Ms Fensom's persistent hypotension despite IV therapy should have caused him to review Ms Fensom's diagnosis and her management plan.

6.7. It is not possible to determine with complete precision whether or not if Ms Fensom had been provided with optimal support during the day of 18 August 2009 or had

been retrieved to a tertiary hospital she would have survived. However, it can be said and I so find that her chances of survival would have been greater in those circumstances.

7. Cause and circumstances of the death of Mr Taankink

7.1. The cause of Mr Taankink's death is taken from the Medical Practitioner's Deposition that forms part of the death report to the State Coroner compiled by a medical officer at the FMC[47]. The deposition explains the course of Mr Taankink's clinical management within FMC ICU. I set that out as follows:

> 'This 26 year old man presented to the Mount Gambier Hospital on 6/8/09 with fever, shortness of breath, cough and chest pain with red sputum. There, he received CPAP nebulised bronchodilators and antibiotics but despite these measures, his shortness of breath and hypoxia worsened. He was intubated on 12/8/09. He was transferred to the Flinders Medical Centre Intensive Care Unit via MedSTAR on 13/8/09 for further management. He was mechanically ventilated, given nitric oxide to improve his oxygenation and dialysed to maintain electrolyte and fluid balance. H1N1 PCRs were positive.
>
> Working diagnoses: Acute hypoxic respiratory failure
> Acute respiratory distress syndrome (ARDS)
> H1N1 viral infection with positive PCRs
> H1N1 pneumonia
>
> Over the course of his ICU admission, his oxygen requirement was high as the compliance of his lungs were poor. In addition, he required drainage of pleural fluid as he developed pleural effusions of his lungs. He developed a right pneumothorax and four chest drains were inserted. Despite this, his pneumothorax only improved marginally as shown on repeated chest x-rays. He received maximal medical management and airway supports but despite these interventions, his blood pressure and oxygen saturation steadily deteriorated. Andrew Taankink died on 1 October 2009 at 18:58 in this Intensive Care Unit.'

[47] Exhibit C25

The cause of death as expressed in the deposition is 'hypoxic respiratory failure, H1N1 pneumonitis, acute respiratory distress syndrome'. I accept all of that evidence. I find the cause of Mr Taankink's death to have been hypoxic respiratory failure, H1N1 pneumonitis and acute respiratory distress syndrome.

7.2. Mr Taankink was a single man of 26 years who lived with his parents in Mount Gambier. He had a past medical history of asthma and anxiety and had been treated for depression in the few years leading up to his death.

7.3. Mr Taankink had a number of presentations to the Mount Gambier Hospital prior to his eventual admission on 10 August 2009. These presentations occurred on 30 June, 1 July, 2 July, 6 August, 8 August and 9 August 2009. As well, he presented to a general practitioner in Mount Gambier on 6 August 2009 complaining of flu-like symptoms in respect of which Dr Jason Johnson prescribed Tamiflu. As alluded to earlier, the prescription was at no stage filled.

7.4. Professor Kelly provided to the Court her overview of Mr Taankink's clinical management. It is as well to discuss her views on the matter as I describe the course of events concerning Mr Taankink's illness. The presentations in June and July 2009 are in Professor Kelly's opinion not related to his final illness. However, the presentations are nevertheless relevant as they evince features of an exacerbation of asthma which was a known condition in Mr Taankink.

7.5. The earlier presentations in August 2009 together with Mr Taankink's management by his general practitioner on 6 August 2009 were also the subject of review by Professor Kelly who expresses the view, which I accept, that in each of these instances there was nothing other than reasonable medical management of a good standard. Specifically, there was nothing to suggest that Mr Taankink required hospital admission on any of the occasions on which he presented between 6 August and 10 August 2009, on which occasion it was quickly recognised that Mr Taankink was very unwell.

7.6. Although the serious manifestations of that illness only became evident on 10 August, there does not appear to be any doubt that on 6 August 2009 Mr Taankink was experiencing symptoms of what would ultimately be diagnosed as swine flu. In fact Professor Kelly expresses the opinion in her report that it is:

> '… likely that Mr Taankink's death might have been avoided if the script for Tamiflu written by his general practitioner had been able to be filled.' [48]

7.7. It is pertinent to enquire as to the circumstances in which Mr Taankink was examined on 6 August, both at the clinic of his general practitioner in the first instance and later at the Mt Gambier Hospital. The general practitioner, Dr Jason Johnson, was called to give oral evidence in the Inquest. He had earlier provided a statement to his solicitors dated 24 July 2012[49]. Dr Johnson practised at the Hawkins Medical Clinic in Mount Gambier. He was one of a number of practitioners at the clinic. This was not the first time Dr Johnson had seen Mr Taankink. Dr Johnson was aware of Mr Taankink's past history of asthma. Dr Johnson had in fact seen Mr Taankink on 9 July 2009 on which occasion Dr Johnson prescribed a Flixotide inhaler as part of an asthma management plan. A Flixotide inhaler is in fact an asthma preventer as distinct from an inhaler such as Ventolin which is used for acute attacks. This prescription implies that Mr Taankink's asthma was, to a degree, to be regarded as chronically problematic.

7.8. As indicated Dr Johnson saw Mr Taankink on 6 August 2009. He regarded Mr Taankink's presentation as a clear case of influenza. In fact in his statement Dr Johnson regarded Mr Taankink's presentation as striking in that his symptoms of fever, muscle ache, headache and fatigue were reported as having come on suddenly. He recorded that Mr Taankink's symptoms had occurred within the previous 48 hours. Accordingly, Dr Johnson prescribed Tamiflu and explained to Mr Taankink the pros and cons of this treatment. Specifically, he explained that it would be most effective if taken within the first 48 hours of when his symptoms started. He also explained that Tamiflu might reduce the risks of any serious complications of the flu. At the time with which this Inquest is concerned Tamiflu was not a PBS medication and Dr Johnson believed that at the time it cost something of the order of $70 to $100 for a 5 day course. The approximate cost of the medication was explained to Mr Taankink. Dr Johnson did not tell Mr Taankink that he was actually required to take the Tamiflu, but simply explained the possible benefits. Dr Johnson was mindful of the cost that it would have incurred to his patient. One of his reasons for prescribing Tamiflu in Mr Taankink's case was the latter's asthma which he believed could increase the risk of more serious complications of influenza. However, Dr Johnson

[48] Exhibit C24a, page 6
[49] Exhibit C18

did not believe that he had mentioned the possibility of swine flu to Mr Taankink and did not take a nasal swab from him to determine the strain of influenza that he believed he was suffering from. In his evidence, Dr Johnson accepted that he should have checked by way of testing whether or not Mr Taankink was suffering from swine flu if for no other reason than to determine whether or not it was now within his community, and also to establish the potential for serious complications[50]. Dr Johnson explained to the Court:

> 'I could of (sic) but I didn't consider it. I considered at the time that it was a clear case of influenza and I just gave him the best advice that I could and treatment that I could.' [51]

7.9. At the time Dr Johnson prescribed Tamiflu for Mr Taankink he had no reason to believe that it would be unavailable. It had never been drawn to his attention from any source that there was any general unavailability in Mt Gambier or specifically at individual pharmacies.

7.10. In the event Mr Taankink did not fill the prescription. That evening he would tell a doctor at the Mount Gambier Hospital that he had gone to a chemist to have the prescription filled but it had not been available. He did not identify the particular pharmacist.

7.11. Dr Johnson was at no stage made aware of the fact that Mr Taankink did not fill the prescription. I find that a discharge summary that was compiled by the doctor who would see Mr Taankink later that evening at the Mt Gambier Hospital and which was sent to the Hawkins Medical Clinic, was not drawn to Dr Johnson's attention. Mr Taankink's later presentations at the hospital were also not drawn to Dr Johnson's attention despite the sending of discharge letters to the Hawkins Medical Clinic. The absence of Dr Johnson's usual markings on such letters, that would have signified his receipt of them, was highly consistent with his evidence that he did not see these letters. It is clear that his practice received the letters, but there is no explanation as to why they were not drawn to his attention. I understood Dr Johnson's evidence to be that he heard nothing more about Mr Taankink following the 6 August 2009 consultation until shortly before his appearance in this Court when he was advised that Mr Taankink ultimately had died and that there was an ensuing coronial Inquest into his death. I accept Dr Johnson's evidence in this regard.

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[50] Transcript, page 239
[51] Transcript, page 209

7.12. Dr Johnson told the Court that if he had seen the discharge letter of the Mount Gambier Hospital presentation of 6 August 2009 it may have raised some concern in his mind as to whether Mr Taankink had obtained any Tamiflu or not. Secondly, he would have been concerned that Mr Taankink's symptoms were persisting and were serious enough to cause him to attend at the hospital[52]. When asked as to whether he would have taken any action in the light of the discharge letter, Dr Johnson suggested that while it was easy to speculate in hindsight, he may have called Mr Taankink to check whether he had taken the Tamiflu because he regarded it as important that he should take it within the first 48 hours of his symptoms. If it had been correct that Mr Taankink's reason for not obtaining the Tamiflu was that the pharmacy did not have any, Dr Johnson asserted that he would have taken steps in an endeavour to obtain it for him[53].

7.13. Similarly, if he had been made aware through a discharge letter that Mr Taankink had re-presented at the Mount Gambier Hospital on 8 August 2009, he would have arranged an appointment to see Mr Taankink to review him.

7.14. In his evidence before the Court, Dr Johnson expressed disappointment with the Mount Gambier Hospital in not having contacted him to inform him that they were not going to follow his recommended course of action regarding the administration of Tamiflu. He expressed disagreement with the notion that would later that day be entertained by the Mount Gambier Hospital clinician that Mr Taankink had not been in a high risk category. Although Mr Taankink may have been at low risk of contracting swine flu, Dr Johnson had acted on the basis that he was at high risk of complications of flu generally[54]. As things unfortunately were to transpire, Dr Johnson's caution about Mr Taankink was ultimately vindicated.

7.15. Enquiries made for the first time in mid 2012 as to the availability of Tamiflu in Mount Gambier in August 2009, and of Mr Taankink's possible attempts to obtain it, revealed that from time to time Mr Taankink had attended at Gambier City Pharmacy outlets situated at the Centro Shopping Centre at Helen Street, Sturt Street and Wehl Street North in Mount Gambier. These were not the only pharmacies in Mount Gambier. Mr Matthew Dixon who is the owner/operator of those three pharmacies

[52] Transcript, page 221
[53] Transcript, page 228
[54] Transcript, page 244

provided a statement to police on 27 July 2012[55]. Mr Dixon's statement reveals that Mr Taankink had attended at one of the pharmacies on 2 July 2009 but there was no record of any attendance between 6 and 9 August 2009. He points out that there would only be such a record in existence if Mr Taankink had a prescription filled at one of the pharmacies. Mr Dixon states that they no longer had records of their Tamiflu stocks as at August 2009, but states that if it had not been available at that time they would have done their best to source it from other pharmacies, from the hospital or from the wholesaler. In addition, they would have contacted the patient's medical practitioner and have advised him or her of the difficulty. Dr Johnson received no communication of that kind. Mr Dixon does say that he does not recall any issues of non-availability in 2009 in respect of his pharmacies. Given the high profile nature of the virus in 2009, one may have expected Mr Dixon to have remembered any significant shortage at that time. Mr Dixon points out that the cost of Tamiflu to the patient in August 2009 would have been in the vicinity of $45 to $50. This is less than what Dr Johnson believed to be the case, but it was nevertheless not an inexpensive medication to acquire. Other police inquiries made in mid 2012, as described in the statement of Detective Sergeant Anthony Scott of Mount Gambier CIB[56], do not reveal any known attempt on the part of Mr Taankink to obtain Tamiflu at pharmacies other than those operated by Mr Dixon. Other evidence of a general and anecdotal nature was to the effect that at this period there was not an overabundance of Tamiflu generally. However, evidence adduced from members of the Mount Gambier Hospital staff revealed that in all probability it would have been available at the hospital at that time.

7.16. It is difficult to be certain as to the reason why Mr Taankink did not obtain Tamiflu on 6 August 2009 at a time before he presented at the Mount Gambier Hospital that evening. It does not seem likely that there was a complete shortage of Tamiflu at pharmacies in Mount Gambier as this would be something that Mr Dixon would have been expected to recall. A competing explanation for why Mr Taankink did not fill the prescription on 6 August 2009 is that he was not prepared to part with the required funds. Another possibility is that Mr Taankink had simply not taken the opportunity to obtain it at that point. As will be seen, following Mr Taankink's consultation at Mount Gambier Hospital on the evening of 6 August 2009 it may well be the case that

[55] Exhibit C6a
[56] Exhibit C7a

he did not consider it worthwhile or necessary to obtain it. This brings me to that attendance.

7.17. Mr Taankink presented at the Emergency Department of the Mount Gambier Hospital at about 10:30pm on the evening of 6 August 2009. He was there seen by Dr Hussain Kajani who at that time was a medical officer employed in the Emergency Department of that hospital. Dr Kajani gave evidence in the Inquest. Dr Kajani recorded a history of a runny nose, fever, headaches and body aches with pain on swallowing. He took a previous medical history that included asthma for which Mr Taankink used Salbutamol as required. Dr Kajani had seen Mr Taankink earlier in the year on 30 June 2009 at which time Mr Taankink had complained of tightness in the chest and of feeling cold and sweaty. Dr Kajani had at that time formed a provisional diagnosis of an acute viral illness and asthma with a plan to provide him with nebulisation, Prednisolone and a further review.

7.18. Dr Kajani stated that although he did know of Mr Taankink's asthmatic condition and that he had been on Salbutamol, I accept his evidence that he did not know that Mr Taankink had been prescribed with the asthma preventer Flixotide and therefore did not know of any possible complication that might be engendered by a chronic as distinct from an acute asthmatic condition. He was aware that Mr Taankink was in possession of the script for Tamiflu and so he did not believe that his treatment would have been any different had he known about Mr Taankink being on an asthma preventer. As to the question of the Tamiflu prescription, Dr Kajani noted that Dr Johnson had prescribed it. However, according to Mr Taankink he had gone to a chemist and it had not been available. Dr Kajani then made the following note in the hospital clinical record:

> 'I explained to pt (*patient)* that he is not in a high risk category but it is up to him to continue Tamiflu. Swine flu info sheet given to pt from (RACGP site).' [57]

7.19. I have already commented about the different possibilities as to why Mr Taankink had not filled the prescription by the time he presented at the Mount Gambier Hospital. A general lack of availability in the region does not seem to be the likely explanation and it may well be that Mr Taanknink's explanation to Dr Kajani was borne out of embarrassment that the real reason may have been cost related. Dr Kajani's note rather suggests that he did not in any way actively encourage Mr Taankink to fill the

[57] Exhibit C11, page 23

prescription and indeed by saying that Mr Taankink was not in a high risk category may have unwittingly provided Mr Taankink with an excuse not to fill it. When Dr Kajani gave evidence he said that he did not know whether the hospital had a supply of Tamiflu at that time and asserted that the hospital had given instructions to administer Tamiflu only to patients who were at high risk of complications from swine flu. Dr Kajani did not feel that Mr Taankink was at high risk[58]. In his evidence Dr Kajani did not accept that what he had said to Mr Taankink had a tendency to make the latter think that it was not really necessary for him to take the Tamiflu[59]. Dr Kajani told the Court that it was his desire that Mr Taankink fill his prescription and that he had an expectation that he would do so[60] the following morning[61].

7.20. As to Dr Kajani's discharge letter to Dr Johnson, Dr Kajani accepted that it should have referred to the fact that Mr Taankink had not filled the Tamiflu prescription[62]. The Court agrees with this concession as it could have reactivated Dr Johnson's concern about Mr Taankink and have resulted in Dr Johnson taking steps to see that Mr Taankink was provided with it. Naturally the difficulty with that observation, however, is that Dr Johnson did not see the letter in any event.

7.21. It is difficult to determine from Dr Kajani's evidence the kind of impression he left with Mr Taankink as far as the need for him to obtain the Tamiflu was concerned. If Dr Kajani was keen for Mr Taankink to obtain it, the above note rather suggests that he left the entire issue to the whim of the patient and did not really give him any encouragement to do so. If Mr Taankink had been wavering about obtaining the Tamiflu because of cost, Dr Kajani's conversation with him may well have settled the matter and have caused Mr Taankink to lose all interest in obtaining it. It would have been better if a firmer arrangement or understanding had been arrived at for Mr Taankink to obtain what had been prescribed for him by Dr Johnson. Clearly the matter was one of patient choice, but the notion that Mr Taankink was given sufficient information make a reasonable decision about the matter of Tamiflu is not a convincing one.

7.22. Professor Kelly stated in her evidence that if on the night of 6 August 2009 at the Mount Gambier Hospital Dr Kajani had been aware that Mr Taankink's asthma had

[58] Transcript, page 422
[59] Transcript, page 431
[60] Transcript, page 413
[61] Transcript, page 424
[62] Transcript, page 425

involved the use of preventive medication as opposed to merely PRN Ventolin, he might have more effectively encouraged Mr Taankink to have his prescription for Tamiflu filled. That said, Professor Kelly herself doubted that the hospital would have provided Tamiflu as, in accordance with some hospital protocols in Victoria, Mr Taankink at that point was not ill enough to require admission to hospital. At that time she believed there had been some safeguards required on Tamiflu stocks with the focus for its use being in respect of people sick enough to require hospitalisation[63].

7.23. Like Dr Johnson, Dr Kajani did not perform any test for swine flu, although the question of swine flu had clearly entered his mind at the time of his consultation with Mr Taankink as evidenced by the fact that he gave Mr Taankink some literature about the viral illness. The first test for swine flu was administered to Mr Taankink on 8 August 2009 when he re-presented to the Mt Gambier Hospital.

7.24. Mr Taankink re-presented at the Mount Gambier Hospital on two further occasions, both of which culminated in discharge. These presentations occurred on the evening of 8 August 2009 and then in the late afternoon of 9 August 2009 on which occasion Mr Taankink actually arrived by ambulance. On the first of these occasions a rapid antigen test for influenza A and B was administered. The result of that was known immediately and it was negative. The PCR test for H1N1 was also conducted but the results were not available immediately and would only be made available after a number of days. Working differential diagnoses of influenza or glandular fever were made on this occasion. Mr Taankink was treated with, among other things, an antibiotic and given intravenous rehydration. He was discharged. On 9 August 2009 he presented with symptoms of vomiting and nausea with a sore throat and difficulty swallowing and a fever. On this occasion a clinical diagnosis of tonsillitis was made with antibiotics given. Mr Taankink was discharged for review by his general practitioner in 3 to 5 days. As indicated earlier, the reviewing expert, Professor Kelly, did not consider admission to the hospital as having been strongly indicated on either of these occasions. On 10 August 2009 Mr Taankink again presented to the Mount Gambier Hospital on which occasion he was quickly recognised as being very unwell. Admission to the High Dependency Unit (HDU) was arranged. Mr Taankink would remain in the Mount Gambier Hospital until he was transferred to the FMC ICU on 13

[63] Transcript, page 497

August 2009. He was not provided with Tamiflu at the Mt Gambier Hospital during this admission.

7.25. Although Professor Kelly was not critical of the delay in admitting Mr Taankink to the hospital prior to 10 August 2009, she is critical of his management in a number of respects once there, that include a failure to prescribe Tamiflu, a failure to recognise and manage ongoing severe hypoxia and a failure to consult earlier with a retrieval service for advice and possible retrieval on 11 August 2009.

7.26. As to the first of these concerns, Professor Kelly's opinion was that it was likely that Mr Taankink's death might have been avoided if the prescription for Tamiflu had been filled on the day that it was written by his general practitioner, or even the day after. In her oral evidence Professor Kelly suggested that the information that is available from studies conducted after the time of this incident would suggest that the incidence of pneumonia can be reduced by up to 80%[64] by the administration of Tamiflu. Thus, she opined, it was quite possible that the development of pneumonia in Mr Taankink could have been completely avoided[65]. Professor Kelly was less certain about the outcome if antiviral therapy had been instituted upon admission on 10 August 2009.

7.27. Nevertheless, Professor Kelly was of the opinion that it would have been advisable for Mr Taankink to have been given Tamiflu upon his admission on 10 August 2009 having regard to the fact that he had been hospitalised with a severe respiratory infection and had a history of a flu-like illness. Professor Kelly acknowledged that by that date the giving of Tamiflu would have been somewhat outside the usual range for its administration, namely 48 hours, but pointed out that the prescribing advice stated that under certain circumstances it might be appropriate to use the agent and that clinicians have usually taken that to refer to people ill enough to be admitted to hospital. She added the important rider that information at that time about swine flu was scant and that there had only been a few cases before this event. When specifically asked to comment on one suggestion raised in the evidence to the effect that the giving of Tamiflu at this juncture to someone with Mr Taankink's presentation would be completely useless, Professor Kelly pointed to the fact that there is research available suggesting that administration of the agent even after 2

[64] Transcript, page 494
[65] Transcript, page 495

weeks did have some effect on the development of the severity of pneumonia. However, in fairness she pointed out that at the time the decisions with which this Inquest is concerned were made, such 'hard evidence' was not available[66].

7.28. I turn to the question of Mr Taankink's clinical management. Mr Taankink's clinical condition during the days of his admission at the Mount Gambier Hospital was characterised by low and severely low oxygen saturation levels that were for the most part reflective of severe hypoxemia. Throughout the entirety of his admission he was administered oxygen by various means in an attempt to improve his oxygen saturation, but all with little therapeutic benefit. In the opinion of Professor Kelly, what Mr Taankink in reality required from an early point in time in his admission was intubation with mechanical administration of oxygen and retrieval to a tertiary institution. In the event, intubation and ventilation would not be administered until a retrieval team from the FMC attended late in the evening of 12 August 2009 by which time Mr Taankink had been admitted for over 48 hours. For the entirety of that period, his oxygen saturation levels were a matter, or should have been a matter, of some considerable concern.

7.29. Professor Kelly explained the various methods by which a patient might be artificially ventilated and supported with the administration of oxygen. The least effective delivery of oxygen is afforded by nasal prongs that direct oxygen into the nose. As well, there are a number of different types of mask which differ in the amount of oxygen that they can deliver and upon the amount of work the patient himself might have to do in order to secure its delivery. These masks include the Hudson mask as well as a non-rebreather mask. Another more effective method of delivery is by way of CPAP which involves the delivery of oxygen by way of a mask but which mechanically delivers oxygen into the patient's lungs with positive pressure and extracts carbon dioxide by the same method. This method is different from other methods of delivery in that the patient does not have to breathe for themselves. There is a disadvantage in that delivery of oxygen by way of CPAP can be very uncomfortable. Professor Kelly explained that a clinician might review CPAP within a certain period of time of its institution, gauge the patient's response within 15 to 30 minutes and then consider other alternative more invasive methods of delivery such as intubation. Intubation involves mechanical ventilation via a tube that is inserted into

[66] Transcript, page 508

the trachea. The patient is heavily sedated and does not have to breathe on his or her own. It was this method of delivery that would not be instituted until after 48 hours but which in the view of Professor Kelly should have been provided earlier. The evidence was that the necessary expertise to institute intubation and mechanical ventilation would have been available at the Mt Gambier Hospital.

7.30. Professor Kelly was of the view that there were in effect a number of missed opportunities to have arrested Mr Taankink's decline by more effective delivery of oxygen, particularly by way of intubation, and then retrieval to a more appropriate clinical setting that could be provided by tertiary institutions such as the FMC ICU to which he was ultimately sent.

7.31. Mr Taankink was admitted to the Mount Gambier Hospital under the bed card of Dr John Yamba who is a consultant physician. Dr Yamba underwent his basic medical training in Ghana and his specialist medical training in South Africa. He came to Australia in 2002. At the time with which this Inquest is concerned he was one of two consultant physicians who worked at the Mount Gambier Hospital. At that time one of those positions was temporarily filled by a locum physician, Dr David Pugsley. Dr Pugsley was at that time a retired physician. At one time he had been the Deputy Director of the Renal Unit at the Queen Elizabeth Hospital. Since his retirement Dr Pugsley has occasionally worked as a locum physician, principally in country hospitals. Both Dr Yamba and Dr Pugsley would have occasion to review Mr Taankink.

7.32. As the evidence in this case unfolded, there emerged an unedifying dispute as to which of the two physicians had been responsible for Mr Taankink's clinical management. Although Mr Taankink was admitted to the Mount Gambier Hospital under Dr Yamba's bed card, Dr Yamba did not regard Mr Taankink as coming under his primary care. He pointed out that Dr Pugsley was a considerably more experienced clinician then he[67]. For Dr Pugsley's part, in his evidence before the Court Dr Pugsley eschewed the suggestion that he was primarily responsible for Mr Taankink's care. He specifically rejected Dr Yamba's suggestion that he was in charge of Mr Taankink's care on 12 August 2009, on the evening of which Mr Taankink was transferred to the FMC ICU. He said that Dr Yamba was responsible.

[67] Transcript, page 258

7.33. Dr Yamba was first consulted by way of telephone on the evening of 10 August 2009. The plan for Mr Taankink that was devised would be administration with intravenous antibiotics and intravenous fluid, for him to be admitted to the High Dependency Unit of the Mt Gambier Hospital and for oxygenation to be instituted. Dr Yamba would not see Mr Taankink for himself, nor personally have anything further to do with Mr Taankink's management, until the morning of 12 August 2009. Dr Yamba told the Court that he did not work at the hospital on 11 August 2009.

7.34. The salient features of Mr Taankink's evaluation on 11 and 12 August are as follows. Dr Pugsley examined Mr Taankink at approximately 9:30am on 11 August 2009 during the medical team round. The examination took place in the presence of Dr Tauhid Kahn who at that time was a salaried medical officer at the Mount Gambier Hospital. Dr Khan himself examined Mr Taankink at 12:35pm that day after which he discussed Mr Taankink's presentation with Dr Pugsley. A decision was made on that occasion concerning the method of oxygen delivery. I return to that issue in more detail in a moment. Dr Pugsley did not examine Mr Taankink for himself again on 11 August 2009. However, it is clear from the clinical record that at 8:50pm that evening he was telephoned by nursing staff and advised of Mr Taankink's situation at that time. No changes in Mr Taankink's treatment were indicated during the course of this communication. When Mr Taankink showed some worrying signs of clinical deterioration in the early hours of the morning of 12 August 2009, as seen by a medical officer by the name of Dr Mangira, neither Dr Pugsley nor Dr Yamba were contacted. I will return to that as well. Dr Yamba examined Mr Taankink on the morning of 12 August 2009 and Drs Yamba and Pugsley examined him again at 2pm that same day. Ultimately Dr Yamba reviewed Mr Taankink again at 6pm at which time he also discussed the case for the first time with a senior registrar at FMC ICU.

7.35. During the entirety of the period that I have so far described Mr Taankink's oxygen saturations were at abnormally low levels and at times alarmingly so. Professor Kelly identified at least three occasions during this period when Mr Taankink's care could and should have been escalated and consideration been given to alternative measures of treatment. When Mr Taankink was examined by Dr Pugsley at 9:30am on 11 August 2009 Mr Taankink was receiving the maximum level of oxygen therapy through a non-rebreather mask and despite this, his oxygen levels were not improving and if anything worsening. At 7:45am his oxygen saturation level was 87% on 15

litres of oxygen. Professor Kelly suggested that in a young person such as Mr Taankink one would hope to see levels about 90%. She agreed with the proposition that anything falling below 90% was a '*red flag*'[68]. Professor Kelly expressed the opinion that given that Mr Taankink was receiving the maximum level of oxygen through the non-rebreather mask and having regard to what appears to be a downward trend in saturation levels, a reasonably emergent medical review was indicated to ensure that at that point that he was on the best and most appropriate form of oxygen delivery device. Such an assessment may have meant a need to escalate to CPAP non-invasive ventilation. Professor Kelly observed that at the 9:30am review by Dr Pugsley there appears to have been nothing specifically addressing the ventilatory issue and the ongoing hypoxia. Professor Kelly observed that Mr Taankink had not been responding to the oxygen therapy that was then being delivered. As far as Mr Taankink's clinical presentation is concerned it was noted at that time that he was short of breath with chest pain and taking deep breaths. The plan as recorded by Dr Pugsley was to observe Mr Taankink with reference to his blood pressure and oxygen saturations and to continue with the antibiotics.

7.36. Professor Kelly also identified Dr Khan's examination at 12:35pm as involving another red flag in respect of Mr Taankink's wellbeing and another opportunity to have considered a more appropriate mode of oxygen therapy and intensive care support. She opined that by 12:30pm things were becoming worse, not better. Dr Khan recorded that at 12:35pm Mr Taankink had oxygen saturations of 75% to 80% on room air. He was called to see the patient because Mr Taankink was no longer able to tolerate the mask. After discussion with Dr Pugsley it was decided that Mr Taankink should be administered oxygen by way of nasal prongs that can deliver 5 litres and, as the clinical note records, to then 'see how he goes'[69]. This naturally involved a de-escalation in oxygen delivery. In respect of this Professor Kelly had this to say:

> 'I would not have chosen that course of action. I don't think that enough oxygen concentration or flow can be delivered for a patient with Mr Taankink's condition.' [70]

7.37. It will be observed that at 1:30pm Mr Taankink's oxygen saturation was 82% which represented a decrease from earlier levels. Professor Kelly believes that at 12:30pm

[68] Transcript, page 510
[69] Exhibit C11, page 148
[70] Transcript, page 514

CPAP should have been started and thoughts about whether or not Mr Taankink continued to be appropriately cared for in the HDU of the Mount Gambier Hospital should have been entertained.

7.38. Professor Kelly gave evidence that at 12:30pm serious consideration should have been given to mechanically ventilating Mr Taankink with a view to transferring him. Although she expressed some doubt as to whether or not Mount Gambier Hospital had the facilities or expertise to intubate a patient for this purpose, Dr Yamba confirmed in his own evidence that they would have been able to intubate and ventilate a patient at the Mount Gambier Hospital prior to any transfer to a tertiary institution.

7.39. In the event, Mr Taankink's oxygen delivery continued to be maintained by way of nasal specs until sometime during the afternoon when he was once again ventilated by way of a non-rebreather mask. Following that, there were oxygen saturation levels as low as 77% on 15 litres of oxygen.

7.40. Mr Taankink would not be placed upon CPAP ventilation until the early hours of the following morning. In her evidence Professor Kelly observed that in the intervening period Mr Taankink's oxygen saturation levels did not improve and in fact were once again becoming worse. She suggested that intervention was indicated earlier than when it ultimately occurred.

7.41. From a clinical viewpoint, during this period it is recorded that Mr Taankink remained anxious and unsettled, was tachypnoeic, which means he was breathing rapidly, and was tachycardic, which means that his heart rate was elevated.

7.42. At 3:05am on 12 August 2009 Mr Taanknink was noted to be coughing up bright red bloodstained sputum with very shallow respirations and was tachycardic. He was still being ventilated by the non-rebreather mask at 15 litres per minute with an oxygen saturation at 82%. At 3:40am nursing staff called Dr Mangira. This appears to have been prompted by the observation that Mr Taankink's oxygen saturation had descended to 75% on 15 litres by way of the non-rebreather mask. This was all quite apart from the fact that Mr Taankink was obviously clinically unwell. Dr Mangira later made a note of this discussion with nursing staff that would find its way on to the progress notes. It is evident that Dr Mangira was informed that Mr Taankink was very anxious and still coughing up blood with increased chest pain, shortness of

breath and fever. It was ultimately recorded by Dr Mangira, through information that was imparted to him by nurses, that Mr Taankink was clinically in serious distress with worsening respiratory failure. In his evidence Dr Mangira told the Court that he had not believed that it had been necessary to involve a consultant physician at that point. He decided to alter Mr Taankink's oxygen delivery method and to commence him on CPAP and to take an arterial blood gas reading after one hour. He noted that his plan included the need to contact a consultant in the morning. Mr Taankink was commenced on CPAP at about 4:10am with maximum oxygen flow. This was all a commendable change for the better, but optimally it should have involved the input of a physician.

7.43. Professor Kelly expressed the strong view in her evidence, which I accept, that the decline in oxygen saturation levels to 75% on 15 litres of oxygen dictated the need for an urgent medical review at that time, preferably by a specialist who was capable of instituting advanced therapy[71]. While she was not critical of Dr Mangira's clinical course of action, she stated in her evidence that Mr Taankink was so severely ill at that stage that it was incumbent to have telephoned the specialist and to have asked that person to review the patient personally. In addition, although she noted that part of Dr Mangira's plan was to contact the consultant in the morning, it would have been much more preferable to have involved the specialist at the time of Mr Taankink's manifest deterioration. Moreover, the arterial blood gas that Dr Mangira ordered to be taken approximately an hour after the CPAP machine was instituted was worryingly low, such that there was a need to obtain the necessary staff to consider mechanical intubation[72]. As to the need for more urgent review at that particular juncture of the morning, and the need to bring in the necessary level of expertise, Professor Kelly had this to say:

> '…… it is actually a common thread that I have seen through several cases, like there is this reluctance to call people in the middle of the night. Again, going back to the question of recognition and recognition of the deterioration in the patient.' [73]

In her evidence Professor Kelly explained that the difficulty with the plan for a trial of an hour or two on CPAP is that it did not include a strategy to consider what one would do if the trial did not work. In particular, she suggested that one would have expected a significant oxygen saturation level of 90% or above. In the event this was

[71] Transcript, page 517
[72] Transcript, page 518
[73] Transcript, page 518

not achieved. When asked as to whether it should have been clear to clinicians on the morning of 12 August 2009 that Mr Taankink required intensive care treatment, Professor Kelly said as follows:

> 'At the very least at that time. I think it was clear, the evidence was clear a time before the morning of the 12th.' [74]

In that answer Professor Kelly is alluding to her stated view that there were opportunities on 11 August 2009 that should have been taken to consider intensive care treatment on that day.

7.44. Dr Mangira personally examined Mr Taankink at 5:30am. While Mr Taankink was tolerating CPAP well, his oxygen saturation was approximately 88%. He was tachycardic with a temperature and still experiencing chest pain. Dr Mangira's plan at that stage included continuing with CPAP and for Mr Taankink to be reviewed by the medical team in the morning.

7.45. The review occurred at approximately 8am and was conducted by Dr Yamba. Mr Taankink was still being ventilated by way of CPAP. Dr Yamba noted that Mr Taankink had experienced difficulty maintaining oxygenation. Upon a neurological examination Mr Taankink was alert and interactive with no focal deficiencies. In respect of this observation Dr Yamba told me in evidence that this was relevant in that if Mr Taankink was significantly hypoxic and his brain was not perfusing with enough oxygen, he would have been expected to have been confused and delirious, which he was not. His assessment was that Mr Taankink was suffering from multi-lobar pneumonia with Type 1 respiratory failure. Dr Yamba decided to alter Mr Taankink's antibiotic regime, to maintain him on CPAP and to perform an arterial blood gas after one hour. At the conclusion of that hour he noted that he would consider ventilation if there had been no further improvement. Dr Yamba gave evidence that he made a further assessment of Mr Taankink at 10:10am. A nursing note timed at 10am suggests that Dr Yamba himself inserted the arterial line and that at that point they were awaiting further arterial blood gas results.

7.46. A nursing note timed at 12:25pm records that Mr Taankink's oxygen saturations were now between 80% to 86% on CPAP. The latest arterial blood gas result involved a PO_2 of 47 and a PCO_2 of 42. It records that the nurse spoke to Dr Yamba and at that

[74] Transcript, page 518

point the patient was planned to have hourly arterial blood gas tests and that Dr Yamba would reassess Mr Taankink if there had been no improvement. It can be seen that this strategy appears to be at odds with Dr Yamba's original plan as devised at approximately 8am when he had noted that he would consider ventilation if there had been no further improvement. The fact of the matter was that there had been no significant improvement and that by 12:25pm that day Mr Taankink had remained on CPAP ventilation for some four and a half hours since Dr Yamba first devised his plan. In his evidence Dr Yamba stated that he believed that during the course of the morning he had spoken to Dr Pugsley about ventilating Mr Taankink. Dr Yamba could not recall what Dr Pugsley's view had been but Dr Yamba said in his evidence:

> 'I can't recall, but the patient was not ventilated so he must have had a different opinion.' [75]

7.47. Dr Pugsley who gave evidence told the Court that he believed that Dr Yamba was in charge of Mr Taankink on the morning of 12 August 2009. He rejected Dr Yamba's assertion that he, Dr Pugsley, had been in charge of the patient. I am uncertain as to whether or not Dr Pugsley was personally involved with Mr Taankink's management during the morning of 12 August 2009, but it is certain that at 2pm that day both he and Dr Yamba together reviewed Mr Taankink. Dr Pugsley said that as far as any earlier interaction with Dr Yamba is concerned, he could not recall any disagreement between himself and Dr Yamba about the possibility of intubating and ventilating Mr Taankink.

7.48. The joint review by Dr Yamba and Dr Pugsley at 2pm is the subject of a very short nursing note. The consensus appears to have been that they would continue Mr Taankink on CPAP with regular arterial blood gas assessments. In addition, he would be continued on intravenous antibiotics. This does not appear to have been a plan that was any different from that which had prevailed since 8am that morning. Furthermore, there had not been any discernible improvement in Mr Taankink. It will be recalled that at 12:25pm the nursing note suggested that there would be a reassessment if no improvement. There appears to be no evidence of any reassessment of significance at 2pm. Furthermore, it appears that for some period of time during 12 August 2009 CPAP had to be administered by way of a nurse holding the mask to Mr Taankink's face such was his level of intolerance. This in itself raises a question as to

[75] Transcript, page 288

whether or not intubation and mechanical ventilation should have been implemented for that reason alone.

7.49. In respect of the 2pm joint review Dr Yamba gave evidence that at that point Dr Pugsley was the primary carer for the patient. In order to support this contention he pointed to the fact that Dr Pugsley had considerably more experience as a clinician than he did [76]. On the other hand, Dr Pugsley said in his evidence that by this time Dr Yamba would have assumed control of the case clinically and that if he had wanted to discuss the case with him that this would have been appropriate. Dr Yamba's recollection, as stated in his evidence, was that at the review they had discussed the need for ventilation, meaning mechanical ventilation by way of intubation. Dr Yamba said:

> 'Why it was still not done at this stage, I do not recall the reason.' [77]

Throughout this morning and early afternoon, for the purposes of any decision that might have needed to be made to intubate Mr Taankink, Dr Yamba appears to have regarded himself as not the primary carer[78]. Although he agreed that the position at 12:25pm was such that a conversation concerning mechanical ventilation and intubation should have occurred, when questioned as to why that did not happen Dr Yamba replied that he was not the patient's primary carer. When, ultimately, a discussion did take place with Dr Pugsley, which must have occurred at around the time of the 2pm review, he said that he had voiced his concerns but that he had regarded Dr Pugsley as the more experienced clinician. When asked as to whether the failure to mechanically ventilate Mr Taankink at a time during the morning and afternoon was less than desirable treatment, Dr Yamba agreed. He suggested that that was a matter that had been Dr Pugsley's responsibility as he was the locum on for that day[79]. Dr Pugsley acknowledged in his evidence that earlier in the day at 8am Dr Yamba had been considering establishing mechanical ventilation in the event that there had been a failure to improve during the day. But he also agreed that as at 2pm there had been such a failure[80]. When questioned about whether at some point it came to him to consider alternative invasive delivery of oxygen, Dr Pugsley suggested that he was not specifically the clinician responsible to give that consideration. He said:

[76] Transcript, page 258
[77] Transcript, page 258
[78] Transcript, page 293
[79] Transcript, page 294
[80] Transcript, page 324

> 'Not me specifically. Whether - I'm not aware that, I mean Dr Yamba had that in his mind from the morning onwards and was looking to see whether a step-up of antibiotics to those that might have covered other pathogens and so on might have had some benefit, and I think he was waiting to see whether occurred and then decided at 6 o'clock that - on the 12th; that's on p.153 - that he had, there was a - we had reached a point where we could not expect to achieve anything more.' [81]

7.50. Regardless of whose patient Mr Taankink actually was, I accept and prefer the evidence of Professor Kelly that by 2pm it was clear that the patient required intubation and mechanical ventilation with a view to retrieval to a more appropriate clinical institution. Indeed her view was that it should have occurred the day before. I accept that opinion as well. It is difficult to determine why as late as 2pm on the 12th August this still had not occurred and that things, even then, were left to drift along for the rest of the day other than by reference to confusion as to who was in charge of the patient, or to a proper lack of dialogue between two experienced clinicians, either of which would amount to a very unsatisfying explanation to say the least.

7.51. In the event Dr Yamba reviewed Mr Taankink at 6pm. At that time Mr Taankink was breathing rapidly, was tachycardic and had experienced unsatisfactory and unchanging oxygen saturations in the 80s. Oxygen had continued to be delivered by way of CPAP during this period. Dr Yamba recorded that he spoke to the MedSTAR Retrieval Service who agreed to retrieve Mr Taankink. The advice that was given was that Mount Gambier Hospital clinicians should not intubate Mr Taankink until the arrival of the retrieval team and to continue with CPAP until that time. I do not need to discuss the appropriateness of that direction. Mr Taankink was ultimately intubated and ventilated at 9:45pm by the retrieval team. Mr Taankink was retrieved to the FMC ICU where he remained until 1 October 2009 on which day he died.

8. Was Mr Taankink's death preventable?

8.1. I have already referred to Professor Kelly's evidence concerning Mr Taankink's chances of survival had he been administered with Tamiflu.

8.2. In the opinion of Professor Kelly, the question of Mr Taankink's oxygen saturation levels as well as his clinically obvious breathing difficulties are matters that could have had some material bearing on the eventual outcome in Mr Taankink's case. She

[81] Transcript, page 325

explained that upon Mr Taankink's admission there had been a progression from a pattern of upper respiratory tract infection to one of a lower respiratory tract infection with a cough, shortness of breath and most importantly, low oxygen saturations of the order of 80% on air which is a serious concern[82]. In order to correct this, oxygen was administered at concentrations and amounts that in themselves implied serious unwellness[83]. Mr Taankink had pneumonia at that time and he was severely hypoxic with a high respiratory rate. This required the support of his oxygen saturation levels. Allowing them to be maintained at a less than satisfactory level involved chronic low delivery of oxygen to the tissues. This in turn can cause tissue damage that can affect the heart, the liver, the kidneys and the gut and can result in organ failure which eventually became an issue for Mr Taankink. In supporting the oxygenation it supports all of those tissues and at the same time provides the lung with an opportunity to heal until such time as the patient is able to breathe adequately for himself. In the event, Professor Kelly was of the view that Mr Taankink's decline and organ failure was likely to have been a combination of the effects of hypoxemia and the infection itself.

8.3. The question of whether or not Mr Taankink's death might have been avoided with more aggressive ventilatory management and earlier retrieval is a question that Professor Kelly addressed in her report in the following terms:

> 'There is a smaller possibility that Mr Taankink's death might have been avoided with more aggressive ventilatory management and earlier retrieval.' [84]

8.4. In her oral evidence Professor Kelly was asked to elaborate on her views. She was asked specifically whether if Mr Taankink had been intubated and mechanically ventilated at some point over the course of 11 August 2009 whether his prospects of survival would have improved. Professor Kelly said that it was difficult to say because cases of pneumonia and adult respiratory distress syndrome have a very high mortality, but certainly it would have given him an improved chance. [85] When also asked specifically about his chances at 2pm on 12 August 2009 at which time he was reviewed by Drs Yamba and Pugsley, her view was more guarded. She said that the writing was certainly on the wall by that time but that care should have been escalated.

[82] Transcript, page 504
[83] Transcript, page 505
[84] Exhibit C24a, page 6
[85] Transcript, page 520

8.5. Professor Kelly's evidence can be distilled into a proposition that in her opinion the management of Mr Taankink should have involved his ventilation and transfer approximately 24 hours before it eventually occurred, that is to say on the evening of 11 August 2009[86]. I accept that evidence. It seems, therefore, that there is some evidence to suggest that had that occurred, Mr Taankink's chances of survival may have been improved. However, as Professor Kelly herself said, it is difficult to be definitive about that issue due to the very high mortality in cases of pneumonia and adult respiratory distress syndrome.

9. Conclusions regarding the matter of the death of Mr Taankink

9.1. When Mr Taankink presented to his general practitioner, Dr Johnson on 6 August 2009, he was suffering from the effects of swine flu. Although this was not confirmed at that point in time, Dr Johnson prescribed Tamiflu for Mr Taankink. Mr Taankink did not fill the prescription for Tamiflu at any time. The reason why he did not fill the prescription has not been established with certainty. I am satisfied that the fact that Mr Taankink did not fill the prescription was at no stage made known to Dr Johnson.

9.2. When Mr Taankink presented at the Mt Gambier Hospital on the evening of 6 August 2009, he told Dr Kajani that he had not filled the prescription for Tamiflu because it had not been available at the chemist. Mr Taankink did not identify the pharmacy or pharmacies at which he had attempted to obtain Tamiflu. I am uncertain as to whether or not Mr Taankink had in fact attempted to obtain Tamiflu at a pharmacy in Mt Gambier. There is no evidence to suggest that there was total unavailability of the medication in the Mt Gambier region at that time. I think it more probable than not that Mr Taankink would ultimately have been able to obtain Tamiflu if he had been so inclined and had tried harder or his general practitioner Dr Johnson had been made aware of the fact that Mr Taankink had not sourced it.

9.3. When Dr Kajani examined Mr Taankink on 6 August 2009, he did not believe Mr Taankink to be in a high risk category for swine flu. He explained this to Mr Taankink and in addition said that it was up to him to obtain Tamiflu. In light of that, it is possible, but by no means certain, that any resolve that Mr Taankink may have

[86] Transcript, page 526

had to obtain Tamiflu was extinguished by this information. It would have been better if Dr Kajani had actively encouraged Mr Taankink to obtain Tamiflu.

9.4. If Mr Taankink had commenced a course of Tamiflu medication on 6 or 7 August 2009, it is possible that the development of pneumonia in Mr Taankink could have been completely avoided, thereby significantly improving his chances of survival.

9.5. I find that none of Mr Taankink's presentations to the Mt Gambier Hospital on 6, 8 or 9 August 2009 required his admission to hospital.

9.6. When Mr Taankink presented at the Mt Gambier Hospital on 10 August 2009, he was suffering from the effects of pnuemonia which in his case was a complication of swine flu. Mr Taankink remained as an admitted patient in the Mt Gambier Hospital until the early hours of 13 August 2009.

9.7. Mr Taankink's clinical management within Mt Gambier Hospital between 11 August 2009 and the evening of 12 August 2009 was sub-optimal. He continued to maintain very low oxygen saturations and was at times severely hypoxic. I find that Mr Taankink required intubation and mechanical ventilation at the latest by the evening of 11 August 2009 at which time consideration should also have been given to him being transferred to an intensive care facility at a tertiary hospital. Failing those measures being delivered to Mr Taankink that night, I find that during the morning of 12 August 2009, following Mr Taankink's deterioration during the early hours of that morning, he should have been intubated and mechanically ventilated at that time with a view to his transfer to an intensive care facility.

9.8. The cause of death of Mr Taankink is hypoxic respiratory failure, H1N1 pneumonitis and acute respiratory distress syndrome.

9.9. It is not possible to determine with certainty whether Mr Taankink's death would have been avoided with more aggressive ventilator management and earlier retrieval. However, I find that his chances of survival would have been enhanced.

10. Recommendations

10.1. Pursuant to Section 25(2) of the Coroners Act 2003 I am empowered to make recommendations that in the opinion of the Court might prevent, or reduce the

likelihood of, a recurrence of an event similar to the event that was the subject of the Inquest.

10.2. The Inquest into the death of Ms Fensom identified a general matter of concern. The prolonged absence of a medical practitioner at the site of a country hospital, such as that at Millicent, is a matter that is undesirable. It will be remembered that Dr Vernugopalan was not present at the hospital at any time between approximately 8:25am and late in the afternoon by which time Ms Fensom was in extremis. In the intervening period Ms Fensom was being cared for by nursing staff. It will also be recalled that Dr Vernugopalan was a Registrar and a relatively junior medical practitioner compared to say, Dr Paul Angus. Dr Angus in his evidence told the Court that following these events there had been a change in practices concerning the activities of Registrars and the manner in which their patients are admitted to the Millicent Hospital and managed therein. He told the Court that in 2010 the partners of the practice examined the system and had then implemented a new regime. The practice established the position of duty doctor being a practitioner who is available 24 hours at a time. The duty doctor manages each patient within the hospital with the exception of obstetrics and palliative patients. There is a handover process whereby every 24 hours a new duty doctor will attend and assume the care of each patient admitted in the hospital. In practice that means that the Registrars who share in the roster would be handing over their patients after 24 hours. In addition there is a plan in place where a practice partner will arrive at the hospital at about 1pm and conduct a ward round, or at the very least, discuss each patient with the Registrar. At the very minimum the status of an inpatient comes to the attention of a senior practitioner every 24 hours. This regime is reinforced by the requirement that there should be a minimum of one partner rostered for work each day, and that the practice ensures that a partner is always available to discuss the management of current inpatients. Dr Angus believed that the system that has been implemented is unique. It seems to the Court that the kind of regime described by Dr Angus is something that ought to be universally implemented in respect of practices and hospitals in country centres in general.

10.3. In her evidence, Professor Kelly referred to a criteria driven approach in respect of the detection and recognition of deteriorating patients such as Ms Fensom. In this context, Professor Kelly referred to the known reluctance of nursing staff and junior medical practitioners to telephone consultant physicians after hours, and in particular

in the middle of the night, with concerns that they may have regarding a deteriorating patient. Professor Kelly advocated the establishment of protocols that would automatically trigger the need for a consultant physician to be asked to review a patient when vital signs such as oxygen sats and blood pressures numerically descend below pre-identified levels. This would eliminate any discretion on the part of a nurse or junior practitioner who was worried about the patient, thereby relieving that person of any concern that they might have of unnecessarily troubling a more senior practitioner after hours. Mr Keane of counsel for the Department of Health tendered the affidavit of Ms Jayne Marie Downs[87] who is employed by Country Health South Australia Local Health Network ("CHSALHN") as the Director of South East Health Service Cluster based at the Mt Gambier and District Health Service. Ms Downs' affidavit describes a project that is being or has been implemented within CHSALHN that is designed to grapple with the issue of recognition of the deteriorating patient. The Millicent Hospital is one of the pilot sites for this project. The affidavit explains that the project fundamentally involves the implementation of a new colour coded observation chart supported by standard guidelines for timing of observations, types of observations to be undertaken, escalation systems and training. The clinically deteriorating patient observation chart will be used to guide staff in the recognition of a patient who may be at risk of deteriorating and to manage that patient appropriately. This will assist staff to recognise and respond to early signs of clinical deterioration. The project is a nationally recognised body of work and is supported by evidenced based practice and research. It seems to the Court that such a system is clearly desirable and one which would need to be implemented universally in respect of country hospitals especially.

10.4. I make the following <u>recommendations</u>:

1) That these Findings be drawn to the attention of the Minister for Health and to the attention of the Chief Executive Officers, or the equivalent, of all public hospitals in both the metropolitan area and country South Australia.

2) That these Findings be drawn to the attention of the Chief Executive Officer, or the equivalent, of the Australian Medical Association, South Australia branch, for the purpose of the education of its members.

3) That the Minister for Health cause systems and protocols to be designed to ensure that country hospitals in South Australia are staffed with appropriate medical

[87] Exhibit C26

expertise and to ensure that patients admitted to such hospitals are properly and regularly reviewed by a medical practitioner of appropriate and relevant experience.

4) That the Minister for Health ensure the promulgation within public hospitals in both the metropolitan area and country South Australia of systems and protocols designed to enable medical practitioners and nurses to recognise and appropriately respond to the deteriorating patient.

Key Words: Influenza A (H1N1), country hospitals

In witness whereof the said Coroner has hereunto set and subscribed his hand and Seal the 8th day of February, 2013.

Deputy State Coroner