CORONERS ACT, 2003

SOUTH  *AUSTRALIA*

FINDING OF INQUEST

An Inquest taken on behalf of our Sovereign Lady the Queen at Adelaide in the State of South Australia, on the 15th, 20th, 21st, 22nd. 28th and 29th days of October 2008, the 4th day of November 2008 and the 30th day of March 2009, by the Coroner's Court of the said State, constituted of Anthony Ernest Schapel, Deputy State Coroner, into the death of Brian Terrance Dalling, Jack William Salotti, Richard John Grzywacz and Brian Leslie Sobey.

The said Court finds that Brian Terrance Dalling aged 55 years, late of 7 Bayview Crescent, Corny Point, South Australia died at White Hut Road, Warooka, South Australia on the 30th day of December 2004 as a result of acute myocardial infarct due to coronary artery thrombosis secondary to coronary artery atherosclerosis.

The said Court finds that Jack William Salotti aged 51 years, late of Senior Road, Bordertown, South Australia died at Bordertown, South Australia on the 21st day of November 2005 as a result of ischaemic heart disease due to coronary atherosclerosis.

The said Court finds that Richard John Grzywacz aged 42 years, late of 1 Tonto Place, Port Lincoln, South Australia died at Port Lincoln, South Australia on the 20th day of March 2006 as a result of ischaemic heart disease.

The said Court finds that Brian Leslie Sobey aged 76 years, late of 1/15a Prince Street, Wallaroo, South Australia died at Wallaroo, South Australia on the 5th day of July 2006 as a result of ischaemic and hypertensive heart disease.

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

1. Introduction and reason for Inquest

1.1. The Inquests into these four deaths were heard concurrently. Each of the four deceased men died from heart disease. In each instance the deceased man had presented either at a country hospital or at a country medical practice complaining of symptoms that were the product of heart disease. In all cases medical practitioners examined the deceased men. The common feature of each man's death is that no definitive diagnosis of an acute coronary syndrome, succinctly summarised in the evidence as involving '*a heart attack in evolution*'[1], was made in each case and they consequently went without effective treatment. The men collapsed and died within a few days, and in one case within a few hours, of their respective presentations and examinations. In my opinion the evidence indicates that in each case the patient had presented with an acute coronary syndrome and that what was required in each case was urgent diagnostic angiography and transfer to a tertiary facility in Adelaide for that purpose. The issue in this Inquest, however, is whether in all of the circumstances their presentations were such that an acute coronary syndrome should have been identified in each case at the time of those presentations. In addition, the question is posed, are there measures in existence that should prevent or render less likely missed diagnoses such as these?

2. The post-mortem examinations and causes of death

2.1. The four deceased men were subjected to post-mortem examinations that included an autopsy in each case.

2.2. **Mr Brian Terrance Dalling** was 55 years of age when he died on 30 December 2004. He met his death when he suffered a heart attack at the wheel of his employer's truck. The truck veered off the road, travelled across a verge and then came to rest in the middle of a paddock. Mr Dalling's post-mortem examination was conducted by Dr Karen Riches, who at that time was a Forensic Pathology Registrar, and Professor Roger Byard who was the supervising Forensic Pathologist. The post-mortem examination revealed in their opinion that Mr Dalling's cause of death was acute

[1] Dr M B S Adams, Transcript, page 411

myocardial infarct due to coronary artery thrombosis secondary to coronary artery atherosclerosis. I find that to be the cause of Mr Dalling's death. The acute myocardial infarct was due to the occlusion of the left anterior descending coronary artery.

2.3. **Mr Jack William Salotti** was aged 51 years of age when he died on 21 November 2005. Mr Salotti collapsed at home. Resuscitation attempts by family members and ambulance officers were unsuccessful. Mr Salotti's remains were the subject of a post-mortem examination conducted by Dr John Gilbert, a Forensic Pathologist. The cause of death at post-mortem examination was found to be ischaemic heart disease due to coronary atherosclerosis. I find that to be the cause of Mr Salotti's death. Mr Salotti's heart disease was as a result of a narrowing of the diagonal branch of the left anterior descending coronary artery.

2.4. **Mr Richard John Grzywacz** was 42 years of age when he died on 20 March 2006. Mr Grzywacz was found dead at home by his wife. A post-mortem examination was conducted by Dr Barbara Koszyca, a Forensic Pathologist. The post mortem report expressed the cause of death as ischaemic heart disease. I find that to be the cause of Mr Grzywacz's death. At post-mortem examination there was evidence of severe stenosing atherosclerosis affecting two of the three major epicardial coronary arteries.

2.5. **Mr Brian Leslie Sobey** was 76 years of age when he died on 5 July 2006. On the evening of 4 July 2006 he experienced ongoing pain and in the early hours of the morning of 5 July 2006 he collapsed and died at home. Resuscitative attempts were unsuccessful. The post-mortem examination was performed by Dr Karen Heath[2], a Forensic Pathologist. Dr Heath's post-mortem report expresses the cause of death as ischaemic and hypertensive heart disease. I find that to be the cause of Mr Sobey's death. At autopsy Mr Sobey had an enlarged heart with moderate to focally severe coronary artery atherosclerosis. Acute plaque haemorrhage was identified in the mid portion of the right coronary artery.

2.6. I will later in these findings deal with the deaths of each of these men in more detail.

[2] Formerly Dr Karen Riches

3. Acute coronary syndrome

3.1. As seen above, pre-existing coronary artery disease was identified in each man at autopsy. This disease can lead to cardiac ischaemia, which essentially involves a deprivation of blood supply, and therefore oxygen, to the heart muscle known as the myocardium. This deprivation of blood supply to the heart can manifest itself in pain that is known as angina. The existence of angina does not necessarily signify an imminent and catastrophic cardiac event, but it is a sign of possible coronary artery disease and is all the more worrying if it is unstable and comes on at rest. A presentation of suspected unstable angina would in itself trigger certain detailed diagnostic investigations that would require the patient to be tested and observed over a period of time in a hospital setting.

3.2. More acutely, however, the deprivation of blood supply to the heart can give rise to a situation that was described in the evidence as an acute coronary syndrome. This implies that there is a substantial risk of an imminent heart attack. There are a number of characteristic features of an acute coronary syndrome. Not all of them will necessarily be present and they may vary in intensity. The classical symptoms include retrosternal discomfort going down one or both arms and possibly to the jaw or back, shortness of breath, sweating, nausea and a feeling of general unwellness. The discomfort in the chest is often a heavy oppressive feeling rather than a pain. One of the difficulties in diagnosing coronary artery disease is that presentations are often atypical in the sense that patients may have only one of these symptoms. As well, patients may have a heart attack without actually experiencing symptoms at the time, the so-called silent heart attack. They will go on with their lives in ignorance of the damage that has occurred to their heart. It was said in the evidence before me that people who suffer from diabetes commonly experience asymptomatic heart attacks. A related difficulty is that because the discomfort may be relatively mild, patients question whether or not they should actually be seeking medical attention[3]. This may especially be so where the patient over a long period of time has experienced symptoms that might in the normal course of events give rise to a suspicion of cardiac origin but which have been attributed to some other illness or pathology such as that associated with a gastric difficulty. This presents a dilemma for the clinician and may involve a difficult differential diagnosis. In this scenario, an experienced cardiologist

[3] Transcript, pages 269-270

told me in evidence that because a cardiac diagnosis is the most serious in terms of possible consequences, it is the diagnosis that needs to be considered and excluded first[4]. To this end there are sophisticated diagnostic protocols and services in existence that appear to have the underlying philosophy that in considering the possibility of a presentation of what is an acute coronary syndrome it is best to err on the side of caution and to assume that the patient's complaints of chest pain are cardiac related until it can be proved otherwise[5]. If these protocols and services are properly understood, adhered to and utilised, the evidence would to my mind suggest that missed identifications of acute coronary syndrome ought to be rare in any setting, rural or otherwise.

3.3. A myocardial infarction, or heart attack, occurs where the deprivation of the blood supply to the heart muscle is so acute and so profound that actual damage is caused to the heart muscle. It is common knowledge that this scenario constitutes a serious threat to the life and well being of the person. It can lead to a cardiac arrhythmia known as ventricular fibrillation which, if not reversed by say the administration of an electrical current, will cause the patient to die. Alternatively, the damage to the heart will be so extensive that irreversible cardiogenic shock will occur and the patient will die as a result of that. For obvious reasons a person who experiences a heart attack in hospital, especially a tertiary hospital, has a better chance of survival than if the person experiences it outside a hospital.

3.4. There are a number of factors that might pre-dispose a person to heart disease. These factors are well known and include smoking, obesity, high cholesterol, a family history of heart disease, poor diet, hypertension (elevated blood pressure) and diabetes. The age of the person is quite clearly also a relevant matter. The correlation between diabetes and heart disease is said to arise from the adverse effects of elevated blood sugar levels on body tissues. One of the manifestations of this is vascular disease or disease of the blood vessels including coronary artery disease. A history of diabetes would considerably increase the likelihood of a suspected cardiac complaint actually being cardiac origin. For reasons that are obvious the presence of one or more risk factors for heart disease in a presenting patient is a matter to be very much taken into account, together with their symptomatology, in considering whether or not the patient is presenting with an acute coronary syndrome.

[4] Dr W Heddle, Transcript, page 306
[5] Dr P A Tideman, Transcript, page 568

3.5. A previous heart attack in one's life increases the risk of further heart attack. A history of previous heart attack in a patient presenting with an already suspected acute coronary syndrome would raise the level of suspicion and would lower the threshold for triggering an investigation into the existence of the syndrome in the patient[6].

3.6. The evidence suggests that there are certain elements associated with living in the country that have put heart patients at a disadvantage. Many country centres do not have specialist cardiology services that are locally resident. Local hospitals tend not to have full-time medical staff but for the most part are staffed by local general practitioners who are on call for this purpose. The skill and confidence of local general practitioners to identify diagnostic electrocardiograph (ECG) abnormalities and changes varies from the good to the ordinary. Local hospitals are not equipped to administer invasive diagnostic measures such as angiography. Patients generally have to be transferred to one of the tertiary hospitals in Adelaide for that purpose. There is an understandable reluctance on the part of a doctor to organise what at times can be the difficult and time-consuming process of transferring a patient to Adelaide for invasive cardiology assessment if it is not shown to be absolutely necessary. The patients themselves may well have a corresponding reluctance to be so transferred. There are other difficulties, but these appear to be ones of relevance in the context of my inquiry.

4. The detection and diagnosis of patients presenting with angina or acute coronary syndrome

4.1. This was a matter that occupied much time during the course of this Inquest. I heard evidence from a number of sources in this regard. Dr Michael Adams, who is a general practitioner, was requested in the first instance to provide an expert overview of the circumstances surrounding the death of Mr Dalling. Before the Inquest commenced Dr Adams had provided two reports that were eventually tendered into evidence[7]. In addition, Dr Adams gave oral evidence. Dr Adams has made a number of general comments about diagnostic practices in respect of acute coronary syndrome from a general practitioner's point of view, having regard to the fact that in Mr Dalling's case, Mr Dalling was seen in a general practice environment. Dr Adams is an experienced general practitioner who has practised extensively both in Australia

[6] Dr M B S Adams, Transcript, pages 418-419
[7] Exhibit C26 and Exhibit C26a

and Canada. He obtained his original medical degrees in 1978 from the University of Adelaide and became a Fellow of the Royal Australian College of General Practitioners in 1993. He also has a Licentiate and a Certificate in relation to medicine, both of which he obtained in Canada.

4.2. Dr William Heddle is a cardiologist. Dr Heddle was asked to examine the circumstances surrounding the deaths of all four individuals with whom this Inquest is concerned. Dr Heddle produced four written reports to the Court[8] and also gave oral evidence in the Inquest. Dr Heddle was conferred with his medical degrees in 1972. He became a Fellow of the Royal Australasian College of Physicians in 1980 and he has a Doctorate in Medicine that was conferred in 1985. He has been practising as a cardiologist both within the Flinders Medical Centre and in private practice since 1982.

4.3. Dr Philip Tideman is also a cardiologist. He is the Senior Staff Cardiologist at Flinders Medical Centre and is the Clinical Director of the Integrated Cardiovascular Clinical Network in South Australia (iCCNet SA), formerly known as iCARNet. Dr Tideman was asked to prepare a report that dealt with the provision of cardiology services and advice to the medical profession and, in particular in the context of this case, to general practitioners especially those operating in rural areas. Dr Tideman also gave oral evidence during the Inquest. Dr Tideman did not provide any detailed overview in respect of the circumstances of each of these presentations and deaths, but commented in his report upon what he believed to have been the level of cardiology services that had existed in the four country locations with which this Inquest was concerned. Dr Tideman did give some general evidence concerning the presentation of Mr Dalling and the circumstances surrounding that matter when he came to give evidence. Dr Tideman obtained his primary medical degrees in 1985 from the University of Adelaide. He obtained a Fellowship of the Royal Australasian College of Physicians as a cardiologist in 1986.

4.4. In this section of my finding I deal with general matters not necessarily related to the specific deaths in question. Already outlined in the previous section are certain matters connected with the diagnosis of acute coronary syndrome such as relevant symptomatology and risk factors that need to be taken into account in any diagnostic setting. Quite apart from those very subjective investigative tools there are other

[8] Exhibit C32 re Mr Grzywacz, Exhibit C33 re Mr Salotti, Exhibit C34 re Mr Sobey and Exhibit C35 re Mr Sobey and Mr Dalling

diagnostic measures in existence that require, it seems to me, an element of rigour and discipline in their administration. I speak here of chest pain protocols that have been in existence for some time now, and in particular that promulgated by iCCNet, that set out certain diagnostic pathways in respect of presentations of suspected acute coronary syndrome or angina.

4.5. Before dealing with that issue it is appropriate to explain a number of diagnostic measures that were relevant in the examination of the four deceased persons upon their respective presentations. I speak here in particular of electrocardiographs (ECG) and a blood test known as a Troponin T test, hereinafter referred simply as the Troponin test.

4.6. An ECG is conducted by electrical recordings that in a 12 lead ECG emanate from 10 electrodes that are placed on the patient's arms, legs and the front of the chest. The ECG records, filters and amplifies the signals recorded from the body's surface which in turn reflect what is taking place internally within the heart. The ECG examines heart rhythm and is able to detect to a greater or lesser degree cardiac abnormalities such as ischaemia and acute myocardial infarction. Dr Heddle explained to me that in detecting abnormalities there is a reasonably wide variation of what might be considered normal. However, the beauty of the ECG lies in the fact that, somewhat like a person's fingerprints, an individual person has a characteristic electrocardiogram that, when repeated, should show those same characteristics. For example, Dr Heddle went so far as to say that a person could almost be identified by their ECG pattern. Because of the individuality of a person's ECG, it is always a worthwhile exercise comparing a person's present ECG with one from the past if a previous one is in existence. Moreover, changes between ECGs taken within a short time of each other also have diagnostic value. Dr Heddle explained to me, as did Dr Tideman, that a change from one ECG pattern to another is always of clinical significance. An ECG that is abnormal and then reverts to normal when the ECG is repeated is of as much diagnostic significance as an ECG that begins as normal and then proceeds to abnormal. This is due to the fact that in either scenario the change means that an acute event has taken place within the heart. The possible detection of changes and an evaluation of their significance is one of the underlying bases for the protocols that have been developed which rigorously insist upon the repetition of ECGs as a diagnostic measure.

4.7. As well, Dr Heddle gave me to understand that an individual ECG can in itself be diagnostic of an acute myocardial infarction, particularly when the ST segments of the trace are elevated to a certain degree. I do not need to explain in detail what an ST elevation is except to say that, while not all ST elevation is necessarily diagnostic, its presence or otherwise can be a matter of some diagnostic significance in determining whether a patient is experiencing an acute coronary syndrome or indeed an actual acute myocardial infarction. For example, in this particular case Mr Dalling, as long ago as October 1999, underwent an ECG examination that was conducted as part of a workup for a surgical procedure and, according to Dr Heddle's interpretation, it demonstrated at the time what he described as a gross abnormality, namely an old myocardial infarction or heart attack that had occurred some time before, possibly as recently as weeks ago. Dr Heddle told me in evidence that such a diagnosis could have been made simply on the basis of that single ECG and Dr Adams agreed with this analysis. I was also given to understand during the course of the evidence that there is a need for caution in respect of ECG interpretation. An ECG printout from the computer very often reports in words what the ECG trace might reveal. For example, it might report that the ECG is normal, abnormal or borderline. It might also report a myocardial infarction as it did in Mr Dalling's case in 1999. However, the written report is not always entirely accurate. Thus it is that diagnosticians, particularly general practitioners or medical practitioners in an emergency setting in a hospital, themselves require a certain level of expertise in interpreting ECG traces. The evidence would suggest to me, and I will come to the relevant cases in due course, that the quality of expertise amongst general practitioners is not always of a universally high standard. Dr Tideman told me that overseas trained doctors especially are over-represented among general practitioners who have low levels of confidence in ECG interpretation[9]. It will be noted here that three of the practitioners who saw the four men with whom this Inquest is concerned were trained abroad. The evidence also suggested that general practitioners may not have a full appreciation of the niceties of, and significance of, subtle but relevant changes from one ECG trace to a subsequent trace. Accordingly, an issue arises as to whether or not a scheme of further education for general practitioners in relation to the interpretation of ECGs might be indicated. This may be more acutely so in respect of rural general practitioners who do not have immediate access to local specialist advice or

[9] Transcript, page 515

assistance. The issue also gives rise to a need for general practitioners to avail themselves of the reasonably extensive cardiology resources that have been at their disposal for some considerable time now, for example the iCCNet service.

4.8. Dr Tideman told me that a person presenting either to a hospital or to a general practitioner with chest pain should have an ECG routinely performed within the first 10 minutes[10]. If there is a suspicion that the patient has a cardiac problem then the patient should have more than one ECG[11]. Dr Tideman was hard pressed to conceive of any circumstances in which a person presenting with chest pain, who had a suite of risk factors for heart disease, would not appropriately being given an ECG to begin with. There might be obvious cases where the chest pain is due, say, to trauma, particularly in a youthful person, but by and large the evidence persuaded me that there would be very few circumstances in which a presentation of chest pain would not be met with an immediate ECG, irrespective of whether the person presents to a general practitioner in the first instance or to a hospital. In fact the iCCNet chest pain protocol that I will discuss in due course requires as much. Dr Tideman was asked about a scenario that involved a patient presenting to a general practitioner with chest and jaw pain and who was expressing some concern about it being cardiac related, and whether in those circumstances there would be any justification to depart from the guideline that suggests that an ECG ought to be performed immediately. He said:

> 'I can't think of any circumstance where one would justify not doing an ECG in that circumstance. In fact of course if the chest pain was reported as having occurred some hours or importantly some days distant from the presentation, even then one would find it hard not to justify doing an ECG in that circumstance, particularly if the patient expresses concern about a potential cardiac cause, I just cannot imagine any circumstances in which you would not do an ECG.' [12]

The scenario in question there was in very broad terms that involved in Mr Dalling's presentation, although in Mr Dalling's case there were a number of risk factors on top of his symptomatology. Complicating Mr Dalling's presentation, as we will see, was a theoretical alternative explanation for the pain. However, the general comment that Dr Tideman made in my view holds good, namely that it would only be in exceptional circumstances that a person presenting with chest pain would not have an immediate ECG.

[10] Transcript, page 501
[11] Transcript, page 503
[12] Transcript, pages 536-537

4.9. It should be said in respect of the utility of ECGs that an acute coronary syndrome will not necessarily be picked up on a first or even subsequent ECG. As a diagnostic measure, therefore, it has some limitations.

4.10. The Troponin test is a blood test. In modern times it can be conducted and analysed by way of a bedside, or 'point of care', machine. The traditional method of analysis of a blood sample taken from the patient involves scientific testing by one of the well-known pathological services such as the IMVS. Either way the result is usually known quite quickly. The test is designed to detect and measure, within certain limitations, the existence of Troponin in the blood stream. The existence of Troponin in the blood stream can be the reflection of myocardial damage caused by an acute infarction or heart attack. A positive Troponin level may well be a reflection of other pathology, but when correlated with symptoms that are suggestive of an acute coronary syndrome, such as chest pain, the test has a large measure of diagnostic utility. The difficulty with the Troponin test is that the myocardial damage from a heart attack is usually only detectable within the bloodstream after about 6 to 8 hours since the onset of the myocardial damage. In other words, if a person were to suffer an acute myocardial infarction, evidence of it would generally only be detected by way of the Troponin test after that many hours. However, the myocardial damage will be reflected in the bloodstream for several days afterwards. Thus it is that the Troponin test still has excellent utility for quite some time after the myocardial infarction has taken place and its utility as a diagnostic tool is not confined to the occasion of the myocardial infarction itself.

4.11. Because evidence of myocardial infarction is usually only detectable after a certain number of hours, Troponin tests should be repeated. The necessity to repeat Troponin tests is also reflected in the chest pain and acute coronary syndrome protocol.

4.12. A negative Troponin test does not necessarily exclude the possibility that a patient is having cardiac difficulty. In an acute coronary syndrome, where there is a blockage of the artery that might in due course lead to the formation of a life-threatening thrombosis, Dr Tideman told me that for a variety of reasons there are a certain percentage of patients in whom the blood test will remain normal. However, Dr Tideman told me that if a patient were observed for 8 hours or more, then around 98%

of affected patients would have a positive Troponin test[13]. Dr Tideman also told me that if a patient is run along a clinical diagnostic pathway that is specifically designed to detect patients who are experiencing an acute coronary syndrome by means of ECG and Troponin testing in combination, the chances of not detecting a patient with an acute coronary syndrome in an 8 hour period is probably in the order of only 1%. On that basis, a failure to detect a patient presenting with an acute coronary syndrome and who has been subjected to the appropriate protocol or clinical pathway should be rare.

4.13. As I have foreshadowed, there is in existence a protocol described as 'Management of Chest Pain / Suspected Acute Coronary Syndrome'. This was promulgated by a South Australian non-profit and government funded entity that was originally known as iCARNet, the precursor to iCCNet. The protocol is based on the 'National Heart Foundation of Australia, Cardiac Society of Australia and New Zealand Guidelines for the management of acute coronary syndromes' which is in booklet format. Tendered to me in evidence is what I understand to be the current version of the protocol document which is copyrighted in the year 2006[14]. On the one sheet, the document sets out three clinical pathways for the investigation of chest pain and suspected acute coronary syndrome. The single sheet can be reproduced in poster or card format. The chosen diagnostic pathway depends upon the level of risk that has been identified in respect of a particular patient. There are three such protocols or pathways, namely the Low Risk Protocol, the Unstable Angina/Non-ST Elevation Protocol and the ST Elevation MI (myocardial infarction) Protocol. The three protocols set out the various diagnostic steps that ought to be embarked upon depending upon the identified level of risk of the patient and the patient's presenting symptoms. Also tendered in evidence were three documents that individually reflect the operation of the three protocols. They were tendered as a bundle[15]. The copyright in respect of each document is said to be of that of iCARNet dated either in April 2002 or April 2003 as the case may be. It is clear to me that this protocol has been in existence for several years and was in existence at the time of the deaths with which these individual Inquests are concerned. While at first blush these documents appear to be daunting, I have little doubt that to the trained medical practitioner they would be relatively easy to digest.

[13] Transcript, page 499
[14] Exhibit C39
[15] Exhibit C38

4.14. Although it is not necessary here to describe each protocol in intricate detail, it is clear that even in the case of a low risk identification, the patient will be subjected to repeat ECG and Troponin testing before consideration is given to discharging the patient. The Low Risk Protocol appears simply to be triggered by a presentation with symptoms suggestive of acute coronary syndrome and this would include, of course, chest pain. I did not understand that the Low Risk Protocol would, of necessity, be triggered by the full gamut of symptoms. It would only be in exceptional circumstances that a person presenting with chest pain would not be subjected at least to the Low Risk Protocol, and as Dr Tideman indicated, this would be confined to circumstances in which there was a clear alternative explanation for the chest pain other than suspected acute coronary syndrome. Dr Tideman suggested that in the case of a patient presenting with, say, chest and jaw pain but who might have other competing explanations for that other than cardiac related, the general practitioner would only send the patient away where the practitioner could reasonably confidently invoke an alternative diagnosis. Such an alternative diagnosis would require some supporting evidence in terms of the patient's history and examination findings[16]. Dr Tideman gave examples of chest pain that might be so alternatively explained such as pain associated with tenderness of the chest wall, or history of trauma. It was in this context that Dr Tideman said that one has to assume that the patient's complaint of chest pain is of cardiac origin until one can prove otherwise. A complaint of chest pain without positive ECG or Troponin testing, unless explained by something other than cardiac origin, would invite non-invasive examination such as exercise stress testing.

4.15. The ECG test and the Troponin test are independent diagnostic measures. I accept the evidence of Dr Heddle that either a positive ECG or a positive Troponin test in combination with the patient's presentation of chest pain would be sufficient to base a conclusion that an acute coronary syndrome was at work[17].

4.16. Dr Tideman sets out a number of services that have been provided by iCCNet since its inception. I have already referred to the clinical pathways for Management of Chest Pain/Suspected Acute Coronary Syndrome. In addition to that, the entity provides a 24-hour, 7 day per week telephone access to consultant cardiology advice and ECG interpretation with access time of less than 10 minutes. Traditionally, country doctors

[16] Transcript, page 567
[17] Transcript, page 325

have accessed advice on cardiac patients through teaching hospital on-call medical or cardiology registrars or by ad hoc relationships with individual specialists. It will be seen that one or more of the medical practitioners in the present cases were, for the most part, reliant upon those relationships. Dr Tideman reports that extensive liaison and needs analysis conducted with rural general practitioners indicated that this system does not meet their requirements, either in terms of advice or arranging access to tertiary care for patients. However, iCCNet uses only consultant cardiologists to provide clinical advice and ECG interpretation. Dr Tideman claims, and I have no doubt this is the case, that the system provides improved accuracy, pertinence, immediacy, comprehensiveness and continuity of advice and triage decision making. A sophisticated contact system for country practitioners has been established by iCCNet by way of a single telephone contact number to a centralised paging service. It is said that under this system no high-risk patient is unable to access immediate tertiary hospital care and most transfers are completed within 24 hours of presentation. This service, at the time with which these Inquests are concerned, would have been available to the medical practitioners involved, but none of those medical practitioners availed themselves of the service nor for that matter obtained any other form of specialist advice when dealing with the four individual patients.

4.17. Other services have either been developed or improved such as more seamless access for country patients to invasive investigational and treatment services. As well, comprehensive cardiac continuing medical education has been provided to medical and nursing staff in rural areas at specifically scheduled meetings.

4.18. In respect of the four country centres with which this Inquest is concerned, Dr Tideman outlines in his report the efforts that have been made since to provide continuing medical education to local practitioners as well as informing them as to the availability of iCCNet cardiology services. The relevant country centres in this case are Yorketown, Keith, Port Lincoln and Wallaroo.

4.19. Dr Tideman states that a meeting with nursing staff and general practitioners occurred Yorketown in February 2002. Dr Tideman maintains that Yorketown medical staff had consistently participated in iCCNet external quality assurance programs for point of care tests and had access to the diagnostic protocols since 2002. However, the doctors in the Yorketown facilities are generally not accessing consultant cardiology advice through iCCNet due to their strong affiliations with the Royal Adelaide

Hospital. This was confirmed in the evidence of Dr Tucker who is one of the local practitioners.

4.20. As far as the medical profession in Keith is concerned, on 23 April 2003 Keith Hospital doctors and senior nursing staff attended a presentation on chest pain and acute coronary syndrome management and the role of iCCNet in Keith. The Keith Hospital formally joined the network and was provided with bedside Troponin assays as of 30 April 2003. Dr Tideman reports that the doctors and nurses of the Keith Hospital have been regular participants in the many continuing medical education activities provided by iCCNet in the South-East and that they have had access to, and have regularly utilised, all of the services provided by iCCNet and have regularly sought advice in relation to patients presenting with potential cardiac pathology.

4.21. Port Lincoln has had the services of a resident consultant physician, Dr Rufus McLeay, for some years. Dr Tideman reports that he had liaised with Dr McLeay since 2000 on aspects of supporting rural practitioners in acute cardiac management. Implementation of the full iCCNet service occurred after an initial meeting in Port Lincoln that was well attended by the general practitioner community on 10 June 2004. A further comprehensive meeting on chest pain and acute coronary syndrome management and the services provided by iCCNet was held in Port Lincoln on 28 February 2006.

4.22. Dr Tideman reports that as far as the Wallaroo Hospital is concerned, informal discussions initiated through senior nursing staff regarding Wallaroo Hospital's involvement with iCCNet have been ongoing since approximately March 2005. Through the local division of general practice, Kadina and Wallaroo doctors would have been invited to the Yorke Peninsula continuing medical education meetings in 2002 and 2004. However, at the date of the incident involving the Wallaroo Hospital, namely 4 July 2006, the hospital did not have access to any of the services now routinely provided to hospitals in country South Australia that we have been speaking of.

5. The circumstances of the death of Mr Brian Terrance Dalling

5.1. Mr Dalling lived with his wife at Corny Point on the Yorke Peninsula. Mr Dalling had for some time been a patient of the Yorketown Medical Practice. Yorketown is one of the major centres on Yorke Peninsula. At all material times Drs Geoffrey Tucker and George Kokar were the principal medical practitioners of the Yorketown Medical Practice. I understand that Mr Dalling saw both doctors from time to time.

5.2. Some of Mr Dalling's previous medical history is relevant. Before I deal with that, I should mention that Mr Dalling's post-mortem examination revealed significant heart disease of an older origin quite apart from the acute pathology that was to take his life in December 2004. When Mr Dalling's heart was closely examined at autopsy it was noted that there was fibrous scarring of the left ventricle which indicated previous myocardial infarction (heart attack). There were also changes of evolving myocardial infarction, the histological appearances of which were those of an infarct of approximately 10 to 14 days in age. These appearances suggested that Mr Dalling had suffered chronic myocardial ischaemia (insufficient oxygen supply to the heart muscle) with a recent previous myocardial infarction and with further acute myocardial ischaemia as the terminal event. In addition, Mr Dalling had pulmonary oedema and hepatic congestion which were consistent with a degree of cardiac failure. To summarise therefore, Mr Dalling had suffered a myocardial infarction in the past and had suffered a further myocardial infarction in the 10 to 14 days prior to his death. As well as that he had also suffered the acute and fatal myocardial ischaemic episode on 30 December 2004, the day of his death.

5.3. Mr Dalling was diagnosed with diabetes in mid 1999. It appears that in the ensuing years his diabetes had been poorly controlled. Mr Dalling had other predisposing factors for heart disease. He had a history of smoking, had elevated cholesterol and hypertension.

5.4. In October 1999 Mr Dalling had undergone surgery at the Yorketown Hospital. This surgery was quite unrelated to his heart. Indeed, as of 1999 no heart disease had been diagnosed or suspected at any previous time in his life. However, as part of Mr Dalling's pre-anaesthetic review in respect of this operation, he underwent a routine electrocardiograph (ECG) and this revealed something of significance in relation to Mr Dalling's heart. An ECG is an examination in which a number of leads are attached to the skin of the chest, arms and legs and is a measure designed to provide information in respect of cardiac function. The ECG prints out a trace in which the

heart's electrical activity, as detected by the various leads, can be examined on a chart that is similar to graph paper. Mr Dalling's ECG was taken on 18 October 1999. It was reported as an abnormal ECG insofar as the various traces of the ECG did in fact reveal abnormalities quite consistent with Mr Dalling having suffered a myocardial infarction, or heart attack, some time in the past. If Mr Dalling had experienced any signs or symptoms of this heart attack at the time it had occurred he does not appear to have reported them to anyone. Suffice it to say if Mr Dalling had experienced symptoms, he had not sought any medical attention at the time. I was told during the course of the Inquest that heart attacks can be silent, as it were, meaning that the affected person does not experience pain or discomfort or other symptoms that a person might experience in a heart attack such as shortness of breath or sweating. A diabetic in particular might not experience symptoms of a heart attack and may be quite unaware that a heart attack has taken place. As already seen from Mr Dalling's autopsy report, he had in fact suffered a heart attack in the past. It therefore seems very likely that the ECG of October 1999, which reported that Mr Dalling had experienced a previous heart attack, had correctly diagnosed that as a fact. An experienced cardiologist who was called to give expert evidence, Dr William Heddle, confirmed that, in his view, this ECG had correctly reported a clear abnormality, namely the myocardial infarction.

5.5. It should be emphasised that the ECG of 18 October 1999 was not performed as a result of Mr Dalling experiencing symptoms consistent with a heart attack. Rather, it was a routine test performed as part of Mr Dalling's workup for his operation. The ECG printout of October 1999 is contained within the records of the Yorketown Hospital that concern Mr Dalling[18]. A copy of the ECG printout is also found within his Yorketown Medical Practice general practitioner records. I understood from the evidence that a copy of an ECG would be sent as a matter of course to the patient's general practitioner. As it happened, the Yorketown Hospital and the Yorketown Medical Practice were virtually adjacent. The October 1999 ECG has been signed by Dr Tucker, one of the practitioners at the Yorketown Medical Practice. Dr Tucker was the anaesthetist during Mr Dalling's 1999 surgery. Dr Kokar, the other Yorketown practitioner, assisted the surgeon. There can be little doubt, therefore, that at least Dr Tucker had been aware of the report and the result of the ECG in 1999.

[18] Exhibit C24c

5.6. The ECG report that bore reasonably strong evidence that Mr Dalling had suffered a heart attack in the past is not the subject of any comment within Mr Dalling's clinical records, either from the Yorketown Hospital or from the Yorketown Medical Practice. His progress notes do not mention the ECG at all.

5.7. A statement verified by affidavit of Mr Dalling's wife, Wendy Ann Dalling, was tendered to the Inquest[19]. Mrs Dalling states that about two weeks prior to her husband's death, which occurred on 30 December 2004, he mentioned to her that he had experienced chest pains while he had been driving his employer's water truck. As a result of this he attended the Yorketown Medical Practice on 13 December 2004. Mrs Dalling's statement does not relate any previous episode of chest pain experienced by her husband.

5.8. On 13 December 2004 Dr Samer Mikhail saw Mr Dalling at the Yorketown Medical Practice. Dr Mikhail obtained his medical degrees from the University of Adelaide in 2001. In 2004 Dr Mikhail was a General Practitioner Registrar undergoing his general practice training. He had been attached to the Yorketown Medical Practice since January 2004 for this purpose and had been under the supervision of Dr George Kokar. Dr Mikhail had undergone his basic training at that practice for about 6 months and had spent his advanced training there as well. He was in the advanced term of his training in December 2004. He spent about 12 months in the practice altogether. Dr Kokar's supervision consisted of a review session once per week and, in addition, Dr Mikhail would have been expected to refer any issues or difficulties connected with complicated cases to Dr Kokar or Dr Tucker for advice.

5.9. In December 2004 Dr Mikhail was not aware of the iCCNet service that provided telephone advice. He told me that if he required such advice he would refer matters to medical staff at the Royal Adelaide Hospital.

5.10. In December 2004 the practice was utilising what Dr Mikhail described as a '*hybrid system*'[20] for their records. There were both handwritten records as well as a computerised system. Dr Mikhail told me that he had become more reliant on the computerised system. However, it is clear in respect of Mr Dalling's appointment of 13 December 2004 that Dr Mikhail made notes in both the written clinical record as

[19] Exhibits C1 and C1a
[20] Transcript, page 35

well as in the computerised record. There are therefore two sets of notes in respect of this presentation.

5.11. Dr Mikhail had never seen Mr Dalling before this appointment. Indeed, he did not have any occasion to see Mr Dalling afterwards, although Mr Dalling would return to the practice about a week later and see Dr Tucker. I return to that subsequent appointment in due course.

5.12. On 13 December 2004 Dr Mikhail noted that Mr Dalling complained of jaw pain and left-sided chest pain and expressed concern about his heart. The clear inference which I draw from this notation is that Mr Dalling himself associated his symptoms of jaw pain and chest pain with a possible difficulty with his heart and that he made his concern in this regard plain to Dr Mikhail. Dr Mikhail told me that he had no recollection of Mr Dalling's presentation on this day. He was accordingly only able to rely on the notes that he made at the time and upon his usual practices. Dr Mikhail, however, believes that Mr Dalling did not say anything about any previous difficulty with his heart. Dr Mikhail said that he would have noted anything of that kind. I accept Dr Mikhail's evidence about this. This, in my view, lends some support to the suggestion that although Mr Dalling, at the time of his consult with Dr Mikhail, entertained concern about heart disease, he did not have any actual knowledge of the same. I add at this point that although at the time of this consultation Dr Mikhail had possession of Mr Dalling's medical file from the Yorketown Medical Practice, he did not see the copy of the 1999 ECG that was within the file. My examination of the file does not reveal any further reference to this ECG result nor any further suggestion or notation of heart disease in respect of Mr Dalling.

5.13. Inside the front cover of Mr Dalling's medical file is a page which for the most part is blank, but at the top of which is a very short account of the salient features of Mr Dalling's medical history at Yorketown Medical Practice. It records amongst other things a torn Achilles tendon in 1999, but more significantly it records on an orange sticker the single word 'DIABETIC'. This notation is against a reference '6/99'. Mr Dalling was first diagnosed as a diabetic in June 1999. The fact that he was a diagnosed diabetic was also recorded in the computerised record under 'past medical history'. This stated that Mr Dalling suffered from 'diabetes mellitus - NIDDM'.

There was further reference to Mr Dalling's diabetes against an entry in July 2002. In spite of these references, Dr Mikhail was unable to recall specifically whether or not he had seen anything in Mr Dalling's written or computerised records about the fact that Mr Dalling was a diagnosed diabetic.

5.14. If Dr Mikhail did not know that Mr Dalling was a diabetic it is not for a want of clear and adequate notations within Mr Dalling's written records and the computerised records. Insofar as Mr Dalling's diagnosis as a diabetic should have been relevant to Dr Mikhail's examination and diagnosis, there seems to me to have been no valid reason that would excuse Dr Mikhail's ignorance of that fact. It was said in evidence that if Dr Mikhail had been ignorant of the fact that a man of 55 years of age presenting with chest and jaw pain was not a diagnosed diabetic, it was a significant omission[21]. It is hard to disagree. The same observation, however, could not be made with respect to the pre-existing heart disease that Mr Dalling had suffered from. As I say, the only material that suggested that Mr Dalling had suffered from heart disease was the ECG that had been incidentally conducted at the time of Mr Dalling's surgery in 1999. Dr Mikhail believes that he did not see that ECG result. It was among the other papers in Mr Dalling's file, the number of which are considerable and run to 84 photocopied pages and, as I say, there is no other reference to the ECG anywhere on the file.

5.15. Complicating Mr Dalling's presentation on 13 December 2004 was the fact that other than jaw and chest pain, there was no other symptomatology of an acute cardiac event such as shortness of breath, palpitations, nausea or vomiting. However, his blood pressure was somewhat elevated at 160 systolic. Mr Dalling also presented on examination with a post-nasal drip and reduced air entry in his chest. In the computerised record Dr Mikhail has recorded that Mr Dalling presented with 'multiple symptoms'. Dr Mikhail told me that his symptoms were non-specific and had believed that they were consistent with an upper respiratory tract infection or throat infection. He put the jaw pain down to Mr Dalling being a chronic smoker and to the infection and believed that they were explicable as having an origin in throat pain and probably from tender lymph nodes. In Dr Mikhail's eyes the chest pain was also consistent with Mr Dalling's heavy smoking. In any event Dr Mikhail observed

[21] Dr P A Tideman, Transcript, page 565-566

that the pain had not been so severe as to prevent Mr Dalling from working or from coming in to the surgery. Specifically, Dr Mikhail did not ascribe Mr Dalling's pain as heart related because Mr Dalling did have a concurrent reason for the pain, namely the upper respiratory tract infection. In addition, the pain was not typical and Mr Dalling did not give any history of a cardiac cause. There was also the absence of other signs that I have already mentioned such as the lack of shortness of breath, palpitations and nausea.

5.16. In the event, Dr Mikhail prescribed Panadeine Forte for the pain and Amoxyl for the post-nasal drip. Regarding Mr Dalling's elevated blood pressure, Dr Mikhail decided to take bloods and advised Mr Dalling to undergo an ECG at the local hospital. He advised Mr Dalling to make another appointment once the blood test and ECG results were available. Dr Mikhail told me that his suggestion that Mr Dalling undergo an ECG had arisen not because he had believed that any acute cardiac event was taking place, but had been made with a view to establishing what Mr Dalling's 'baseline' was. However, Dr Mikhail did allude to the possibility that in the light of Mr Dalling's uncontrolled and elevated blood pressure an ECG might reveal an enlarged heart. He discharged Mr Dalling from the practice having reassured him about his presentation and having counselled Mr Dalling on his smoking habit.

5.17. Dr Mikhail did not make any specific arrangement for Mr Dalling to undergo the ECG at the hospital, which was in very close proximity to the practice, believing that Mr Dalling would arrange it for himself. Dr Mikhail did not believe that there was any need for an urgent ECG.

5.18. As indicated earlier, Dr Mikhail told me that he did not recall whether he knew about Mr Dalling's diabetes, but said in effect that it would not have made any difference to his approach to Mr Dalling. For my part, it is difficult to see how a history of diabetes could have been anything other than highly relevant to a possible diagnosis of acute coronary syndrome. To that extent, Dr Mikhail ignorance of the fact that Mr Dalling was a diabetic would mean that any diagnosis that expressly or by implication excluded a cardiac cause of Mr Dalling's symptoms was intrinsically flawed.

5.19. The same would apply of course to ignorance of previous heart disease, although I do not think in the circumstances Dr Mikhail could personally be held responsible for not

knowing about that. Dr Mikhail did concede that the ECG result that was latently on Mr Dalling's file would have altered his approach. Dr Mikhail also noted that Mr Dalling was at risk of heart disease and I take it that he was referring to Mr Dalling's risk factors of elevated blood pressure and his chronic smoking[22]. When Dr Mikhail was asked in court to examine the copy of the 1999 ECG that was in the Yorketown Medical Practice file, he regarded the ECG as demonstrating an abnormality. Specifically, he said that there appeared to be small Q waves in leads 2, 3 and AVF which indicated that Mr Dalling might have experienced a previous myocardial infarction. In saying so, Dr Mikhail would have preferred to have aligned the ECG with clinical evidence that may have existed in respect of Mr Dalling at the time it was taken, but when asked as to whether his treatment on 13 December 2004 or his management on that day would have been different in the light of the old ECG, he said '*yes*'[23]. He said:

> 'I would most I think quiz him again about his cardiac history and we will get him to get the ECG more urgently because he did have some changes in the past, so I would like to know what his heart, his ECG, looks like this minute.' [24]

It will be noted here that the ECG that Dr Mikhail suggested Mr Dalling should undergo was not an urgent ECG. It was an examination that Mr Dalling was expected to arrange for himself in his own time. An urgent ECG would have involved Mr Dalling being sent immediately to the adjoining hospital in Yorketown. As a measure of this, Dr Tucker told me in evidence that he would personally walk a patient over to the hospital if an urgent ECG was indicated. However, in the circumstances that existed after Mr Dalling's appointment with Dr Mikhail, whether Mr Dalling was to have the ECG was a matter very much dependent upon whether or not Mr Dalling felt so inclined. In that sense, too much was left to chance or to Mr Dalling's personal whim. Incidentally, I mention here that during his evidence Dr Mikhail was also shown the much clearer version of the 1999 ECG kept within the Yorketown Hospital records. This copy confirmed in Dr Mikhail's mind that Mr Dalling had suffered a previous myocardial infarction.

5.20. Dr Mikhail told me that if he had seen the 1999 ECG he would have sent Mr Dalling for a Troponin test in addition to ordering an urgent ECG. A Troponin test is the

[22] Transcript, page 45
[23] Transcript, page 65
[24] Transcript, page 65

blood test that may indicate myocardial damage experienced in the course of an acute heart attack. I return to the significance and subtleties of a Troponin test later in these findings.

5.21. I find that if Dr Mikhail had been aware of the ECG results from the past, it is highly likely that he would have sent Mr Dalling straight to the hospital for an immediate ECG and Troponin test and generally followed the protocols that existed in respect of suspected acute cardiac presentation. But Dr Mikhail was of the belief that none of these protocols had been enlivened because there were explanations for Mr Dalling's jaw and chest pain that were quite consistent with a presentation of something not cardiac related. Mr Dalling as it happened had multiple symptoms and Dr Mikhail did not believe that they were cardiac related or that they required immediate evaluation with an acute cardiac event in mind.

5.22. Mr Dalling's wife observes in her statement that following Mr Dalling's visit to Dr Mikhail, Mr Dalling told her that Dr Mikhail had said that his chest pains were the result of high sugar, high cholesterol and a throat infection.

5.23. In the event, Mr Dalling did not have an ECG, or for that matter a Troponin test, prior to his death on 30 December 2004.

5.24. The suggestion was made during the Inquest by counsel acting for Dr Mikhail that the evidence was in any event insufficient to warrant the conclusion that Mr Dalling's presentation on 13 December 2004 had involved a cardiac difficulty. I reject that contention. Mr Dalling's symptomatology on that occasion was indicative in itself of at least unstable angina if not an acute coronary syndrome. He had both chest and jaw pain and his presentation occurred against the background of his age and a raft of risk factors for heart disease. It was said in evidence that he virtually had a '*full house*' of such factors[25]. As well, although Mr Dalling's fatal episode did not occur until 30 December 2004, it will be remembered that at autopsy it was revealed that he had experienced a myocardial infarction 10 to 14 days before his death. I accept that unchallenged evidence. Mr Dalling's heart attack therefore occurred between 16 and 20 December 2004 which is between 3 and 7 days after the consultation of 13 December 2004. This confluence of circumstances to my mind renders it highly unlikely that Mr Dalling's presentation on 13 December 2004 was anything other than

[25] Dr M B S Adams, Transcript, page 418

cardiac related. Dr Adams' view of the matter was that the possibility that Mr Dalling's presentation was reflective of something else was only a remote one. In fact, when pressed, Dr Adams suggested that an acute coronary syndrome was a 98% certainty[26]. At one point in his evidence Dr Tideman suggested that he did not think that Mr Dalling had an acute coronary syndrome at the time of his appointment with Dr Mikhail. He appeared to base this view upon the fact that Mr Dalling came back to the practice 8 days later on 21 December 2004 and that it was apparent that in the intervening period he had not had an acute event. To my mind, this did not properly take into account the fact that the post mortem evidence revealed the existence of the myocardial infarction that had occurred between 13 and 21 December 2004. Although Dr Tideman is a cardiologist and Dr Adams is a general practitioner, in my view Dr Adams' opinion was a more fully informed one and I prefer his evidence. In my view the evidence enables me to conclude on the balance of probabilities that on 13 December 2004 Mr Dalling was experiencing an acute coronary syndrome and I so find. If I am wrong in that conclusion, to my mind the evidence would in any event lead to the inescapable conclusion that Mr Dalling was experiencing unstable angina.

5.25. The results of the blood tests that Dr Mikhail did order were made available in due course. On 21 December 2004, which was 8 days after his previous consultation with Dr Mikhail, and 9 days before his death, Mr Dalling saw Dr Tucker in the rooms of the Yorketown Medical Practice. I am not entirely certain as to how and when this appointment was arranged. However, Dr Tucker told me that he had deduced that his partner, Dr Kokar, had earlier gained access to Mr Dalling's computerised blood results and so it was to be inferred that Mr Dalling had been asked to come into the practice to have the results explained to him. The consultation proceeded simply on that basis.

5.26. If, as stated in the post-mortem report, Mr Dalling had suffered a myocardial infarction 10 to 14 days before his death, this would have occurred in the period between Mr Dalling's consultation with Dr Mikhail on the 13 December 2004 and his consultation with Dr Tucker on 21 December 2004. Notwithstanding this, there is no evidence that Mr Dalling experienced any outward signs or symptoms of a heart attack in that intervening period and he certainly did not mention any of the same to Dr Tucker on 21 December 2004. Indeed, Dr Tucker believes that he would have

[26] Dr M B S Adams, Transcript, page 477

asked Mr Dalling simply as a matter of routine whether he had suffered any such symptoms. Dr Tucker told me that he would have asked Mr Dalling whether he had any chest pains, whether he had been unusually short of breath and whether he had been generally well. He believed Mr Dalling had said that he had not had any problems.

5.27. Dr Tucker told me that because of the extremely busy nature of his practice at Yorketown, servicing as they did several thousand residents of the Yorke Peninsula, and that does not take into account the influx of holidaymakers, he had to restrict his appointments to approximately 10 minutes. There is no reason to suppose that Mr Dalling's appointment exceeded that on this occasion. Dr Tucker discussed Mr Dalling's blood results with him. As it happened, the blood results revealed significantly elevated blood sugar and cholesterol levels. The raised blood sugar level was in keeping with Mr Dalling's poorly controlled diabetes, a condition that was well understood in the practice. The high cholesterol level is another risk factor for heart disease. Dr Tucker counselled Mr Dalling about his lifestyle and suggested that certain aspects of it required change. He suggested that Mr Dalling come back and provide Dr Tucker with some blood sugar readings for further assessment. It appears that access to Mr Dalling's computerised clinical record was limited on this occasion to a perusal of the blood results. However, during this appointment Dr Tucker was in possession of Mr Dalling's written practice record and he made an entry into the same. Regarding the outcome of the consultation Dr Tucker has written:

> 'Not on diet. Ignoring it – diet sheet
>
> To chart BSLs & C'

This notation is written on the same page as, and directly below, the handwritten notes that had been made by Dr Mikhail on 13 December 2004. In fact Dr Mikhail's name-stamp, as well as a stamp of that date in red ink, is clearly visible and particularly so to a person who was writing on the very same page. It will be remembered that Dr Mikhail had noted the complaint of jaw pain and left-sided chest pain and had also noted his recommendation that Mr Dalling undergo an ECG. Dr Tucker told me, and I accept his evidence, that he did not read Dr Mikhail's entry in the notes and was not otherwise aware of the type of presentation that Mr Dalling had exhibited during his previous appointment with Dr Mikhail.

5.28. As seen earlier in these findings, Mr Dalling had been a patient of the Yorketown Medical Practice for several years. Dr Tucker had acted as the anaesthetist at Mr Dalling's orchidectomy operation in 1999 and had administered the ECG in the pre-anaesthetic workup. Dr Tucker had therefore been aware of the ECG results in 1999 that had reported an inferior myocardial infarction in terms, with trace results consistent with that diagnosis. But it is fair to say, I think, that Dr Tucker did not have those results in his mind when he saw Mr Dalling on 21 December 2004, some 5 years later. However, Dr Tucker regarded the 1999 ECG as having limited relevance in any event. Dr Tucker believed that the ECG result may have been consistent with a very small myocardial infarction but was more likely to have been reflective of an episode of minor myocardial ischaemia that could have been the result of a narrowed coronary artery. Dr Tucker believes that on its own it did not constitute evidence of a heart attack as such. Dr Tucker said that in 1999 when the ECG had been performed he would have asked Mr Dalling whether he had suffered any pain and he obviously had denied it, although Dr Tucker acknowledged that 20% of infarctions are painless. Dr Tucker told me that the ECG result had not been an impediment to Mr Dalling being anaesthetised for the 1999 operation, and the fact that he had been anaesthetised, operated on without incident and had survived the experience constituted evidence that there had been no underlying serious cardiac disease at the time. Dr Tucker believed that in 1999 he would have explained the ECG abnormalities to Mr Dalling and that he would also have told him that the possible underlying atherosclerosis responsible for the abnormal ECG result would have been reflective of his diabetes.

5.29. I add here that Dr Tucker's view that the 1999 ECG simply reflected an episode of minor myocardial ischaemia and not an infarct, was not shared by any other witness who was asked to view it. Drs Heddle and Adams both held the view that it clearly represented evidence of an old infarct, as did Dr Mikhail himself. I prefer the evidence of those practitioners to that of Dr Tucker on this subject. Dr Heddle in particular, a specialist cardiologist, was significantly more qualified than anyone to express an opinion about the significance of an abnormal ECG result. I find that the 1999 ECG revealed that Mr Dalling had experienced a myocardial infarction some time before the ECG was taken in October of that year. The ECG which reported itself as abnormal and reflective of a previous myocardial infarction, signed as it was by Dr Tucker at the time both on his own copy and on that belonging to the local

hospital, was thus true to label. Even allowing for the fact that this diagnosis was, as it were, stumbled upon during pre anaesthetic procedures for an unrelated illness, it is astonishing that a matter so fundamental to the continued well being of an individual should go unremarked upon in any other notation on what is otherwise a comprehensive file.

5.30. To summarise, Dr Tucker was not aware of Mr Dalling's previous presentation of chest and jaw pain on 13 December 2004 and did not have uppermost in his mind the ECG result from 1999. A cardiac problem was therefore simply not on the table as far as Dr Tucker's consultation with Mr Dalling was concerned. Dr Tucker was naturally asked in evidence what he would have made of Mr Dalling's previous presentation when seen by Dr Mikhail if he had been aware of that presentation. Dr Tucker was of the view that even leaving aside the 1999 ECG result, Mr Dalling's presentation when seen by Dr Mikhail on 13 December 2004 should have dictated a more urgent clinical response at that time. The presentation of chest pain, together with what Dr Tucker assumed was radiating jaw pain, was indicative of a possible acute coronary syndrome. There is no direct evidence that Mr Dalling's jaw pain on 13 December 2004 was a radiating pain, in the sense that it radiated from the chest, but the juxtaposition of chest pain and jaw pain and the coincidence of its timing is suggestive in itself of cardiac origin and I think it would be splitting hairs to suggest that because Mr Dalling did not himself describe it as 'radiating' that the jaw pain did not have an association with the chest pain. It will be remembered of course that Mr Dalling himself, in the context of describing his pain, expressed concern to Dr Mikhail about his heart.

5.31. Dr Tucker conceded that the 1999 ECG result should have been flagged in Mr Dalling's notes[27]. He also conceded that it was his usual practice to review the notes of the immediately previous consultation of the patient[28]. When asked by counsel assisting, Dr Gray, whether Dr Tucker's management on 21 December 2004 may have been different if he had reviewed the practice notes and seen the 1999 ECG results, he said that he would not have needed any ECG results because he described Mr Dalling's presentation to Dr Mikhail on 13 December 2004 in any event as a '*classic history of precordial chest pain with radiation*'[29]. For him a presentation of

[27] Transcript, pages 111-112
[28] Transcript, page 113
[29] Transcript, page 124

chest pain with radiation to the jaw was enough to tell him that a possible adverse cardiac event may have occurred and that Mr Dalling needed immediate investigation for that. The other factor in Mr Dalling's presentation and longitudinal history was his diabetes which for Dr Tucker would indicate that he probably had atheroma in most of his major arteries[30]. Dr Tucker suggests that he would have subjected Mr Dalling to the usual protocols, even without the old 1999 ECG test. Dr Tucker said:

> 'He would have been taken by me bodily out of my room and through a room into a passage and straight into the Accident and Emergency.' [31]

5.32. Dr Tucker expressed some measure of disappointment about the manner in which Mr Dalling's presentation on 13 December 2004 had been handled by Dr Mikhail. Dr Tucker told me that they had undertaken to supervise the trainee registrars who had been allotted to their practice and to this end he and his partner had always been totally available to trainees. He made the point that they were entitled to telephone the doctors or to walk into their offices and see them at any time of the day or night. Dr Tucker told me that if Dr Mikhail had thought it necessary for Mr Dalling to have an ECG, it was also very important that he told one of the partners about that. While accepting that Dr Mikhail could not be criticised for the fact that Dr Tucker had failed to read Dr Mikhail's note of the previous presentation, Dr Tucker suggested that it would have been better if Dr Mikhail had 'flagged' Mr Dalling's presentation and had told the partners of the practice about it, given that there may have been in existence a clinical concern about a life threatening problem in Mr Dalling's case. Dr Tucker colourfully put it this way:

> 'If you can't follow that up you can't rely on the pixies to tell you by luck that that is going to be done with certainty, unless you tell people.' [32]

Dr Tucker said that if a life threatening issue were potentially involved in a patient's presentation, a doctor in Dr Mikhail's position would be expected to walk into the supervisor's room and tell him[33].

5.33. In the event, following Dr Tucker's consultation Mr Dalling left the clinic and was not to seek any further medical attention prior to his death. Dr Tucker told me that Mr

[30] Transcript, page 125
[31] Transcript, page 126
[32] Transcript, page 134
[33] Transcript, page 134

Dalling's last chance of having his life saved more or less walked out the door with him[34]. Dr Tucker was in my assessment genuinely regretful about that.

5.34. Mrs Dalling's statement[35] does not deal with the period between her husband's first presentation on 13 December 2004 and the day of his death except, as already referred to, that her husband had told her that Dr Mikhail had said that his chest pains had been down to high sugar, high cholesterol and the throat infection. If Mr Dalling had any further episodes of chest pain prior to his death Mrs Dalling does not mention that fact. However, in the late afternoon of Thursday 23 December 2004, which was two days after Mr Dalling's consultation with Dr Tucker, Mr Dalling had a conversation with Senior Constable Beaumont of the Yorketown Police. They had known each other for a number of years. They bumped into each other outside a chemist's store in Yorketown. Mr Dalling told Senior Constable Beaumont how he had recently been suffering chest pains and that a doctor had diagnosed it as a combination of diabetes, blood cholesterol and a throat infection, virtually the same as what he had told his wife. Mr Dalling, however, also told Senior Constable Beaumont that he personally believed that the chest pains had been caused through the turning of the steering wheel when driving his truck.

5.35. There was a faint suggestion posed during the Inquest that Mr Dalling may have downplayed his symptoms out of a concern that a diagnosis of heart disease might jeopardise his truck licence. Such a conclusion is simply not available, especially having regard to the fact that when he saw Dr Mikhail he explicitly associated his symptoms with possible heart disease. In addition, it is unlikely that Mr Dalling would have raised the subject of his health with an officer of the law if he had been so concerned.

5.36. It was while driving his employer's truck that Mr Dalling suffered his final and fatal heart attack on 30 December 2004. The truck was seen to leave the road unexpectedly and then come to a stop in a paddock. Mr Dalling was there located deceased.

5.37. <u>Commentary upon the circumstances of the death of Mr Brian Terrance Dalling</u>

As indicated earlier, the circumstances of Mr Dalling's presentation and death were,

[34] Transcript, page 128
[35] Exhibit C1a

in the first instance, examined by Dr Adams. Dr Adams provided two reports[36] and he gave oral evidence in the Inquest. Dr Heddle also commented upon Mr Dalling's circumstances. Dr Tideman also made some general comments concerning Mr Dalling's death to which I have already alluded.

5.38. Dr Adams' reports indicate that in his opinion that when Dr Mikhail saw Mr Dalling on 13 December 2004, Mr Dalling was suffering from an acute coronary syndrome. Dr Adams referred to the disorderly nature of Mr Dalling's casenotes at the Yorketown Practice. He suggested that the 1999 ECG result of a previous myocardial infarction, and Mr Dalling's history of diabetes, were matters that were apparently not known to Dr Mikhail but were nevertheless relevant to a consideration of Mr Dalling's risk factors for heart disease. In particular, Dr Adams suggested that Dr Mikhail could be forgiven for not knowing that Mr Dalling had experienced a previous myocardial infarction, given the obscurity of the 1999 ECG within the practice notes. I have already commented to the effect that I agree with that observation. I am unable to agree that being ignorant of Mr Dalling's diabetes was reasonable in the light of the fact that this diagnosis had been displayed in both the written and computerised records. In his second report, Dr Adams observes that given that Dr Mikhail knew, or ought to have known, that Mr Dalling was at high risk for coronary artery disease, that Mr Dalling himself was concerned about coronary artery disease as a cause for his pain, that the pain was consistent with angina or an acute coronary syndrome, and that it had been occurring on the day of consultation, Dr Mikhail ought to have taken further diagnostic and therapeutic steps in respect of Mr Dalling.

5.39. Dr Adams also makes certain observations about Dr Mikhail's suggestion to Mr Dalling that he ought to have a non-urgent ECG conducted in his own time. He observes that on a retrospective analysis of the written material, including the practice notes, Dr Mikhail ought to have thought the existence of an acute coronary syndrome to be enough of a possibility to have followed the iCCNet protocol or similar. This would have involved Mr Dalling having an immediate ECG at the local hospital. In his original report[37], Dr Adams points out, however, that even a properly undertaken and read ECG is not necessarily a reliable vehicle for the diagnosis of coronary heart disease. In this regard Dr Adams has referred to the Troponin test as a recognised

[36] Exhibit C26 and Exhibit C26a
[37] Exhibit C26

marker of myocardial damage, even in people who are having so-called 'unstable angina' which frequently precedes a heart attack. It is a valid observation in my view that the iCCNet protocol[38] dictates that persons who would enter the low risk pathway would, in any event, have an initial Troponin test as well as the ECG and also have a repeat ECG and Troponin tests before any question of discharge from the medical facility arose.

5.40. The difficulty with Dr Mikhail's approach in my view was that he did not know of Mr Dalling's risk factors in their entirety and thought he could explain Mr Dalling's presentation by reference to a chest infection.

5.41. Dr Adams also commented in his reports upon the consultation of 21 December 2004 with Dr Tucker. Dr Adams suggests that Dr Tucker ought to have identified the probability of the previous myocardial infarction as revealed by the 1999 ECG. As well, Dr Adams suggests that Dr Tucker ought to have read the progress notes that had been made by Dr Mikhail and then enquired about the presentation of pain on the earlier occasion. As well, he should have followed up whether or not Mr Dalling had undergone his ECG.

5.42. In his evidence before me Dr Adams made a number of further observations. He suggested that insofar as Mr Dalling's presentation may have been explained by an upper respiratory tract infection, he suggested that the symptoms were not in any case typical of the same. In this regard the comment by Mr Dalling himself that he was concerned about the possibility of heart disease was sufficient reason in itself for that to be taken seriously. He agreed with counsel that the presentation of chest discomfort radiating to the jaw, together with the concern expressed by Mr Dalling himself, should have been sufficient to require some form of testing to exclude the possibility of a cardiac event such as an urgent ECG.

5.43. Dr Adams made the observation that in this particular case, even if Mr Dalling were only to have undergone an urgent ECG, that of itself would probably have shown at least evidence of the previous myocardial infarction. That piece of information obviously would have added to the overall picture in respect of Mr Dalling. It clearly would have signified yet another risk factor as far as the possible development of heart disease was concerned and indeed would have strengthened the case for having

[38] Exhibit C39

Mr Dalling processed under the iCCNet protocol. Incidentally, Dr Adams disagreed with counsel's suggestion that the earlier ECG had simply reflected ischaemia rather than infarction. He repeated his view that the 1999 ECG reflected a '*high likelihood that the person had a previous inferior myocardial infarction*'[39] and that it was consistent with it having taken place at some time prior to the taking of the ECG. While agreeing that a new ECG would not necessarily have demonstrated something acutely taking place as at 13 December 2004, the revelation of previous myocardial damage would have increased the concern that the current pain could be due to cardiac ischaemia and would have resulted in more vigorous action at that time. It would also cause the practitioner to consider the existence of other possible risk factors. Again I make the observation that in any event if one were to embark upon a course of action that would involve even a journey along the low risk clinical pathway under the protocol, it would involve more than one ECG in any event as well as repeat Troponin tests.

5.44. Counsel suggested to Dr Adams that if Dr Mikhail had not thought that Mr Dalling was actually experiencing an acute coronary syndrome, it would follow that he would not have been offered a Troponin test[40]. To this Dr Adams said:

> 'Look, one is dealing with general practice where you are looking at probabilities. So I suppose you could say if Dr Mikhail thought that Mr Dalling's probability of having an acute coronary symptom (sic) was extremely low he ought not to have offered a troponin test; and I guess that's one of the other alternatives.' [41]

However, while Dr Adams agreed that a diagnosis other than acute coronary syndrome may have been a plausible one, in my view the difficulty is that if the alternative diagnosis is only at a level of plausibility, the possibility of the patient experiencing an acute coronary syndrome has hardly been discounted. In re-examination by counsel, Dr Gray, Dr Adams said that even if a coronary event based upon the signs and symptoms as recorded by Dr Mikhail on 13 December 2004 might not have been the most likely explanation, because of the seriousness of it and what he termed the '*red flag nature of it*'[42] that even something that was of a relatively low probability had sufficient probability to be worthy of an investigation.

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[39] Transcript, page 411
[40] Transcript, page 476
[41] Transcript, page 476
[42] Transcript, page 478

5.45. Dr Heddle in his report[43] suggested that whether or not the strong risk factors had been noted by Dr Mikhail, the presentation with chest discomfort radiating to the jaw should always raise the suspicion of coronary artery disease and that the iCCNet protocol should have been followed. Dr Heddle also agreed in his report that the 1999 ECG did show an old inferior myocardial infarction.

5.46. In his evidence Dr Heddle suggested that with Mr Dalling's presenting signs and symptoms, an urgent referral for an ECG ought to have been made[44]. He said that in general, general practitioners would organise for an ECG to be done at least that day, if not during the attendance within the consulting rooms. I observe here of course that the practice did not have an ECG machine, but the practice was virtually next door to the hospital.

5.47. Dr Heddle also made the powerful point in my opinion that it was not an appropriate strategy for Dr Mikhail to have dealt with Mr Dalling simply on the open ended basis that an ECG that Mr Dalling might organise in his own time could be reviewed at the same time that the blood results became available. Dr Heddle stated that when a patient is presenting with chest discomfort, and where coronary artery disease is a consideration, the correct management is to perform an immediate ECG, and that if there is a possibility of there being an acute coronary syndrome one would also conduct the Troponin test. This of course would be in keeping with what is required by the relevant protocol, even the low risk clinical pathway of that protocol. I have already referred to the general evidence of Dr Tideman which suggested that a person presenting with symptoms of the kind that Mr Dalling was experiencing would be subjected to acute coronary syndrome protocol and that it would be difficult to conceive of circumstances where an ECG would not be administered.

5.48. Dr Mikhail in his consultation proceeded without any knowledge of any prior heart disease or any knowledge of a history of diabetes. This lack of knowledge in itself somewhat rendered Dr Mikhail's analysis that the possibility of acute coronary syndrome could be dismissed as flawed. Even without that knowledge, it is difficult to see how Dr Mikhail's confidence in putting Mr Dalling's symptoms down to something quite unrelated could be sustained. In my view it is clear that Mr Dalling

[43] Exhibit C35
[44] Transcript, page 287

ought to have been, on 13 December 2004, subjected to the iCCNet protocol or the equivalent.

6. The circumstances of the death of Mr Jack William Salotti

6.1. Mr Salotti was 51 years of age at the time of his death on 21 November 2005. He had lived with his partner of 15 years, Ms Tosca Pilat, near Bordertown in the South East. Mr Salotti and Ms Pilat were both patients of the Keith & District Surgery the partners of which were Dr Tomy Varghese and his wife Dr Leeza Kurian. There was no cardiology specialist resident in the district, although a cardiologist by the name of Dr Straznicky visited from time to time. Within Mr Salotti's casenotes kept at the Keith & District Surgery is correspondence that relates to a presentation in April 2004 following an episode of chest pain that he had experienced some four days previously. The pain had lasted for 15 to 20 minutes and had been associated with sweating and burping. There was no radiating pain on that occasion. Troponin and CK tests were recorded as having been negative. An ECG was performed. A number of risk factors for heart disease were recorded as relating to Mr Salotti including a family history of the same, a sedentary lifestyle, the fact that Mr Salotti smoked about 30 cigarettes a day and had mild hypercholesterolemia (elevated cholesterol level). Mr Salotti had been examined by the cardiologist, Dr Straznicky, in May 2004. Dr Straznicky had identified Mr Salotti's risk factors. Dr Straznicky wrote a letter to Dr Kurian pointing out, amongst other things, that during his episodes of chest pain Mr Salotti had described the sensation of his heart racing with a particularly bad episode a few weeks beforehand. Dr Straznicky decided to perform stress testing to identify whether or not his pain was in some way reflective of cardiac ischaemia. On 18 June 2004 Dr Straznicky reported by way of letter to Dr Kurian that Mr Salotti had undergone 10 minutes of cardiac stress testing without ECG changes or chest pain. The letter goes on to state 'clearly his pain is non-cardiac'. Nevertheless, Dr Straznicky reported that he had counselled Mr Salotti about the risk factors that adhered to his lifestyle, especially his smoking, and had also recommended that Mr Salotti take aspirin regularly so as to reduce that heart attack risk. There is also discussion about Mr Salotti's cholesterol.

6.2. It will therefore be seen that while there was no positive diagnosis of heart disease or heart ischaemia in June 2004, there was certainly the suspicion of the same and, as

well, there had been a thorough identification of the risk factors that Mr Salotti operated under.

6.3. Ms Pilat's statement[45], verified by affidavit, states that Mr Salotti's health in the previous few weeks prior to his death had not been good. He had experienced what he thought was indigestion in as much as he was burping a lot and had some stomach and chest pain. Mr Salotti's most recent medical appointment had occurred on Saturday 12 November 2005. He had attended at the Keith & District Surgery and had been seen by Dr Kurian on that occasion. He was complaining of epigastric pain associated with burping and vomiting which, although present for the last 20 years, had become worse in the past 2 to 3 weeks. He had run out of the Nexium that he had been taking for what was thought to be gastro reflux. Dr Kurian has recorded that there was no radiated pain, no associated sweating, no shortness of breath, no palpitations and that the pain was not related to exertion. The pain was said to be relieved with Mylanta and vomiting. Mr Salotti's smoking habit, that is described this time as 20 per day, was noted. His blood pressure was recorded as $^{110}/_{20}$ (normal) during that presentation. He was prescribed something for his heartburn and it was suggested that he be reviewed for an endoscopy. Dr Kurian has noted the negative stress test from the previous year and her note also suggests that during the current consultation she gave consideration to Mr Salotti undergoing a cardiology review although I do not understand there to have been any firm arrangement made in that regard.

6.4. The following evening, Sunday 13 November 2005, Ms Pilat noticed that Mr Salotti was quite ill. He was complaining that his chest and his left arm hurt and he was burping a lot and vomiting, all in keeping, of course, with an acute coronary syndrome. Ms Pilat rang an ambulance. According to her statement, by the time the ambulance arrived Mr Salotti was sitting on the couch and 'seemed okay'. He was unenthusiastic about going to hospital. However, he was in any case taken by ambulance to the Keith and District Hospital where he alighted from the ambulance under his own power and appeared by then to be in a light-hearted mood. This demeanour is to be contrasted with his presentation as described by South Australia Ambulance Service (SAAS) personnel whose record of their attendance upon and conveyance of Mr Salotti forms part of the Keith and District Hospital clinical

[45] Exhibits C20 and C20a

record[46]. The SAAS record states that the ambulance arrived at Mr Salotti's home at 8:20pm and departed again at 8:38pm, arriving at the hospital at 9:20pm. Upon arrival at his home, the paramedics noted that Mr Salotti was sitting in a lounge chair, was in no pain but was 'very stressed'. The ambulance officers have recorded that they were given a history to the effect that Mr Salotti had been burping and vomiting that day with chest pain and that the pain from the centre of his chest had run down his left arm. It also records that he had experienced pins and needles during this attack. It is also recorded that he was, or at least had been, hyperventilating. It appears that there was a complaint of multiple attacks of this nature because the paramedics recorded that an attack lasted about 10 minutes with a 40-minute break 'between attacks'. It is also recorded that Mr Salotti could not keep his last meal down. There is a further notation that at 8:40pm, which must have been at a time when Mr Salotti was enroute in the back of the ambulance, that he had pins and needles and chest pain and also one assumes pain in the right arm on this occasion. Blood pressure recorded at 8:24pm and 8:35pm reveal readings of 150 systolic which is elevated.

6.5. Dr Varghese saw Mr Salotti at the Keith and District Hospital. Dr Varghese had practising rights at the local hospital.

6.6. Dr Varghese gave evidence in the Inquest. He received his medical qualifications in 1988 in India. He spent 3 years in practice there and moved to South Africa in 1992 where for a number of years he worked in a country hospital as a country general practitioner. He and his wife came to Australia in 2000. He was an emergency registrar in the Toowoomba Base Hospital in Queensland and after that moved to South Australia as a general practitioner. He worked in Peterborough for 6 months and then moved to Keith where he spent 5 years working as a country general practitioner.

6.7. In November of 2005 Dr Varghese was aware of the iCCNet protocols and had utilised them. He had also used the iCCNet specialist advice services including the ECG reviewing service.

6.8. When Mr Salotti arrived at the Emergency Department on the evening of 13 November 2005 Dr Varghese was already at the Keith Hospital seeing another

[46] Exhibit C27a

patient. Dr Varghese says he observed Mr Salotti's casual demeanour upon the latter's arrival. The nursing staff, however, have recorded Mr Salotti's behaviour as anxious, a description in keeping with that recorded by SAAS personnel. The nurses noted his presenting problem as chest pain, burping and vomiting since that morning and it is recorded by the nursing staff that he had a similar attack approximately 12 months previously. Thereafter the notes are made by Dr Varghese. He recorded lower sternal epigastric pain with radiation to both arms lasting for 5 to 10 minutes but that burping relieved the pain completely. There was no heaviness of his chest or sweating but Dr Varghese noted that Mr Salotti indicated that his heart rate went up with the pain and settled once the pain went away. Mr Salotti described similar incidents of this nature as having occurred on and off for many years. Mr Salotti seems to have made a point of telling Dr Varghese that he had undergone a cardiac assessment 6 months ago that had included a negative stress test. Dr Varghese has recorded that the stress test had been conducted by Dr Straznicky in March 2005. This information, coming as it did from the patient himself, was incorrect insofar as the cardiac assessment and stress test conducted by Dr Straznicky had occurred in mid 2004, some 18 months before Mr Salotti's presentation in November 2005. Dr Varghese took Mr Salotti's assertions about the timing of these tests at face value. It will be noted here that it was Dr Kurian, Dr Varghese's partner, who had referred Mr Salotti to the specialist in 2004 and to whom the specialist had reported, but Dr Varghese did not have access to the practice notes during the course of this current presentation.

6.9. On examination Dr Varghese found Mr Salotti to be comfortable and completely pain free. Nevertheless, systolic blood pressure readings of 194, 161 (possibly 167) were recorded which are all elevated. I note here that during the previous day's presentation to Dr Kurian, she had recorded his systolic blood pressure as 110 which is normal.

6.10. Dr Varghese decided to admit Mr Salotti to the hospital. A Troponin test was conducted and this was negative. The Troponin result is at page 19 of Exhibit C27a and is timed at 9:54pm. Later in these findings the desirability of conducting subsequent Troponin tests will be discussed in the light of the fact that a negative Troponin test, particularly at a time before Troponin is released into the blood stream

following a myocardial infarction, does not of itself indicate that there has been no acute cardiac event.

6.11. Dr Varghese conducted two ECGs. The first, which appears at page 18 of Exhibit C27a, bears a time of 8:23pm. This time must be incorrect given that Mr Salotti did not arrive until 9:20pm. Be that as it may, the ECG appears to have been taken not long after Mr Salotti's arrival at the hospital. This ECG describes itself as an 'ABNORMAL ECG' with 'MULTIPLE VENTRICULAR PREMATURE COMPLEXES'. The second ECG timed at 10:44pm which appears at page 17 of Exhibit C27a describes itself as an 'OTHERWISE NORMAL ECG'. In spite of the description of the earlier ECG as abnormal, Dr Varghese regarded both ECGs as normal. In his view what changes that there were, were very minor. However there is, as will be seen later in these findings, a significant change between the first and second ECGs that, together with Mr Salotti's clinical presentation, were in reality indicative of an acute coronary syndrome.

6.12. Mr Salotti was kept overnight in the hospital. Mr Salotti remained pain free for the rest of the night. An ECG was ordered to be performed were Mr Salotti to experience any further pain. No further ECG was performed.

6.13. Dr Varghese recorded in the hospital notes a differential diagnosis of 'oesophageal spasm and angina'. Although it was noted that Mr Salotti's blood pressure was raised, it descended and by 10:15pm it was recorded to be 150. There was further evidence later that night that his blood pressure descended to as low as 131 systolic at 11:40pm. Dr Varghese did not regard the earlier high readings as having any cardiac significance, but viewed them as being consistent with a level of stress at the time of Mr Salotti's original presentation. Dr Varghese's '*working diagnosis*'[47] was based on his conclusion that his pain could be of gastric origin. Dr Varghese believed that this was in keeping with the history that Mr Salotti had given him, particularly having regard to the ongoing nature of the pain, the ECG report and the negative Troponin test. In any case he admitted the patient on the basis that Mr Salotti ought to undergo all night observation to see whether he experienced any further pain. In the event, it does not appear that Mr Salotti experienced, or reported, any further pain.

[47] Transcript, page 157

6.14. In the morning, the clinical record notes that Mr Salotti had experienced no further epigastric or chest pain since admission and that his blood pressure was 146. Dr Varghese saw Mr Salotti that morning and recorded that there was no further pain or abdominal discomfort, that Mr Salotti's vital signs were stable and that he could be discharged. Mr Salotti was discharged in the afternoon without any further complaint of pain or discomfort. He took with him Pariet and Maxalon with the notation that he would be reviewed by Dr Kurian at the clinic at some point in time.

6.15. Mr Salotti only had the one Troponin test. The two ECGs were conducted within a fairly short space of time of each other and in any event, before midnight on 13 November 2005. No further Troponin test or ECG was conducted on 14 November 2005 before Mr Salotti's discharge. Dr Varghese, however, agreed that a repeat Troponin and an ECG test should have been conducted before Mr Salotti was discharged. This approach would have complied with the relevant protocols. Dr Varghese defended these omissions by suggesting that the results would not have made any difference because no changes from the results from the night before would have in any event been expected. He based this assertion on the fact that Mr Salotti had not experienced any further pain since his admission. As well, having regard to the similar episodes that Mr Salotti had experienced in the time leading up to his presentation on the evening of 13 November 2005, he would have expected the first Troponin test to have been positive if the episodes had been cardiac related.

6.16. Dr Varghese conceded that he had placed some reliance upon Mr Salotti's assertions that he had undergone a negative stress test for a cardiac related illness as recently as 6 months ago. He agreed with counsel that the information from Mr Salotti that he had undergone a negative stress test within the last 6 months had been a comforting consideration in determining whether or not Mr Salotti's current presentation was heart related. However, if he had been told the correct history in relation to the timing of the negative stress test and that it had in truth occurred approximately 18 months previously, not 6 months, Dr Varghese said that in those circumstances he would have ensured that the Troponin and ECG tests were repeated and would have treated Mr Salotti's presentation as an acute coronary syndrome, meaning that he would have placed him on blood thinners and probably have arranged for his transfer to Adelaide for immediate evaluation. He added:

> 'But the history I got and the findings I got from the initial assessment was of low suspicion and I thought probably it is the gastro intestinal ... since he stopped his medication, causing the symptoms. He told me that he'd just had the first tablet one day prior to his presentation. Normally it takes a few days for him to settle again once he starts his medication.' [48]

6.17. Mr Salotti's partner, Ms Pilat, records in her statement that in the week following Mr Salotti's discharge from the hospital he continued to take his indigestion medication and appeared to be, and said he was, feeling well with no chest or neck pain. However, at about 1:30am on Monday morning 21 November Ms Pilat was awoken by Mr Salotti vomiting. He was in the lounge room sitting on the lounge and was clearly very ill. He then collapsed. Paramedics attended. Mr Salotti was unable to be revived.

6.18. The post-mortem examination of Mr Salotti was conducted by Dr John Gilbert, a Forensic Pathologist. I accept Dr Gilbert's report[49], verified by affidavit, that the cause of Mr Salotti's death was ischaemic heart disease due to coronary atherosclerosis. Dr Gilbert has reported that Mr Salotti's left ventricular myocardium was hypertrophic, that is to say enlarged. There were also microscopic foci of subendocardial scarring and granulation tissue formation as well as occasional small groups of hypereosinophilic muscle fibres noted in the anterior wall of the left ventricle. There was also a severely narrowed branch of the left circumflex artery. All this suggested that Mr Salotti had suffered some ischaemic injury to the heart other than that experienced on the day of the acute heart attack that caused his death. Dr Gilbert also reports that the deceased's symptoms a week before his death would very likely have resulted from ischaemic heart disease. I find that to have been the case.

6.19. <u>Commentary upon the circumstances of the death of Mr Jack William Salotti</u>

This matter was commented upon by Dr Heddle both in his report[50] and during the course of his oral evidence. In his report Dr Heddle refers to the existence of an ECG that was conducted during Mr Salotti's assessment in April 2004 which is reproduced at page 22 of Exhibit C27. That ECG had been reported by the computer as demonstrating a lateral myocardial infarction, probably old, as well as ischaemic changes. Dr Heddle states that in his view this ECG had misreported a myocardial

[48] Transcript, pages 168-169
[49] Exhibit C19a
[50] Exhibit C33

infarction and ischaemic changes. He suggested that the ECG does not demonstrate any such pathology. However, he makes the comment that the 2004 ECG would have provided a useful point of comparison with the ECGs that were taken during the course of Mr Salotti's presentation on the evening of 13 November 2005.

6.20. As far as the two ECGs of 13 November 2005 are concerned, Dr Heddle suggested in his report that they are both abnormal. Dr Heddle does not make it clear in his report what the abnormalities in his opinion are, nor what their significance in the setting of Mr Salotti's presentation was. He said that the two ECGs recorded that evening showed no differences from each other apart from a technical artefact and a slight difference in heart rate in one instance. However, Dr Heddle suggested in his report that a comparison between those two ECGs with the ECG that had been taken in April 2004 would have demonstrated that there were in fact differences over that time and that these ought to have alerted a practitioner to the possibility of an acute coronary syndrome in November 2005. Dr Heddle suggested in his report that such a comparison should have been made, if not at the hospital during Mr Salotti's presentation then at least during the following day. It will be observed here that although the 2004 ECG was part of Dr Varghese's practice file for Mr Salotti, it was not available at the hospital during the course of Mr Salotti's admission. Mr Salotti survived for another week and there is no evidence that any comparison between the ECGs that had been taken on the night of the 13 November and the ECG of April 2004 was made in that time.

6.21. Dr Heddle also suggested that it would have been appropriate for Mr Salotti to undergo a further ECG 8 to 12 hours after his admission to hospital to assess whether there had been any changes. He suggested that the subsequent ECG would have been in accordance with the relevant guidelines.

6.22. As to the abnormalities that were said to exist within the two ECGs that were taken within close proximity of each other, Dr Heddle's report suggested that he would not have expected a general practitioner to recognise those abnormalities, but points out that the readily accessible expert opinion on ECGs could and should have been utilised. He is there referring to expert opinion that is available by telephone through the iCCNet entity. I return to the question of the relevance of the ECGs in a moment because Dr Heddle gave some oral evidence on the topic that does not seem to conform in all respects with what he said in his report.

6.23. As well as dealing with the question of ECG administration in his report, Dr Heddle also deals with the question of Troponin testing in Mr Salotti's case. It will be remembered from the previous section that Mr Salotti's one and only Troponin test was reported as negative. The Troponin test is purportedly timed at 9:54pm. In his report Dr Heddle points out that one negative Troponin test merely excludes previous infarction within hours or days prior to the admission but that when a patient presents with acute chest pain of sufficient severity to bring them to hospital, it is appropriate and recommended in the guidelines that Troponin tests should be repeated 8 to 12 hours after the first sample. Accordingly, in his view, a single negative Troponin test was insufficient to exclude a diagnosis of acute coronary syndrome or myocardial infarction. In this regard, Dr Heddle expressed a general concern that negative Troponin tests were being used diagnostically, whereas in reality a negative test does not in fact necessarily exclude acute coronary syndrome. It will be remembered that evidence of a myocardial infarction that might be revealed in a Troponin test will probably not be present until 6 to 8 hours have passed. It will be remembered that Mr Salotti had presented to Dr Kurian at the Keith Clinic the day before his presentation to the hospital and had reported epigastric pain that had been worse for the past 2 to 3 weeks. On that occasion there had been no ECG or Troponin testing. Mr Salotti had simply been provided with medication and it had been suggested that he be reviewed by way of an endoscopy and possibly a cardiological assessment with no firm arrangement made. It is not clear what time of the day this consultation had occurred, but clearly it was at least 24 hours prior to Mr Salotti's presentation at the Keith Hospital on 13 November 2005. The available point to be made is that if Mr Salotti had been experiencing a heart attack when seen by Dr Kurian the day before, evidence of it should have shown up in the Troponin test conducted the following evening – ergo there had been no heart attack the day before. I return to this contention in a moment.

6.24. In his oral evidence Dr Heddle suggested that Mr Salotti's symptoms on 13 November 2005 would in themselves raise a suspicion of coronary artery problems, particularly the radiation of the discomfort down the arm. This scenario would raise the need for procedures to be implemented to exclude a coronary event. Dr Heddle believed that the previous stress test of 2004, whenever it had occurred, would not have been of particular help in dealing with Mr Salotti's presentation on 13 November 2005 and that no comfort could sensibly have been derived there from.

6.25. In his evidence in chief, Dr Heddle provided further commentary upon the two ECG tests that had been taken on the night of Mr Salotti's presentation. It will be remembered that they were taken within possibly an hour of each other, the second of which was taken at 10:44pm. As far as the first ECG is concerned, this was reported by the computer as being an 'ABNORMAL ECG'. The abnormality is reported as being 'multiple ventricular premature complexes'. Dr Heddle regards this feature as simply an electrical artefact of the computer and not an abnormality. However, Dr Heddle told me that the ECG reveals in a number of leads ST segment depression of probably half a millimetre and that although this is non-diagnostic, it arouses suspicion of myocardia ischaemia in somebody presenting with chest pain. Therefore, to the extent that there was any abnormality in the first ECG, the abnormality had been misreported and that the true abnormality was not reported or otherwise highlighted. In itself, Dr Heddle opined that a general practitioner would probably not pick up the actual abnormal ST segment changes that he identifies in the first ECG, but suggested that a general practitioner would want to compare the first and second ECGs with a diagnostic purpose in mind.

6.26. As to the second ECG, which is reported as an 'OTHERWISE NORMAL ECG', Dr Heddle pointed out what appears to be a visible change on the trace on the number 2 lead. In fact what Dr Heddle is saying is that while the first ECG demonstrated an abnormality in respect of that lead, the feature that he regarded as abnormal had returned to normal in the second ECG. Irrespective of the fact that the ECG trace at that location had returned to normality, the change was a diagnostic feature in itself. Dr Heddle told me that when one combines that ECG change with the patient's clinical presentation of chest pain radiating down the arms, it is very close to becoming diagnostic of an acute coronary syndrome. Dr Varghese did not identify the change nor did he pick up on its significance. It will be remembered that Dr Varghese had regarded both ECGs as normal and that any changes were minor. Dr Varghese did not refer specifically in his evidence to the change on the number 2 lead that Dr Heddle identified. To the untrained observer it is not difficult to observe the change in the pattern, especially if one were looking for changes. However, interpretation of those changes is quite another matter altogether. I have not overlooked the unspoken possibility that Dr Varghese was wrong-footed by the misdescription of the abnormality on the first ECG, which Dr Heddle suggested was

not in fact an abnormality. It is possible that as a result Dr Varghese was distracted from identifying other changes that were of greater significance.

6.27. In cross-examination by Dr Varghese's counsel, Dr Heddle conceded that the observation in his report that both ECGs were abnormal had been misleading insofar as it may have suggested that they were identical. But Dr Heddle's oral evidence that there was a significance difference between them to my mind was nevertheless convincing. The difference is readily seen and Dr Heddle was not seriously challenged as to the existence of, nor the significance of, that difference. I find that Dr Heddle's evidence that there was such a difference and that this when coupled with Mr Salotti's presentation, was virtually diagnostic of an acute coronary syndrome.

6.28. Counsel assisting me, Dr Gray, asked Dr Heddle whether he would expect the change in lead 2 to be have been observed and detected by a general practitioner and to have then referred the ECGs for specialist opinion. Dr Heddle responded by saying that the obtaining of an expert opinion would have been highly appropriate in this case even if the general practitioner had not appreciated the full significance of any change. Nevertheless, Dr Heddle was very guarded in respect of any criticism that he might have directed towards Dr Varghese's handling of the ECG results. I note here that the iCCNet protocols do not spell out and direct the attention of practitioners to the presence of, and significance of, ECG changes from what is said to be an abnormality in one ECG to a state of normality in a subsequent ECG.

6.29. Irrespective of whether or not the ECG changes ought to have been identified, Dr Heddle suggested in his evidence that a repeat ECG and Troponin test would in any event have been recommended by the guidelines as needing to take place the following morning. But Dr Varghese testified that a second Troponin test prior to discharge would have been superfluous. He says that it would inevitably have been negative having regard firstly to Mr Salotti's symptom-free night and secondly to the fact that if Mr Salotti's previous episodes in the time leading up to the presentation of the evening of 13 November 2005 had been cardiac related, the first Troponin test would have been positive. As to this, Dr Heddle said that while it was possible that a repeat Troponin test might have been negative, one does not know this in advance for certain. Secondly, the repeat Troponin test is undertaken in any to demonstrate whether the patient is in the low risk category as against a high-risk category. Dr Heddle did concede that if Mr Salotti for the 2 to 3 weeks prior to his admission had

been having chest pains of the same nature, had seen a general practitioner for them the day before and had been experiencing them for an extended period on the day of his presentation, that with no new pain after the Troponin test it was acceptable to have regarded the first and only negative Troponin test as a reasonable exclusion of cardiac damage. There are three difficulties with that concession. Firstly, Mr Salotti's presentation on the evening of 13 November 2005, in spite of his bravado when alighting from the ambulance, was of a more dramatic kind than in the past. It will be remembered that the SAAS record suggests that Mr Salotti told them that he had experienced a similar attack about 12 months previously which suggests that Mr Salotti himself was not equating his symptoms of the night of 13 November 2005 with anything recent. Secondly, he is recorded as having been hyperventilating that day between attacks. He had clearly vomited after his most recent meal and was observed to have been very distressed when the ambulance crew arrived at his home. SAAS recorded his blood pressure as being 150 systolic when it had been 110 in Dr Kurian's rooms the day before. The nursing staff at the hospital also recorded that Mr Salotti had experienced a similar attack 12 months prior to this and that he was anxious. Everything suggested that Mr Salotti was acutely ill. To my mind, it is difficult to equate Mr Salotti's acute presentation on the night of 13 November 2005 with anything that he may have experienced recently. The remaining difficulty is that irrespective of what the negative Troponin test may have implied, the ECG change was virtually diagnostic of acute coronary syndrome in itself.

6.30. It will be remembered also that Dr Varghese in any case accepted that he should have repeated the Troponin test, as well as the ECG test before discharge. I agree with that analysis of the situation.

6.31. Irrespective of whether a Troponin test should have been repeated, and leaving aside what the result may have been, for Dr Heddle the salient feature of Mr Salotti's presentation was the ECG change which having regard to the symptomatology and history of the patient was virtually diagnostic of an acute coronary syndrome.

6.32. Dr Heddle stated that he would not have expected a general practitioner to have been able to detect the abnormalities on the ECGs unless they were compared with his past ECG. Indeed, Dr Heddle suggested that a general practitioner might not pick up the significant differences between the two ECGs when looking at them in isolation. However, Dr Heddle suggested that if the difference had been spotted then a general

practitioner might appreciate their significance[51]. Either way it seems to me that if the difference had been spotted, and even if the significance was not identified, an opinion from an available cardiologist through the services I have spoken of would have been appropriate before any consideration was given to discharging a patient in these circumstances.

6.33. In short, to my mind there is no doubt that during his presentation on 13 November 2005 Mr Salotti was experiencing an acute coronary syndrome and that he should have been subjected to the full rigours of the diagnostic procedures contemplated by the relevant protocol.

7. The circumstances of the death of Mr Richard John Grzywacz

7.1. Mr Grzywacz was 42 years of age when he died on Monday, 20 March 2006 at Port Lincoln. He and his wife, Margaret, lived in Port Lincoln. Mr Grzywacz did not have any previously medical history of direct cardiac significance, although he had been diagnosed with elevated blood pressure at one point in time. He had also been a smoker. He was to tell medical staff at the Port Lincoln Hospital that he had a family history of heart disease.

7.2. Mr Grzywacz's autopsy was conducted by Dr Koszyca and the cause of death was ischaemic heart disease. There was severe stenosing atherosclerosis affecting two of the three epicardial coronary arteries. Although no acute ischaemic changes were identified at the autopsy, these take a number of hours to develop before they can be seen. This does not necessarily mean that Mr Grzywacz had not experienced an acute myocardial infarction or heart attack. It may well be that he died as a result of ventricular fibrillation before any histological evidence of damage to the heart muscle materialised. However, there was microscopic evidence of ischaemic injury to Mr Grzywacz's heart that had occurred in the past. None of Mr Grzywacz's pre-existing coronary artery disease or ischaemic damage had previously been diagnosed. Moreover, there does not appear to be any evidence that Mr Grzywacz had ever knowingly experienced any acute cardiac event that had been associated with chest pain or had any other symptom of cardiac ischaemia prior to Saturday, 18 March 2006.

[51] Transcript, page 341

7.3. There were no cardiology specialists based in Port Lincoln at that time. However, a Dr Rufus McLeay practised as a resident physician in Port Lincoln. He had rooms in Port Lincoln and also practised at the local hospital.

7.4. Dr Samuel Olaiya was one of a number of general medical practitioners who practised in Port Lincoln in 2006. Dr Olaiya had gained his original medical qualifications in Nigeria where he practised for a number of years. He had also practised in Zimbabwe and South Africa as a general practitioner. He moved to Australia in 2001 and worked as a general practitioner firstly in Ceduna and then later in Port Lincoln where he was, as I say, practising in 2006.

7.5. In March 2006 Dr Olaiya was not aware of the iCCNet services, but is now aware of them and he utilises them.

7.6. Dr Olaiya gave evidence before me. He acknowledged that Mr Grzywacz was a patient of his and also recognised that Mr Grzywacz had certain risk factors for heart disease. These included hypertension and smoking. As well, Mr Grzywacz had described a family history of cardiac events. He was 42 years of age. Dr Olaiya had seen Mr Grzywacz from time to time and had done so most recently on 19 January 2006 when Mr Grzywacz presented to have his blood pressure checked and to have a rash examined. On this occasion Mr Grzywacz's blood pressure was around the 150 systolic mark which is elevated.

7.7. On Saturday, 18 March 2006 Mr Grzywacz was at home with his wife when he complained of not feeling very well. He was experiencing severe pain to the chest and jaw region and he felt nauseous, again all consistent with an acute coronary syndrome. He was taken to the Port Lincoln Hospital. Dr Olaiya was on call at the hospital when Mr Grzywacz presented.

7.8. Nursing staff at the Port Lincoln Hospital saw Mr Grzywacz in the first instance. They have recorded in the patient record[52] that Mr Grzywacz complained of a sore jaw and a slight tingle in the left hand. He had been previously hot and sweaty. The pain in the jaw had radiated to the chest and the left arm, was sharp and felt like indigestion. Quick-Eze had not relieved the pain. This was the first occasion upon

[52] Exhibit C28a, page 25

which Mr Grzywacz had felt pain of this type. His blood pressure was 160 systolic, which of course is elevated.

7.9. Dr Olaiya saw Mr Grzywacz at 6:45pm. He recorded a presentation of chest pain and the tingling sensation in the left arm. It was here that Dr Olaiya recorded Mr Grzywacz's claim of a family cardiac history. An ECG had been performed prior to Dr Olaiya seeing Mr Grzywacz. Dr Olaiya organised for Mr Grzywacz to be given aspirin and GTN spray beneath the tongue.

7.10. The ECG bears a time of 1805 hours, which would be incorrect given that Mr Grzywacz did not present until after that time. Suffice it to say the ECG that appears at page 23 of Exhibit C28a was the first ECG conducted upon Mr Grzywacz and was available to Dr Olaiya when he saw him at 6:45pm. The ECG describes itself as a 'BORDERLINE ECG'. Dr Olaiya interpreted the ECG as showing ST elevation on leads V2 and V3. Dr Olaiya could not recall whether he had made any observation at the time in respect of the V1 lead, but observed in the witness box that it also appeared to bear an ST elevation. Based upon Dr Olaiya's interpretation of the ECG, his presentation and his history, Dr Olaiya's initial impression was that there was a possibility of Mr Grzywacz potentially experiencing a heart attack. In his progress notes Dr Olaiya has recorded a differential diagnosis of 'cardiac event'. In the same note Dr Olaiya has recorded the ST abnormalities in leads V2 and V3 but not any abnormality in lead V1 of the ECG.

7.11. As to whether any other possible diagnosis occurred to Dr Olaiya at the time, he told me that there was a possibility of oesophageal reflux, angina and musculoskeletal pain in his mind. However, a cardiac event was the most likely explanation at that time.

7.12. Dr Olaiya ordered a Troponin test and continuous ECG monitoring was instigated. Mr Grzywacz was admitted. The continuous ECG has little significance in this case, as it would not show anything other than a very acute cardiac event such as an arrest.

7.13. Dr Olaiya indicated in his evidence that the pathology that was identified at Mr Grzywacz's post-mortem examination was in keeping with the ST elevation that was identified in the ECG.

7.14. Dr Olaiya reviewed Mr Grzywacz again at 9pm. On this occasion Mr Grzywacz indicated that he was in 'not much pain' but Dr Olaiya has noted that he was still

experiencing 'niggling' pains once in a while. All of Mr Grzywacz's vital signs were stable. Dr Olaiya indicated that the partial resolution of Mr Grzywacz's chest pain was in keeping with the usual response from the administration of aspirin and the GTN spray. Mr Grzywacz's systolic blood pressure had come down to 120. A Troponin test level was reported as being less than 0.03ug/L which is a negative result meaning that it did not display any evidence of an acute myocardial infarction. Nevertheless, this result in itself is not necessarily indicative of the absence of any acute event and that is why Troponin tests are repeated in the normal course of events. Dr Olaiya noted that the Troponin test should be repeated at 12:30am. I set out here the explanatory comments contained within the IMVS computerised result:

> 'Myocardial damage is indicated by a Troponin T of more than 0.1ug/L.
>
> Troponin T of more than 0.1ug/L is consistent with a possible acute myocardial infarction.
>
> Any detectable troponin is a risk factor for further cardiac events.
>
> A negative troponin test in the clinical situation of possible myocardial ischaemia should be followed by a second estimation in 6 to 10 hours to exclude the possibility of an acute coronary syndrome.'

7.15. It appears that by the time of Dr Olaiya's examination at 9pm a second, more comprehensive, 12 lead ECG had been conducted. This ECG appears at page 24 of Exhibit C28a and is reported as 'NORMAL ECG'. Notwithstanding this, Dr Olaiya again viewed the ECG as demonstrating ST elevations in the V2 and V3 leads. Again, when Dr Olaiya examined this ECG he did not observe any ST elevation in respect of the V1 lead. Both this ECG and the earlier ECG had shown ST elevation on the V1 lead. However, the ST elevation on lead V1 had diminished by the time of the second ECG which is of some significance. I return to that in due course. Suffice it to say Dr Olaiya maintained that he could not recall having noticed any abnormality in respect of the V1 leads in either ECG taken up to that point, or by extension any difference in them for that matter. However, in his evidence he acknowledged the change that had taken place as observed in the ECG traces in respect of the V1 lead, namely the diminishing of that elevation. Dr Olaiya told me that if he had noticed that change at the time, he might have regarded it as being in keeping with relief that had been established through the use of the GTN spray. He also said that the changes in the ST elevation on the V1 lead, if he had noticed them, would have led him to

make further investigations[53]. To digress, Dr Olaiya told me that following this incident he regarded himself as having been in need of further education in respect of identifying the significance of ST elevation changes. However, it does not appear to me to be at all certain what the state of Dr Olaiya's knowledge and expertise in respect of the close analysis of an ECG trace was at the time. As to what action Dr Olaiya may have taken had he noticed the ST elevation change on the V1 lead and had appreciated its significance, he said he would have called Dr McLeay the sole consultant physician resident in Port Lincoln. In the event, Dr Olaiya told me that he did not think that Dr McLeay had been available that evening. He had tried to ring Dr McLeay, not necessarily in respect of any ST elevation changes in the ECG, but because of Mr Grzywacz's general presentation.

7.16. Dr Olaiya gave directions to nursing staff that Mr Grzywacz was to be monitored continuously throughout the night and that they should administer Clexane which is an anticoagulant. This was based on his thinking that Mr Grzywacz '*could be having a probable cardiac event*'[54]. Dr Olaiya also ordered, as necessary, the administration of morphine that he said was routinely given to relieve the pain of a patient that has a cardiac problem manifesting itself in chest tightness. Clexane was administered at 9:15pm. This appears to have elicited an allergic response in Mr Grzywacz. To counteract it, Claratyne was given to Mr Grzywacz in the early hours of the following morning. I do not see any evidence of the administration of morphine in Mr Grzywacz's Port Lincoln Hospital casenotes. Evidently he did not require it, as he was not experiencing any further pain.

7.17. Dr Olaiya did not see Mr Grzywacz until the following day. However, a nursing note of 0605 hours reveals that throughout the night Mr Grzywacz's observations were stable. There is reference to the possible allergic reaction and to the administration of the Claratyne with good effect. A Troponin test repeated at about 2430 hours revealed a level of less than 0.1ug/L which, while greater than the previous result of less than 0.03ug/L, also is said not to reveal evidence of an acute myocardial infarction. Apparently the Troponin was repeated very shortly thereafter with the same result. Recorded in the nursing note are nil complaints of chest pain or jaw pain.

[53] Transcript, page 220
[54] Transcript, page 222

7.18. At 10am on 19 March 2006 the nursing note reveals that Mr Grzywacz was pain free and was keen to mobilise. No abnormalities were detected as far as his vital signs were concerned. It appears that at that stage an assessment to detect elevation in pain upon exertion was contemplated. There is no evidence that any such test was conducted.

7.19. Dr Olaiya's next contact with Mr Grzywacz occurred when Dr Olaiya was telephoned at around midday on 19 March 2006 and informed that Mr Grzywacz was trying to discharge himself. A nursing note timed at 12:20pm records that Mr Grzywacz was in fact endeavouring to discharge himself and that Dr Olaiya would be asked to review him as soon as possible. There is a notation to the effect that Mr Grzywacz's drip jelco was already removed. Dr Olaiya reviewed Mr Grzywacz at 12:40pm or thereabouts. Mr Grzywacz was indeed intent on discharging himself from the hospital. Dr Olaiya has recorded that Mr Grzywacz was much better, was pain free in the chest and noted that the last Troponin test was negative. The evidence suggested that the last Troponin test had occurred at approximately 2am and was recorded as being less than 0.1ug/L. Dr Olaiya still entertained some concern about Mr Grzywacz and told me in effect that he would have preferred if Mr Grzywacz had remained in hospital for further observation. In his note timed at 12:40pm on 19 March 2006, Dr Olaiya has written a differential diagnosis of angina. Dr Olaiya told me that he did not necessarily entertain any concern that Mr Grzywacz was experiencing symptoms of an acute heart attack or that he would have an acute heart attack imminently. However, he told me that in his view the angina that he had in mind was unstable angina because of its unprecedented onset. He devised a plan for Mr Grzywacz whereby he would be allowed to go home but would present to Dr McLeay, the specialist physician, for a stress test and that Dr Olaiya himself would see him in his own clinic. Dr Olaiya told me, and I accept his evidence, that he wanted Mr Grzywacz to remain until Dr McLeay could see him personally on the Monday morning, that is the day after on 19 March 2006. Mr Grzywacz's attitude was that he was not happy to stay because he had had a bad night's sleep. He indicated, however, that he would see Dr McLeay the following day. Dr Olaiya told me, and I accept this evidence, that he made it plain to Mr Grzywacz that he was possibly experiencing angina pain and that there was potential for a heart attack. He also told him that he would have preferred him to stay for a stress test.

7.20. Before Dr Olaiya reviewed Mr Grzywacz on 19 March 2006, before his discharge, a third ECG had been taken. This appears at page 22 of Exhibit C28a. It purports to have been taken shortly before 10am on 19 March 2006. The earlier ST elevation on leads V2 and V3 are still present. Dr Olaiya told me that he did not at that time, again, necessarily have any regard to the suspect V1 lead trace in which there had been changes since the first ECG. The third ECG is also reported as a 'NORMAL ECG'. As far as the V2 and V3 leads were concerned, I did not understand Dr Olaiya to have entertained any concern that they indicated an acute infarction or indeed heralded one. As far as the V1 lead is concerned, Dr Olaiya told me that he would have placed more emphasis on that had the significance of it been plain to him at the time. He told me in effect that he did not have the expertise at that stage of his career to make a proper interpretation of the changes on the V1 lead[55]. I return later to the precise significance of the changes in the V1 lead. I think it is fair to say at this stage that if the true significance had been identified at the time, Mr Grzywacz's management should have been different. Dr Olaiya himself said that he would have conducted further investigations and spoken to Dr McLeay as soon as he became available. I add here that given what Dr Olaiya was an episode of unstable angina, he said that if he had understood that it had come on at rest, he would have insisted that Mr Grzywacz stay in hospital. The evidence was that Mr Grzywacz had first experienced the pain while he had been playing a Playstation game, hardly a strenuous activity in itself.

7.21. Dr Olaiya did not have access to any protocols concerning possible cardiac presentations. Nor did he know of any service that would have been available to him through which he could have consulted more experienced practitioners via the telephone. I deal with this issue later in these findings.

7.22. The factors that worked against Mr Grzywacz were his own determination to leave the hospital combined with Dr Olaiya's inability to identify the true significance of the ECG results. When Mr Grzywacz made the decision to discharge himself in the afternoon of 19 March 2006 it seems clear that he did not believe that he was in imminent danger of suffering an acute and fatal cardiac event, although I accept that Dr Olaiya did explain to Mr Grzywacz that it would have been much more preferable if he had stayed in hospital. To my mind, Mr Grzywacz's decision to leave the

[55] Transcript, page 239

hospital was not a truly informed one given that the ECG results, which had not yet been properly interpreted, had not been fully explained to him.

7.23. Dr Olaiya told me that he had a telephone conversation with Mr Grzywacz on the Monday about the necessity for him to follow through on a stress test. It was on this day that Mr Grzywacz died. Mr Grzywacz's wife, Margaret, whose statement, verified by affidavit I received in evidence[56], confirms that her husband was told to make an appointment with Dr Olaiya which he thought to be on the Tuesday. Mrs Grzywacz's statement also reveals that when she left for the work on Monday morning her husband appeared to be well. When she returned from work at about 7:45pm she found her husband deceased.

7.24. There seems little doubt, and I so find, that Mr Grzywacz had presented at the Port Lincoln Hospital with an acute coronary syndrome.

7.25. Commentary upon the circumstances of the death of Mr Richard John Grzywacz
Dr Heddle provided a report in respect of Mr Grzywacz. He also gave oral evidence.

7.26. In his report Dr Heddle suggests that the critical piece of information relating to Mr Grzywacz's circumstances was the first ECG which showed precordial ST segment elevation that Dr Heddle states was no longer present in the second and third ECGs. Dr Heddle suggests that the ST segment elevation is really only prominent in the lead V1. It will be remembered that Dr Olaiya said in evidence that he could not recollect having observed anything noteworthy in respect of the V1 lead in any of the three separate ECGs. Dr Heddle in his report states that the difference between the first ECG and the subsequent ECGs was likely to be highly significant clinically. If he were to see such changes in one of his own patients, bearing in mind of course that he is a cardiologist, he would refer them within 24 hours if possible for coronary angiography. The series of ECGs were, in his mind, indicative of significant coronary atherosclerosis and potential acute myocardial infarction.

7.27. However, Dr Heddle suggested that the ECG changes were subtle and that probably only an expert would have detected them. Dr Heddle makes the observation that it cannot be expected that all doctors be experts in reading ECGs, but repeats that there are, and indeed were at the time, systems in place whereby ECGs of patients with

[56] Exhibit C9 and C9a

suspected acute coronary syndrome could promptly be referred to a cardiologist for their opinion. Like with Mr Salotti, Dr Heddle suggested that is probably what should have taken place here.

7.28. Dr Heddle does make the observation that the first ECG could possibly be '*within normal limits*'[57] for many persons. However, there was ST elevation on leads V1, V2 and V3 and in Dr Heddle's opinion the possibility of acute infarction or acute ischaemia would still need to be ruled out[58]. Although the first ECG may have reflected what was simply a normal variant, Dr Heddle gave me to understand that one would be reluctant to pass it off as a normal variant. One would, in his opinion, regard the first ECG as meeting the criteria for the patient to be taken straight to the angiograph lab for angiography. The point is, however, that it was the ECG change from one ECG to another that, as in Mr Salotti's case, had greater significance and was potentially diagnostic.

7.29. In his report Dr Heddle makes another point that is relevant. He refers to a possible 'false sense of security' as having been engendered by the normal Troponin test and cardiac enzyme test. It will be recalled that the two Troponin tests were reported as negative, albeit the first one was reported as being less than 0.03ug/L and the second was reported as less than 0.1ug/L. I did not understand that in this case any particular significance should have been placed on the fact that the second Troponin test was reported in different terms. It will be recalled that the explanatory material attached to the first Troponin test[59] suggests a Troponin T of greater than, not less than, 0.1ug/L is consistent with a possible acute myocardial infarction.

7.30. However, Dr Heddle had more to say about this particular matter when he came to give oral evidence. Dr Heddle here suggested that Mr Grzywacz's symptoms were suggestive of the possibility of an acute coronary syndrome. In particular, the history documented upon Mr Grzywacz's hospital admission as set out on page 25 of Exhibit C28a, namely 'pain in the jaw radiating to the chest and left arm, feeling like indigestion in the chest with associated nausea and tingling in the left hand', would be '*characteristic of the presentation of coronary syndrome*'[60]. Dr Heddle agreed that

[57] Transcript, page 357
[58] Transcript, page 357
[59] Exhibit C28a, page 29
[60] Transcript, page 308

the differential diagnosis of a cardiac event arrived at by Dr Olaiya was the most likely diagnosis at the time and was indeed the correct and appropriate diagnosis.

7.31. As far as the first ECG is concerned, Dr Heddle again suggests that what appears in it could well be a normal variant. He did say that the ST elevation present was suspicious of myocardial ischaemia, but that a more informative exercise would consist a comparison with subsequent ECGs, which in this case became available in due course. In any event, Dr Heddle said that when the first ECG is looked at in isolation, he would not expect a general practitioner to interpret it as showing myocardial ischemia. He suggested that some general practitioners might infer that, but repeated that in the context of chest pain there were resources within the State to obtain an expert opinion on an ECG very rapidly[61].

7.32. In Dr Heddle's opinion the second ECG was normal but the fact of its normality had significant clinical implications just as they had in Mr Salotti's case. It will be remembered that a reversion to normality has diagnostic implications of its own. In addition, Dr Heddle expected that in this case a general practitioner would have been able to pick the difference and to recognise that the change had clinical significance. Dr Heddle suggested in effect that Dr Olaiya's misinterpretation of the series of ECGs lay in interpreting the second and third ECGs as demonstrating persistent ST elevation but not appreciating that ST segment on lead V1 had returned to normality which in fact was the significant diagnostic facet of the series of ECGs[62].

7.33. In his view the changes in ECG were indicative of an acute coronary syndrome and suggested that they would override a negative Troponin test[63]. Dr Heddle reiterated that if a patient has chest pain and has either ECG changes or a Troponin rise they should have urgent cardiological referral including angiography[64]. This of course would be in accordance with the relevant protocols that were in existence at the time.

7.34. The third ECG that was taken the following morning was again normal and consistent with the second ECG, but of course the third ECG simply confirmed the fact that there had been a change in ST elevation since the first ECG had been undertaken.

[61] Transcript, page 310
[62] Transcript, page 360
[63] Transcript, page 311
[64] Transcript, page 312

7.35. It will be remembered that Dr Olaiya was keen to obtain the views of another more experienced practitioner, namely Dr Rufus McLeay, but had been unable to do so. The evidence would seem to be clear, however, that if Dr Olaiya had required some better opinion about Mr Grzywacz's presentation and ECG results, he could have availed himself of the telephonic and facsimile services that were available at the time. In cross-examination Dr Heddle reiterated his opinion that what was required here, in the absence of a practitioner being able to interpret the ECGs for himself correctly, was a reference of the series of ECGs to a cardiologist for an opinion. The same would apply where a practitioner was simply not confident in his ability to read ECGs.

7.36. As far as the Troponin tests were concerned, Dr Heddle viewed the first Troponin test as appropriately administered and that it was negative. The second Troponin test that was reported as being less than 0.1ug/L, and which apparently was repeated approximately an hour later with the same result, was also negative. To Dr Heddle the tests may have excluded a myocardial infarction at that time, but it did not exclude coronary artery disease. The negativity of the Troponin tests had to be examined against the fact that there had been ECG changes that in themselves should have dictated Mr Grzywacz's further management. Dr Heddle said this:

> 'Yes, if the initial ECG had been normal and everything else was as occurred the management as stated here would have been correct. But what was not recognised is the changes between the first and the second electrocardiogram, and with those changes the appropriate management would have been referral to a tertiary cardiology centre for the investigation, and we would expect that the patient would have been medically evacuated from Port Lincoln to the teaching hospital and then have had early coronary angiography and also have some anticoagulant therapy in addition to the aspirin.' [65]

With the change in his ECGs, the inevitable course of action should have been to have transported Mr Grzywacz to Adelaide for angiography. Dr Heddle also agreed with the proposition that Mr Grzywacz's decision to discharge himself was one that was essentially not fully informed. Dr Heddle said:

> 'A. If Mr Grzywacz had been aware that he was putting his life at risk by discharging himself he may have interpreted things differently than I would presume he was told that he'd be okay to come back in a few days and have a treadmill test and, in fact, the medical practitioner would interpret things differently as well, and that interpretation may have been conveyed to the patient.

[65] Transcript, page 316

Q. Just so I can get this clear: the advice that he make an appointment to have a stress test was, looked at in complete isolation from anything else, pretty good advice, I take it.

A. That was - would have been correct advice according to the protocol if the initial ECG had been normal and everything else was as it was. The point that was missed is that initial electrocardiogram being abnormal and that would need to - and according to the protocols that actually would change the management stream into which he went in the algorithm.

Q. Rather than wait a day or so to do the stress test, more urgent action would have been taken even before that.

A. Correct.

Q. And that would have been in the form of getting him over to Adelaide for angiography.

A. Correct.' [66]

7.37. Dr Heddle found Dr Olaiya's administration of Clexane somewhat difficult to reconcile with the rest of his management. It is seen from the iCCNet protocol that the administration of Clexane, which is an anticoagulant, is contemplated as part of the treatment where the patient is managed along the unstable angina/non-ST elevation pathway. Dr Heddle stated that one would only administer Clexane where either the initial ECG or the Troponin test was viewed as abnormal. In that event, the Clexane administration is a necessary treatment given prior to transfer of the patient for angiography. As I understand Dr Heddle, the administration of Clexane would have been more in keeping with Dr Olaiya having identified some abnormality in the first ECG test. Having accepted as I do that Dr Olaiya had not identified any abnormality in either the ECGs or the Troponin tests, I confess to not fully understanding Dr Heddle's objection to the administration of Clexane. I do not think it is suggested that Dr Olaiya's understanding of the protocol was totally perfect. Dr Olaiya himself candidly told me that he had recognised that Mr Grzywacz may have been suffering from a cardiac related illness, albeit one that to him had not been revealed by any ECG or Troponin abnormality. In the event, I make no comment about Dr Olaiya's administration of Clexane. The difficulty with Dr Olaiya's management was his inability to properly read and interpret the ECG changes, particularly on lead V1. If Dr Olaiya had himself interpreted them correctly, or had obtained an accurate second opinion about them, I have little doubt that a course of action would have been embarked upon whereby Mr Grzywacz would have been

[66] Transcript, page 318

transferred to Adelaide for diagnostic angiography and treatment. I cannot imagine for a moment Mr Grzywacz being discharged from the Port Lincoln Hospital in those circumstances.

7.38. It will be remembered that Mr Grzywacz was only 42 years of age. Dr Heddle made the observation that even with a angiographic diagnosis of what was a severe disease in Mr Grzywacz's case, he may successfully undergone a triple vessel coronary graft or event successful angioplasty and stenting. Dr Heddle suggested that Mr Grzywacz would have had a good prognosis[67].

8. The circumstances of the death of Mr Brian Leslie Sobey

8.1. Mr Sobey died on 5 July 2006 at the age of 76 years. Mr Sobey lived with his wife in premises at Prince Street, Wallaroo. Wallaroo is a centre on the Yorke Peninsula. It has a number of medical practices as well as a local hospital. At the time of his death Mr Sobey's regular general practitioner was a Dr Mei Wee who was one of the principals of a general medical practice in Wallaroo. As well, Dr Wee had practising rights at the local hospital. It seems that Dr Wee had been Mr Sobey's general practitioner since about the beginning of the decade. Dr Wee obtained her medical qualifications in Singapore in 1977.

8.2. As seen earlier, Wallaroo Hospital did not have access to the iCCNet services.

8.3. It was said during the Inquest that at some time in the past Mr Sobey had suffered from ischaemic heart disease and the suggestion was that he might have had a previous myocardial infarction in the 1980s. There is reference to this possibility in a letter to Dr Wee dated 18 March 2005 from a Dr Sam Porter who was a respiratory specialist and who had seen Mr Sobey around that time[68]. It was also said in a letter from a Dr Colver dated May 2000 that Mr Sobey suffered from angina[69]. Dr Wee told me in evidence that she understood that Mr Sobey was taking aspirin on a regular basis for a heart condition. On 21 September 2002 Mr Sobey had presented to Dr Wee complaining of a sharp pain that was suspected to be angina which had lasted for a number of hours. Dr Wee told me that she was not entirely convinced that it had actually been angina that Mr Sobey had been experiencing but it was on that occasion

[67] Transcript, page 353
[68] Exhibit C29a, page 87
[69] Exhibit C29a, page 125

that she had suggested that he take aspirin regularly. Mr Sobey also had a history of hypertension.

8.4. Mr Sobey's post-mortem examination report[70] does not mention any previous evidence of overt old myocardial infarction. Indeed, the report reveals that there is no histological evidence of acute myocardial infarction although Dr Heath points out that these changes can take several hours to develop before they can be seen microscopically and their absence does not preclude myocardial infarction as the cause of death. In addition, as we have seen, lethal arrhythmia can occur in the absence of overt infarction. However, it is clear that Mr Sobey had an enlarged heart which, paradoxically, had not identified by radiology in June 2006, a month before Mr Sobey passed away.

8.5. Whatever the position concerning Mr Sobey's previous medical history, it is clear that he possessed a number of risk factors for heart disease that included obesity and a long history of smoking that prompted Dr Porter to observe in his letter that Mr Sobey had 'amassed a very great number of pack years' by the time of his examination in March 2005. Mr Sobey admitted to hospital staff the day before he died that he was smoking in excess of 60 cigarettes per day.

8.6. In the late afternoon of Tuesday, 4 July 2006 Mr Sobey was at home and at that time told his wife that he was experiencing pain in the chest, left side back shoulder and down his left arm. Mr Sobey drove himself to the Wallaroo Hospital which was only a few minutes away. Mr Sobey told his wife that he had taken four Panamax tablets in the previous hour which suggested that the pain had been experienced for some time that afternoon before Mr Sobey had made his complaint of it. Dr Wee was to tell me that before Mr Sobey attended at the hospital he had telephoned her at her clinic and had told her that he was experiencing chest pain and wanted to know what he should do. Her advice to him was that he should attend at the hospital and have an ECG undertaken. Dr Wee had then telephoned the hospital and had advised the nurses that Mr Sobey would imminently arrive there and that they should perform an ECG. Dr Wee was not to attend at the hospital to see Mr Sobey until she had finished her clinic for the day, but I note that she arrived at around 6pm so the delay was not significant.

[70] Exhibit C14a

8.7. The Wallaroo Hospital record of his presentation records that Mr Sobey was triaged at 5:15pm. The nursing staff have recorded that Mr Sobey was experiencing pain in the left side of his chest that was radiating down the left bicep. Mr Sobey's blood pressure was not elevated. An ECG was undertaken and Dr Wee was to interpret this as not demonstrating any abnormality. It is possible that two ECGs were performed at that time, namely 5:20pm[71]. Dr Wee arrived at the hospital and examined Mr Sobey from about 6pm onwards. Dr Wee has recorded:

'Gripping Ⓛ sided chest pain → Ⓛ arm since this afternoon'

It appears that he complained that this pain had persisted for about 4 hours but that, according to Dr Wee's note, it had eased by the time of her examination. In evidence, Dr Wee interpreted this as meaning Mr Sobey was no longer experiencing any pain at the time of this examination, but later nursing observations made before Mr Sobey's discharge that night suggest that he was at least experiencing some pain in the arm when he left.

8.8. Dr Wee ordered a Troponin test which was the only Troponin test that Mr Sobey experienced. Dr Wee told me that she had left the hospital by the time the Troponin test was reported. She had left instructions that she be telephoned at home with the result. Later that evening the result was made available to Dr Wee. At that stage Mr Sobey was still at the hospital.

8.9. Dr Wee told me that Mr Sobey looked comfortable clinically. He was not sweaty, he was not in distress, there was no shortness of breath and his blood pressure and pulse were satisfactory. To Dr Wee, those symptoms singly or collectively would have been highly suggestive of an acute coronary syndrome, but they did not exist in Mr Sobey's case. Having examined Mr Sobey and his ECG, Dr Wee told me that she informed him about the result. Her differential diagnosis at that time included whether Mr Sobey could possibly be developing an acute coronary syndrome, whether it was a case of severe angina that had settled or whether it could be musculoskeletal in nature. When asked as to whether she favoured any one or other of those differential diagnoses, Dr Wee said that at the time of her examination and before the blood test result was available, she thought that Mr Sobey was either experiencing an acute coronary or severe angina that had settled. Dr Wee told me that

[71] Exhibit C29a, page 144 and 145

Mr Sobey was very unenthusiastic about the prospect of being admitted to Wallaroo Hospital overnight. He wanted to go home. This wish on Mr Sobey's part is corroborated in the statement, verified by affidavit, of Kaye Elizabeth Walkington[72] who was one of the Registered Nurses on that evening and who has compiled and signed Mr Sobey's discharge note signed at 7:20pm. Mr Sobey in her words was 'keen to go home'[73]. Dr Wee agreed that Mr Sobey could go home if the Troponin test results, which were not yet available, turned out to be negative. In addition, she told me that she gave Mr Sobey to clearly understand that if he were to experience any further chest pains he should re-present at the hospital. In the event, Mr Sobey drove himself home. Dr Wee left the hospital before Mr Sobey. When the Troponin test became available later that evening the result was phoned through to Dr Wee. The Troponin test result is at page 146 of Exhibit C29a and records the fact that the blood test was collected at 6:20pm with the result of the test being printed at 6:54pm. The Troponin test result was less than 0.03ug/L and was reported as negative. The Troponin test, it will be observed, was taken approximately 5 hours after Mr Sobey's reported onset of chest pain. The Troponin test would have had greater significance if it had been taken after 6 hours from the onset of symptoms. In the event, the Troponin test had limited evidentiary value in terms of refuting the existence of an acute cardiac event. The same goes for the ECG result which I will come back to in due course.

8.10. And so Mr Sobey was allowed to leave under his own power. At that time Registered Nurse Walkington recorded that Mr Sobey had reported that the pain in the arm had settled and was now 2 out of 10. This indicates that although Mr Sobey's pain had resolved, perhaps to a greater degree, he was still experiencing some pain. I note here that Mr Sobey had been given GTN spray at 6:10pm. He had not been given any blood thinning aspirin because it was understood by Dr Wee that he was taking aspirin regularly in any event.

8.11. According to Mrs Sobey[74], when Mr Sobey arrived home, he told her that his pain was actually very bad to the point where she persuaded him to lie down. Mrs Sobey obtained a heat pack for his pain. Mr Sobey also took two more Panamax. The description of Mr Sobey's discomfort as give by his wife seems at odds with that

[72] Exhibits C16 and C16a
[73] Exhibit C16a, page 2
[74] Exhibit C15a

described by nursing staff. The contemporaneous note made by Registered Nurse Walkington at that time of Mr Sobey's discharge states 'Pt also reports pain in arm settled now 2/10. Pt happy to drive home'. It carries an upbeat flavour that is consistent with Mr Sobey having been reassured by the negative Troponin test result and the diminishing of the pain in his arm to a significantly lower level. For instance, he volunteered that he was happy to drive home.

8.12. At about 12:15am the following morning Mr Sobey came into the kitchen and sat at the table where his wife also happened to be sitting at that time. Having sat down he collapsed and died. An ambulance was called but they were unfortunately unable to revive him.

8.13. In her evidence, Dr Wee accepted that the single Troponin test had limited significance having regard to the fact that it was taken approximately 5 hours after the onset of chest pain. The single ECG result was of somewhat limited significance as well. Ideally, of course, both of those tests should have been repeated and Mr Sobey should have been admitted overnight for that purpose. Dr Wee, was asked:

> 'Q. Why did you make that judgment, that he could go home then and not stay in hospital.
>
> A. That judgment was - normally I would, in the majority, in all the patients even at that time I would have admit a patient who come in complaining of chest pains and for further management, monitoring, for further blood tests, not just a one-off blood test. But Mr Sobey, because of the way he presented and that the chest pain has altogether gone when I saw him, and because his basic blood pressure, pulse, was normal, the way he look at the time I saw him being comfortable, he was not in distress, and that the ECG was normal and over - and he was very keen to go home, and he fully understood that he is to come back when his - if, overnight, if his chest pain gets worse.
>
> Q. Would you - do you have a different practice now or would you have a different practice now.
>
> A. Definitely so since this incident.
>
> Q. What would you do now if in the presence of a patient asking you to be released overnight.
>
> A. I will be more - I would probably - I would be more firm with a patient as far as insisting that they be - at least they stay overnight and we monitor and not let patients' wishes override my clinical judgment.' [75]

[75] Transcript, pages 382-383

However, Dr Wee then suggested that she took some comfort from the negative Troponin test and at one point said she believed that the Troponin test would have been positive if he had been experiencing acute coronary syndrome[76]. In the event, I do not think Dr Wee persisted with that rather bullish interpretation of the Troponin test result, particularly having regard to the fact that the negative result taken within the first 6 hours of the onset of symptoms may not have any particular significance. In her evidence in chief Dr Wee suggested that ideally Mr Sobey should have been admitted so that further blood tests could have been repeated, including a CK test.

8.14. In the event, I am not persuaded that when Mr Sobey elected to go home, he had been fully informed of the risks of him doing so. On the contrary, the impression is that he had been somewhat reassured by a number of factors that included the partial resolution of his symptoms and the negative ECG and Troponin test. This impression seems to have been engendered by the fact that the significance of negative ECG and Troponin tests in the circumstances was not fully explained to him. While Dr Wee states that she did stress the need to return if Mr Sobey re-experienced chest pain, and although she explained that a normal ECG does not tell the doctors very much, the negativity of the Troponin test and its significance was never really explained to him. Indeed, Mr Sobey was no doubt alive to the fact that his being able to leave the hospital was contingent upon the Troponin test being negative and upon Dr Wee's satisfaction, based on that result, that he could leave. The Troponin test was negative, so he left.

8.15. When Dr Wee was pressed on the issue of whether Mr Sobey had really made a fully informed decision to leave the hospital, Dr Wee told me that even if Mr Sobey did not re-present overnight with further chest pains she would have reviewed him in the morning. She told me that she intended to call him in the morning to follow-up for a further ECG and probably another Troponin test and to refer him to a cardiologist. I add here that Wallaroo Hospital did not have any specialist cardiologists resident there, but two cardiologists visited from time to time and, in any event, Dr Wee could, one assumes, make a referral at any time to a specialist in Adelaide. I also add here that Dr Wee told me that she had access to cardiology advice if she wanted it. On this occasion, however, it is clear that she did not avail herself of that resource. In making her claim that she intended to contact Mr Sobey the following morning, Dr Wee

[76] Transcript, page 383

pointed to a notation in her writing in the hospital record to the effect that she would review Mr Sobey PRN. PRN is a medical abbreviation signifying that an event will be carried out when necessary. In the circumstances, I do not regard that entry as necessarily conveying an intention on Dr Wee's part to contact Mr Sobey the following morning in any event, irrespective of whether or not he experienced further symptoms. My hesitation in accepting Dr Wee's evidence about her intention to contact Mr Sobey the following morning was exacerbated by the fact that in neither of two letters that Dr Wee provided to the investigating police in this matter did she make any claim that she intended to contact Mr Sobey the morning after her consultation with him at the hospital. The first of these letters[77] was in fact written on the day of Mr Sobey's death. In this letter Dr Wee records:

> 'Mr Sobey was happy to drive home but was instructed by myself earlier on and also the nurse before discharge that he was to return to hospital should the chest pains recur in the night.'

There is no suggestion of any further consultation as such the following day. The second letter to the investigating police was written by Dr Wee on 17 March 2007 and it is clear from the terms of that letter that prior to writing it Dr Wee had been furnished, and I infer had read, with Dr William Heddle's report concerning Mr Sobey. In that report Dr Heddle had expressed[78] the opinion that correct management of Mr Sobey would have included keeping him in hospital and repeating the Troponin test and the ECG the following morning. He observed that if those tests had been normal it would then have been reasonable to discharge him home. The tone of Dr Heddle's report is somewhat critical of the decision to allow Mr Sobey to go home. That criticism of course would be ameliorated by any arrangement that Dr Wee may have put in place, or had intended to put in place, to contact Mr Sobey the following morning with a view to performing the very tests that Dr Heddle should have been conducted before Mr Sobey's discharge from hospital. In Dr Wee's letter of 17 March 2007[79] she acknowledges receipt of Dr Heddle's report and emphasises, among other things, Mr Sobey's lack of enthusiasm to remain at the hospital. Dr Wee does not suggest in this letter that she had any intention to perform repeat ECG or Troponin tests the following morning when one would have expected her to do so. When the omission to mention her intentions in this regard was pointed out to Dr Wee in her

[77] Exhibit C37
[78] Exhibit C34
[79] Exhibit C37a

evidence, she was unable to explain why she had made that omission[80]. In the event, I am not persuaded that Dr Wee did have any such intention. To me it seems unlikely. Dr Wee had an expectation in her mind that if Mr Sobey were to have experienced chest pains again, he would inevitably have re-presented at the hospital. Dr Wee in her letter of 17 March 2007 makes this observation:

> 'Wallaroo is a small country town. It will take no more than 5 minutes of travel to return and the ambulance service is very efficient. I felt that he could go home as he had understood the instructions that were given. Unfortunately Mr Sobey's chest pains returned and became worse when he came home and, for reasons known only to Mr Sobey, he failed to re-present himself back at the hospital. Apparently the chest pains continued for about 3 hours before he collapsed and could not be resuscitated.'

As seen earlier, the failure of Mr Sobey to re-present himself back at the hospital may have stemmed from a conviction on his part that he was not experiencing any life-threatening event. It is perhaps not entirely surprising that Mr Sobey did not return to the hospital. On the other hand, to be fair to Dr Wee, she understood that Mr Sobey would so return if he experienced chest pains again. The only comment I would make about that is that, were Mr Sobey to have an extremely acute event that required urgent treatment at a tertiary centre in Adelaide, he may as well have been several hours from the Wallaroo hospital as opposed to 5 minutes.

8.16. In cross-examination by Dr Gray, counsel assisting, Dr Wee agreed that the history of pain coming on at rest was suggestive of the possibility of Mr Sobey experiencing acute coronary syndrome. She conceded that she had placed too much trust or dependence on the fact that Mr Sobey would return if the pain became worse. She admitted that upon discharge she thought that Mr Sobey had experienced a severe angina attack that had settled at the moment, although she had thought it unlikely that he was experiencing an acute coronary syndrome at the time. Dr Wee admitted that she did not give consideration to any of Mr Sobey's previous history of heart problems. She agreed in hindsight that he should not have been allowed to go home[81]. She admitted that irrespective of his past history, the fact that Mr Sobey had left-sided chest pain that was gripping in nature was quite indicative that it was of cardiac origin.

[80] Transcript, page 402
[81] Transcript, page 393

8.17. It seems clear to me, and I so find, that Mr Sobey was experiencing an acute coronary syndrome while at the Wallaroo Hospital.

8.18. Commentary upon the circumstances of the death of Mr Brian Leslie Sobey

Dr Heddle's written commentary about Mr Sobey's circumstances is contained within his report Exhibit C34. In that report Dr Heddle observes that although initial ECG and blood tests were normal, in reality neither of them had excluded a cardiac cause. Dr Heddle was in any event guarded about the utility of the single Troponin test given that it was taken approximately 4 hours after the onset of symptoms when the relevant protocols suggest that they should be taken not before 6 hours. Dr Heddle also observes in his report that it would have been appropriate for Mr Sobey to have been given aspirin as well as the sublingual GTN which was administered upon his arrival.

8.19. The correct management would have consisted of keeping Mr Sobey in hospital and in due course repeating the blood tests and ECG. If the results had still been normal it would only then have then been reasonable to discharge him. Dr Heddle believes that there is a reasonable probability that either the repeat ECG or Troponin test would have been abnormal and that if Mr Sobey had been kept in hospital for this purpose he would possibly have avoided, or at least received correct treatment for, the cardiac episode which caused his death. Dr Heddle opines that although the outcome may have been the same, in his view there was a possibility that Mr Sobey might have survived if he had stayed in hospital. He also points out that if Mr Sobey had gone into ventricular fibrillation while in the Wallaroo Hospital, he may have been shockable.

8.20. Dr Heddle was critical of the decision to allow Mr Sobey to leave the hospital without repeat ECGs and Troponin testing. Exacerbating this situation was the fact that, although Mr Sobey's pain had settled somewhat, he was still reporting pain in the arm at the time of discharge. In the absence of a specific cause for chest pain having been determined, it was not appropriate for the patient to be discharged. Dr Heddle gave me to understand that Mr Sobey's partial pain relief was in any event not indicative of any real improvement in Mr Sobey's presentation. Dr Heddle explained that while the pain can come and go, one still has the underlying potential difficulty, namely plaque rupture being the basis of the acute coronary syndrome.

8.21. Dr Heddle disagreed with counsel's suggestion that it would be reasonable for a doctor to allow a patient to go home overnight on the understanding that he would return to hospital if the pain came back. The basis for Dr Heddle's disagreement with that proposition was firstly, that the protocols and guidelines did not allow for that scenario and secondly, it was in any event potentially hazardous to do so.

9. Conclusions

9.1. In each instance each of the four men with whom this Inquest is concerned presented with an acute coronary syndrome.

9.2. Although in some instances the relevant medical practitioners suspected or believed that the man's symptoms may have been due to cardiac illness, none of the four men were believed to be experiencing an acute coronary syndrome as such.

9.3. In Mr Dalling's case, when he was seen by Dr Mikhail insufficient attention was paid to Mr Dalling's risk factors of previous heart disease and diabetes. While Dr Mikhail could not be held responsible for not knowing about Mr Dalling's previous ECG result, there was material available to Dr Mikhail upon which he should have concluded that Mr Dalling was a known diabetic.

9.4. In Mr Dalling's case, Mr Dalling should have been subjected to the relevant protocol and been administered with an immediate ECG at the local hospital.

9.5. None of the medical practitioners involved in the care of the four men specifically referred to any protocol or clinical pathway designed to identify an acute coronary syndrome and to diagnose heart disease.

9.6. Although Dr Olaiya in Mr Grzywacz's case wanted to obtain an opinion concerning Mr Grzywacz from the local physician, Dr McLeay, none of the practitioners involved in the care of the four individuals sought expert or specialist assistance in attempting to diagnose each individual.

9.7. In the cases of Mr Salotti and Mr Grzywacz, the treating medical practitioners did not identify ECG changes that, together with the symptomatology of each man, was highly indicative, if not diagnostic, of an acute coronary syndrome in each case.

9.8. In the cases of Mr Salotti and Mr Grzywacz, no expert or consultant assistance was sought in relation to the interpretation of the ECGs when such assistance would have

been available. If such assistance had been sought, there is little doubt that the ECG changes would have been regarded as indicative of an acute coronary syndrome.

9.9. In Mr Sobey's case, Mr Sobey only underwent the one ECG and one Troponin test, both of which were negative. Repeat ECG and Troponin tests were not performed. Mr Sobey was discharged in circumstances in which, to my mind, he was not fully informed of the risks involved in leaving the hospital.

9.10. It is not possible to conclude with certainty whether any of the individuals would have survived if they had been treated differently or if their acute coronary syndrome had been identified. On the other hand, if the acute coronary syndrome in each instance had been identified, transfer to a tertiary hospital in Adelaide would have been arranged with a view to each man receiving invasive diagnostic measures such as cardiography. However, in Mr Sobey's cade, transfer for that purpose may not have occurred in time.

9.11. An inference is available, which I draw, that in each instance the individual's chances of survival would have been better if the acute coronary syndrome in each case had been identified.

9.12. In my view, the evidence demonstrates that over the period with which this Inquest was concerned, there was a widespread ignorance of the existence of the iCCNet Management of Chest Pain / Suspected Acute Coronary Syndrome protocol and a widespread ignorance of or a reluctance to utilise the 24-hour, 7 day per week service that I have identified at the beginning of these findings.

9.13. The evidence also demonstrated to me that the skill and ability of general practitioners who practice in the country to read and interpret ECGs is not uniform and in some instances was lacking.

10. Recommendations

10.1. In my opinion this Inquest has identified a clear need for further and continuing education to be directed to general practitioners in respect of the identification of acute coronary syndrome presentations and acute coronary events generally, especially to those general practitioners who practise in the country.

10.2. As far as the issue of education and training is concerned, the need to have ECGs referred for specialist opinion has to be balanced against the undesirability of

unnecessarily over-burdening the available services. In his evidence Dr Tideman highlighted the undesirability of a situation developing whereby all ECGs conducted in country centres would be referred for specialist opinion. The capacity for the iCCNet service, for example, to deal with such a scenario would be overstretched. Dr Tideman said this:

> 'No, we have to defer to the judgment of the GP, again largely because of capacity issues, but a GP has to have some appreciation of their level of confidence in interpreting the ECG. There are many longstanding GPs in country areas who have a high level of confidence in their ECG interpretation skills and do not need us to add to their assessment. Then on the other end of the spectrum there are quite a number of GPs - and, to be honest, the overseas trained medical practitioners would be over-represented in that group, which is a well recognised fact - who would have low levels of confidence and may refer ECGs to us for interpretation, and it is fairly easy to determine whether they are normal or abnormal. We provide that information. We understand it is on the treating doctor's confidence and their abilities and therefore we will always leave it up to the GP to fax us an ECG and, even if we think there is an ECG that doesn't need to be faxed, we will provide that service because it is designed to support the GP intrinsically.'[82]

10.3. Dr Tideman also referred to the desirability of general practitioners understanding the need to employ what he referred to as a '*global risk assessment*'[83] when presented with possible cases of acute coronary syndrome. The risk assessment ought to take into account risk factors such as the patient's history and their symptomatology when all of that is examined in the round.

10.4. Dr Tideman also agreed that there was a need for general practitioners to be subjected to a period of orientation before they are permitted to practice in an unsupervised manner in the country. He said this:

> 'It should be part of the orientation, if they are an overseas-trained doctor, going into a country setting, having not trained here or in the postgraduate training, it is expected of practitioners before they go into solo or unsupervised practice in the country, one would expect that these things would be a mandatory part of the curriculum if you like.' [84]

10.5. As to the other area of need, Dr Gray who appeared as counsel assisting during the course of the Inquest, invited me to consider making a recommendation that the Department of Health consider a review of information sharing practices between country general practitioners and country hospitals with a view to implementing

[82] Transcript, pages 514-515
[83] Transcript, page 565
[84] Transcript, page 572

systems that allow for a greater flow of information to assist in the continuity of care. In this regard Dr Gray referred to the scenario that applies in country centres whereby the local general practitioners staff the local hospitals but that the practitioner who sees the patient in the hospital may not necessarily be the practitioner who has been seeing the patient on an ongoing basis. I am not entirely certain that the scenario that Dr Gray identified had any impact on any of these cases before me. In Mr Dalling's case the Yorketown Hospital was in possession of an ECG that demonstrated that Mr Dalling had quite likely suffered a myocardial infarction in the past, but I observe that the same ECG formed part of Mr Dalling's file at the Yorketown Medical Practice. While I do not intend to make any recommendation in this regard, I would nevertheless endorse the desirability of information sharing between practitioners and hospitals in country settings where possible.

10.6. Pursuant to section 25(2) of the Coroner's Act 2003 I am empowered to make recommendations that in the opinion of the Court might prevent, or reduce the likelihood of, a recurrence of an event similar to the event that was the subject of the Inquest. I make the following recommendations:

1) That the Minister for Health, in conjunction with other stakeholders and interested entities, cause training programs to be directed towards the education of general practitioners, and in particular those general practitioners who are practising or who aspire to practise in country areas, as to the following:

   a) The importance of informing oneself of and examining a patient's risk factors for heart disease when considering, determining and identifying presentations of acute coronary syndrome;

   b) The importance of conducting a global risk assessment when endeavouring to identify an acute coronary syndrome that takes into account, on a global in-the-round basis, the patient's risk factors for heart disease and the patient's symptomatology;

   c) The importance of general practitioners having regard to observations made by paramedics of patients presenting with suspected acute coronary syndrome;

d) The importance of obtaining, if possible, records of previous presentations of the patient that might be relevant to the identification of an acute coronary syndrome, for example, previous ECGs taken in respect of the patient;

e) The need to study and understand the relevant protocols, guidelines and clinical pathways that are available to general practitioners such as the 'Management of Chest Pain / Suspected Acute Coronary Syndrome' protocol promulgated by iCCNet; as well as the 'National Heart Foundation of Australia, Cardiac Society of Australia and New Zealand Guidelines for the management of acute coronary syndromes';

f) The need to rigorously follow the acute coronary syndrome diagnostic pathways to their conclusion, with particular emphasis in respect of cases where the Low Risk Protocol has been embarked upon;

g) The importance of general practitioners considering what might be the worst case scenario when a patient presents with chest pain and/or other symptoms of an acute coronary syndrome; and the importance of identifying with some certainty an explanation for those symptoms before dismissing them as not being due to an acute coronary syndrome;

h) To promote amongst general practitioners, especially those who practice in country areas, and especially those who have been trained overseas, greater knowledge, skill and confidence in interpreting ECGs;

i) To promote a greater understanding amongst general practitioners of the need to compare ECGs within a series so as to more readily identify the presence of, and significance of, ECG changes within that series;

j) To promote a greater understanding amongst general practitioners of the need to identify occasions upon which there is a need to refer ECGs for expert specialist interpretation such as that provided by iCCNet; and to promote a greater awareness of the availability of such specialist services;

k) The importance of providing patients with sufficient information to enable them to make fully informed decisions as to whether they should receive

further diagnostic measures and treatment in respect of a suspected acute coronary syndrome;

l) The importance of general practitioners understanding the pathophysiology involved in an acute coronary syndrome.

2) That the Clinical Director of iCCNet give consideration to amending the 'Management of Chest Pain / Suspected Acute Coronary Syndrome' protocol so as to include within it specific reference to:

i) The need to keep in mind that single ECG and Troponin test results that are negative do not exclude the presence of an acute coronary syndrome;

ii) The importance of examining, within a series of ECG tests, the presence of, and significance of, ECG changes within that series;

iii) To identify appropriate occasions for referral of ECGs for expert specialist opinion.

3) That the Royal Australian College of General Practitioners and the Royal Australian College of Rural and Remote Medicine, as overseen by the South Australian Medical Board, include as part of their curricula more rigorous training of prospective admittees to the Colleges with respect to the identification of acute coronary syndrome with particular emphasis upon (a) interpretation of ECG results and the need to look for and identify the presence of and significance of changes in a series of ECG results; and (b) the utility of, the necessity of proper timing for and the significance of Troponin levels; and that the Colleges include these topics as part of their examination material.

4) That the Minister for Health cause advice to be promulgated to general practitioners relating to the need for general practitioners to highlight within their individual patient records the presence of risk factors for heart disease in respect of those patients.

5) That the Minister for Health cause careful scrutiny to be maintained of the expertise and knowledge of overseas trained medical practitioners in respect of the identification of symptoms of acute coronary syndrome, and in particular as to their level of skill and expertise in the interpretation of ECGs.

Key Words: Country Areas - Medical Services; Heart Disease; ECG Interpretation

In witness whereof the said Coroner has hereunto set and subscribed his hand and Seal the 30th day of March, 2009.

Deputy State Coroner

Inquest Number 35/2008 (3916/2004, 2948/2005, 0386/2006, 0974/2006)