

CITATION: *Inquest into the death of Dion Daniels* [2014] NTMC 024

TITLE OF COURT: Coroners Court

JURISDICTION: Darwin

FILE NO(s): D0033/2012

DELIVERED ON: 3 November 2014

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HEARING DATE(s): 9 - 11 September 2014

FINDING OF: Mr Greg Cavanagh SM

**CATCHWORDS:** **Death of young Aboriginal man from coronary artery thrombosis; early recognition/diagnosis of coronary artery disease in young Aboriginal people in particular**

**REPRESENTATION:**

Counsel Assisting: Mr Mark Thomas

Sunrise Health Service

Aboriginal Corporation:

Alistair Wyvill SC on day one of inquest;  
Mr Chris McDuff for remainder of inquest

Department of Health:

NAAJA acting on behalf

of family of deceased:

Mr Nigel Linklater and

Ms Sujin Le:

Mr Greg McDonald

Mr Jared Clow

Mr Ron Hope

Judgment category classification: B

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IN THE CORONERS COURT  
AT DARWIN IN THE  
NORTHERN TERRITORY OF  
AUSTRALIA

No. D0033/2012

In the matter of an Inquest into the  
death of  
**DION DANIELS**  
**ON 19 FEBRUARY, 2012**  
**AT SUNRISE MEDICAL CLINIC,**  
**NGUKURR**

## FINDINGS

Mr Greg Cavanagh SM:

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## **PART 1: INTRODUCTION**

1. At the commencement of this inquest I stated to those members of Mr Daniels' family who were present in court that these findings are to be read to them at Ngukurr community by the relevant senior Police officer located there. Mindful of this, I shall endeavour to strive for clarity and plain speaking in these findings, especially in regard to the medical terminology that must be used.
2. Mr Daniels was born on 2 June 1987 at Katherine. His life up until his death was, relatively speaking, uncomplicated. He was married with two young children, had obtained work, and played in a local Australian Rules football team on the weekends. On the afternoon of 19 February 2012 Mr Daniels was playing football in a competitive game at Ngukurr oval. The game commenced at about 4.30pm. It started deliberately late in the day due to the heat at that time of the year. The maximum temperature that day in Ngukurr was 38 degrees (Celsius). Mr Daniels played for the first half of football. The first half consisted of two quarters of approximately 20<sup>1</sup> minutes in duration. There was a break at half time. Other people saw Mr Daniels at this point. He complained of being dizzy and feeling "pain inside". He didn't say where the pain was located although he did say that he was getting a lot of cramps in his fingers. He was not taking any medication at the time. He had not been seen to be struck or to be the subject of any untoward conduct during the course of the game. He was neither a diabetic nor an asthmatic and had not previously suffered a heart attack.

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<sup>1</sup> The umpire in an Australian Rules football game traditionally "stops the clock" when the ball goes out of play, amongst other things, so that a quarter frequently takes about 30 minutes in real time: this is standard procedure for Australian rules games from the elite level (the Australian Football League) down.

3. Mr Daniels was also observed at this point to be sweating a lot and to be breathing very hard despite the passage of the half time break<sup>2</sup>, which would normally be sufficient to allow most players to regain their breath. One of the people present, Mr Owen Turner, was so concerned for Mr Daniels that at the end of the half time break he drove him directly to the medical clinic, which was run by Sunrise Health Centre. There was nobody at the clinic at the time (just before 5.45pm). Mr Turner used the emergency telephone at the clinic and spoke to the nurse on duty, Mr Nigel Linklater, who at that point was located elsewhere. He arrived at the clinic, a couple of minutes later, at about 5.45pm. Mr Turner told Mr Linklater that Mr Daniels had complained of feeling dizzy, short of breath, getting cramps and feeling pain inside his body. Mr Daniels could not walk from the car to the clinic. A wheelchair was obtained to take him the short distance to the clinic.
4. At the clinic Mr Linklater did a number of things- according to his police statement. It should be noted at this point that Mr Linklater was not able, due to his own health problems, to give evidence in this inquest. Mr Linklater said, in his police statement<sup>3</sup>, that Mr Daniels was very tired, that he looked exhausted and that he was soaking wet with sweat. Mr Linklater added<sup>4</sup> that he was unable to obtain Mr Daniels' blood pressure then (or subsequently). Mr Linklater said<sup>5</sup>, further, that Mr Daniels complained of cramp in all his limbs. Mr Linklater said<sup>6</sup> that that the symptoms fitted a provisional diagnosis of dehydration. He said<sup>7</sup> he gave Mr Daniels water to sip. After Mr Linklater said<sup>8</sup> that he made two failed attempts to insert a cannula into Mr Daniels' arm, one was eventually inserted at about 6.30pm<sup>9</sup> by Registered Nurse Joan Tibballs who had briefly looked in on Mr Daniels

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<sup>2</sup> In Australian Rules Football the half time break is usually about 20 minutes

<sup>3</sup> Nigel Linklater, statement to police, dated 8 March 2012, paragraph 2

<sup>4</sup> Nigel Linklater, Statement to police, dated 8 March 2012, paragraph 3

<sup>5</sup> Nigel Linklater, Statement to police, dated 8 March 2012, paragraph 3

<sup>6</sup> Nigel Linklater, Statement to police, dated 8 March 2012, paragraph 3

<sup>7</sup> Nigel Linklater, Statement to police, dated 8 March 2012, paragraph 3 and 4

<sup>8</sup> Nigel Linklater, Statement to police, dated 8 March 2012, paragraph 4

<sup>9</sup> See Patient notes.

at about 6.15pm at the request of Mr Daniels' family, some of whom were waiting outside the clinic. After Ms Tibballs inserted the cannula an IV drip was set up. After she did this Mr Linklater said that he was right to continue alone. Nurse Tibballs then left the clinic at shortly after 6.30pm. Mr Linklater administered<sup>10</sup> to Mr Daniels a one-litre bag of intravenous saline solution at 6.45pm. At 7.45pm he said<sup>11</sup> he gave him another 500 mls of saline solution intravenously. This was consumed in about 30minutes. In addition,<sup>12</sup> he set up another bag of saline solution, this time of 500mls to give to Mr Daniels at 8.15pm.

5. At 8.15pm Mr Linklater said<sup>13</sup> that Mr Daniels without warning became unresponsive and slumped to the side of the bed. His pupils were fixed and dilated. He took two long breaths about a minute apart.
6. At 8.17pm Mr Linklater said<sup>14</sup> that he commenced CPR (cardio-pulmonary resuscitation). Mr Daniels vomited at 8.25pm.
7. At 8.30 pm Mr Linklater said<sup>15</sup> he phoned his colleagues Nurses Sujin Lee and Joan Tibballs to assist him. He asked Nurse Lee to contact the Dr Tanya Davies. At 8.35 Nurse Joan Tibballs arrived. Nurse Sujin Lee and Dr Davies arrived at 8.40pm.
8. On the arrival of other medical staff Mr Daniels was attached to a defibrillator and was administered a shock on one occasion. He was given adrenaline six times. CPR continued to be administered to Mr Daniels. Regrettably, there were no signs of life.
9. Mr Daniels was declared dead at 9.15pm. He was 24 years old.

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<sup>10</sup> Nigel Linklater, Statement to police, dated 8 March 2012, paragraph 4

<sup>11</sup> Nigel Linklater, Statement to police, dated 8 March 2012, paragraph 5

<sup>12</sup> Nigel Linklater, Statement to police, dated 8 March 2012, paragraph 6

<sup>13</sup> Nigel Linklater Statement to police, dated 8 March 2012, paragraph 6

<sup>14</sup> Nigel Linklater Statement to police, dated 8 March 2012, paragraph 7

<sup>15</sup> N Linklater Statement to police, dated 8 March 2012, paragraph 8

10. During the period from 5.45pm to 8.15pm whilst Mr Daniels was, except for a brief period, solely in Mr Linklater's care, Mr Linklater did not contact or attempt to contact the District Medical Officer or Rostered Rural Medical Practitioner whose advice was available by telephone (or facsimile) on a 24 hour basis seven days a week to, relevantly, medical staff operating from Sunrise. It made no difference that Sunrise was a private service and not part of the Department of Health. Expert cardiology advice was available if this phone service had have been utilised. Further, Mr Linklater did not contact Dr Tanya Davies who was the general practitioner at the clinic who was off duty at the time but in Ngukurr. In addition, Mr Linklater did not utilise or seek to utilise an ECG (electrocardiograph) machine during the key period specified above. In fact, the ECG machine that was in place at the clinic was not used until after the resuscitation process had commenced, by which time it was clearly too late. Moreover, Mr Linklater did not obtain any blood pressure reading of Mr Daniels- at any point. Finally, Mr Linklater did not obtain any Troponin<sup>16</sup> levels of the Mr Daniels. Instead, he proceeded on a path that was predicated upon his provisional diagnosis of dehydration. In reality, in the crucial two and a half hours between 5.45pm and 8.15pm Mr Daniels, who had already sustained a blockage to his left anterior descending artery whilst playing football or shortly after playing football that afternoon, was desperately clinging to life and in urgent need of immediate medical assistance to attend to the problem, and hence save his life. It never came. After he suffered a second, probably catastrophic cardiac event at 8.15pm (when he collapsed after a cardiac arrest) any treatment after that point was, almost certainly too late to save his life.

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<sup>16</sup> Troponin is a cardiac enzyme, which consists of three regulatory proteins that is found in cardiac and skeletal muscle. Certain types of Troponin are very sensitive and specific indicators of damage to the heart muscle. A person who has had a myocardial infarction or who is experiencing cardiac problems such as angina will release Troponin at an elevated level into his or her blood. A blood test can be conducted to determine its presence. If it is present, it is a reliable indicator, of, depending upon the extent of its presence in the blood, a serious cardiac problem and specifically heart muscle damage. An ISTAT device can test for Troponin in five minutes, according to Dr Heggie.

11. On 23 February 2012 Mr Daniels was the subject of an autopsy. The Forensic Pathologist who conducted the autopsy, Dr Terrence Sinton, examined the body of Mr Daniels and determined that the cause of death was Coronary Artery Thrombosis. Dr Sinton stated that the morbid condition giving rise to this cause was Coronary Atherosclerosis. Heart disease remains the leading cause of death in Australia. Atherosclerosis refers, in plain terms, to the depositing of fatty material on the inside of the artery walls. This condition can build up over years although on some occasions it can build up in a much shorter time. It is impossible to say with precision how long Mr Daniels was suffering from atherosclerosis. What can be said with certainty is that unless it was treated and dealt with it was potentially deadly. This is because it can lead to a thrombus or blood clot inside an artery. This can lead to the complete blockage of an artery, which unless dealt with promptly, will usually be fatal. In Mr Daniels' case, Dr Sinton determined that Mr Daniels was suffering from a severe form of atheromatous coronary artery disease, which had recently resulted in blood clot formation (coronary thrombosis) that had led to the complete blockage of the left anterior (front) descending coronary artery. As this is a vital artery, its complete blockage, unless treated immediately or very rapidly through a combination of drugs and, in most cases, surgery will result in certain death.
12. Dr Herman described in concise terms the process by which death occurred, which I accept. This was as follows: death occurred due to cardiac arrhythmia (ventricular fibrillation<sup>17</sup>), which was secondary to acute myocardial ischaemia (lack of blood supply to the heart muscle) that occurred as a consequence of the blocked left anterior descending artery.
13. In terms of his risk of coronary artery disease Mr Daniels' risk factors can be summarised as follows:

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<sup>17</sup> Uncoordinated contraction of the ventricles of the heart.

- He was a cigarette smoker
- He did not exercise regularly
- He was obese
- He had high blood pressure
- He had proteinuria (protein in the blood).
- He was a male

14. Operating against the above risk factors, was the young age of Mr Daniels and the fact that when he presented at the clinic on the evening that he died, he did not complain of pains to his chest or to his arms, which are often experienced by those people who have or about to have, or are at risk of suffering from a major cardiac event such as a myocardial infarction (heart attack). He did however complain of “pain inside” but did not specify where it was located. As will be discussed in these findings, cardiac disease in one as young as Mr Daniels is not rare among young Aboriginal men in the Northern Territory (and Australia). Further, myocardial infarctions (heart attacks) can frequently occur without any prior symptoms.
15. It is important to state at this point what was not in issue in inquest, which was, firstly, that Mr Linklater had made a provisional diagnosis of dehydration; and, secondly, the nature of the medical treatment that was delivered to Mr Daniels by other medical personnel when they became involved in the matter from 8.30pm until Mr Daniels’ death.
16. Four matters constituted the key topics that this inquest largely focused upon. They were:
  - (i) What exactly was the treatment delivered by Mr Linklater (between 5.45pm and 8.30pm) and why?
  - (ii) If Mr Daniels had have been the subject of an accurate provisional diagnosis, was there a realistic way to save his life?

- (iii) What are the ways and means to reduce mistakes as to provisional diagnoses of this type being made in the future?
  - (iv) What are the ways that cardiac health can be improved for young Aboriginal men in general and persons in the position of Mr Daniels in particular?
17. Two eminent experts, with particular expertise in NT Aboriginal rural health, gave evidence in this inquest. Their evidence assisted me considerably. They were Dr Heggie and Dr Ilton. The latter was the Director of Cardiology at Royal Darwin Hospital and an NT cardiologist of great experience and the former the Chief Rural Medical Practitioner in the Department of Health and a doctor of great expertise in this area. In addition I was assisted by the report of Dr Herman, a cardiologist from Sydney who provided a report on this matter.
18. Mr Mark Thomas appeared at the Inquest as Counsel Assisting. Mr Alistair Wyvill SC appeared on day one of the inquest hearing on behalf of Sunrise Health Service Aboriginal Corporation. Mr Chris McDuff appeared for the remainder of the inquest on behalf of Sunrise. Mr Greg McDonald appeared for the NT Department of Health. Mr Jared Clow of the North Australian Aboriginal Justice Agency (NAAJA) appeared for the family of the deceased. Mr Ron Hope appeared for two nurses: Mr Nigel Linklater and Ms Sujin Le, both of whom, for medical reasons did not attend and deliver evidence at the inquest.
19. I received into evidence the investigation brief prepared by Senior Constable Jody Lovett, which comprised, in essence, the key materials in this matter. In addition, a bundle of other materials were tendered, which included the medical records (clinical/patient notes) of Mr Daniels (from Katherine Hospital and Sunrise Health Clinic at Ngukurr), the birth certificate of Mr Daniels, the autopsy statement of Dr Sinton; the expert medical reports of Dr Heggie, Dr Herman and Dr Ilton; medical statements explaining Mr

Linklater and Ms Le’s non-attendance at this inquest; a document dated 20 February 2012 described as a “Debrief” document; and, finally, the Central Australian Remote Practitioners Association (CARPA<sup>18</sup>) Standard Treatment Manual (STM)(fifth edition).

20. Senior Constable Jody Lovett gave evidence at the Inquest as did Dr Hugh Heggie, Ms Daphne Daniels, Ms Doreen Daniels, Ms Beatrice Roberts, Mr Owen Turner, Registered Nurse Joan Tibballs, Dr Terrence Sinton, Dr Marcus Ilton and Dr Tanya Davies. The Aboriginal members of the family of the deceased who were also witnesses in this case attended on the first day of the inquest and received an explanation of the death of Mr Daniels from Dr Heggie, who addressed them in plain terms and, utilised an anatomical dummy in so doing. I shall turn to this matter later in the findings. I should add at this point that I extend to the widow and family of the Mr Daniels my sympathy for their tragic loss.
21. Pursuant to section 34 of the *Coroner’s Act* (hereafter “the *Act*”), I am required to make the following findings:
  - (1) A Coroner investigating-
    - (a) A death shall, if possible, find-
      - (i) The identity of the deceased person;
      - (ii) The time and place of death;
      - (iii) The cause of death;
      - (iv) The particulars needed to register the death under the *Births, Deaths and Marriages Registration Act*.
22. Section 34 (2) of the *Act* operates to extend my function as follows:

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<sup>18</sup> The CARPA manual is the “bible” of remote area medical practitioners (doctors and nurses) in rural NT. It is regularly updated. The fifth edition has on the introductory page the date “2009”. I was informed, subsequent to the inquest, by those responsible for its publication, that the fifth edition was released in hard copy form in June 2010 and in electronic form in May 2011.

“A Coroner may comment on any matter, including public health or safety or the administration of justice, connected with the death or disaster being investigated.”

23. Furthermore, I may make recommendations pursuant to section 35 (1), (2) and (3):

- “(1) A Coroner may report to the Attorney-General on a death or disaster investigated by the Coroner.
- (2) A Coroner may make recommendations to the Attorney General on a matter, including public health or safety or the administration of justice connected with a death or disaster investigated by the Coroner.
- (3) A Coroner shall report to the Commissioner of Police and Director of Public Prosecutions appointed under the *Director of Public Prosecutions Act* if the Coroner believes that a crime may have been committed in connection with a death or disaster investigated by the Coroner.”

### **Reported When and by Whom**

24. Dr Tanya Davies reported the death to Police at Ngukurr at 9.45pm on 19 February 2012.

## **PART 2: RELEVANT CIRCUMSTANCES SURROUNDING THE DEATH**

### **Part 2.1: Background to Coronary Artery disease and its causes.**

25. It is very important that this disease and its causes be clearly understood. This is especially so as the evidence in this inquest clearly indicates that Aboriginal people in particular are vulnerable to this disease and are prone, in the Northern Territory at least, to experience the onset of this disease in their twenties or even earlier, which is much younger than the rest of the population. It is not extremely rare, as Dr Herman <sup>19</sup>stated, for a 24-year-old man to be suffering from atherosclerosis in the NT in the absence of a

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<sup>19</sup> Dr Herman, was an expert cardiologist from Sydney who was consulted by lawyers for Sunrise Health to provide a report on this matter.

profound family history of premature vascular disease<sup>20</sup>. Much is said in contemporary public discourse in Australia of “closing the gap” between Aboriginal and non-Aboriginal life expectancies. Coronary artery disease, as the principal killer of all Aboriginal people, and younger Aboriginal people in particular, lies at the heart of effectively dealing with the problem of increasing Aboriginal life expectancies. The matter is thus of singular importance.

26. I am grateful to the efforts of Dr Heggie, who with the aid of an anatomical dummy, provided a most helpful explanation to the family of Mr Daniels, who were present in court on the first day of the inquest. It was very important that this be done and done in terms that the family could understand. Dr Heggie can be very pleased with his efforts in this regard.
27. All three specialists who provided material for this inquest did not differ greatly as regard the key matters in this inquest. However on one key matter they did. It is important to note that Dr Herman did not give evidence but the other two experts did. As a consequence of the fact that the two NT specialists (Dr Ilton and Dr Heggie) have particular and lengthy expertise with regard to Aboriginal rural cardiac health in the NT, I have focused upon their evidence. This is not meant to undermine the importance of Dr Herman’s contribution but to recognise the strengths of the NT specialists, who were particularly familiar with the situation at the coalface of rural Aboriginal health in the NT and the relevant protocols concerning medical intervention. It is important to note that Dr Herman regarded the presence of cardiac disease in this case as extremely rare in a male of Mr Daniels’ age. The other two experts did not agree with this- in the case of Aboriginal persons.

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<sup>20</sup> The genetic history of Mr Daniels as regards coronary artery disease was not produced to this inquest.

28. Coronary artery disease, which refers to a disease of the heart's arteries, affects those arteries adversely by causing them to "clog up" with, in plain terms, fatty material which, firstly, reduces the flow of blood to and from the heart, but also, make a complete blockage of an artery leading to the heart much more likely. If such a blockage occurs, this may well be deadly, as the heart needs its arteries to be fully open in order for it to pump and circulate blood around the body properly. In plain terms, the heart is a pump, which needs the arteries (similar to pipes) leading into and from it to be functioning properly in order for it to do its job. If one or more of the important pipes is clogged up and then blocked with material known as plaque, which happened in this case, then death is likely unless the problem is very quickly identified and urgent action is taken to address the problem.
29. As to why this problem occurs so early in the life of a significant number of young Aboriginal people such as Mr Daniels, the eminent doctors who gave evidence in this inquest were unable to say. What they did say, though, is that a person's possession of cardiac disease risk factors make it much more likely that heart disease will develop in that person. Amongst these cardiac disease risk factors is, firstly, high blood pressure, which means that the blood pushes around the body at significantly higher levels than normal, which places the heart and its arteries under greater pressure and places the heart at greater risk of a blockage in one of its cardiac arteries.
30. A second risk factor is cigarette smoking, which is a major cause of heart disease. Cigarette smoking, amongst other things, introduces a particular problem that may have occurred in this case (according to Doctor Heggie), which is that an artery might have suffered a spasm or contraction, which will probably lead to reduced blood flow through it. In addition, cigarette smoking introduces a range of toxins into the body of a smoker, raises the body's blood pressure, increases significantly the prospects of the person developing a cancer, and damages in some way virtually every important organ of the smoker. It must be understood by Aboriginal people who smoke

that cigarette smoking of itself is dangerous to their health and that at least half of those people who smoke will die of diseases or illnesses directly related to smoking. Furthermore, it will likely lead to their death at a much younger age (compared with non-smokers in the same circumstances). This is a vital matter given the evidence of Dr Heggie, in this inquest, that smoking rates of Aboriginal rural folk are now in the order of 70%, which is a much greater rate than the rest of Australia, in respect of which there have been very significant declines, over the last couple of decades.

31. A third risk factor for cardiac disease is a poor diet, especially one full of saturated fat, which raises cholesterol levels as well as blood pressure and introduces fats into the arteries.
32. A fourth risk factor is a sedentary lifestyle, constituted by little or no exercise, which has now been clearly identified by the medical experts in this case (and around the world) as a significant factor that raises the risk of developing heart disease.
33. A fifth risk factor is obesity, which will increase the risk of developing high blood pressure and diabetes, as well as increasing the likelihood of developing atherosclerosis.
34. It is possible for a person to possess all of these cardiac disease risk factors and not, nevertheless, to develop heart disease. Such a person would be exceptional. The reality is that the possession of multiple risk factors such as those specified above significantly raises the risk of developing coronary artery disease, the development of which increases in likelihood as time goes by. For Mr Daniels, this regrettable reality applied to him, although he did not know it. He was, in hindsight, suffering from advanced coronary artery disease even though he was only 24 years of age.

## **Part 2.2: Background of Mr Daniels**

### *2.2.1: Personal history:*

35. Mr Daniels was born in Katherine and resided at Ngukurr all his life. He was the youngest of three brothers and went to school at Ngukurr and also at Urapunga outstation (25 kms from Ngukurr). His aunty Daphne Daniels raised him after his mother left the family when Mr Daniels was about 7 years of age. After finishing school Mr Daniels was employed in community development employment programmes in Ngukurr mostly in sports and recreation. He was active in the community, well-liked and participated in football as well as playing the guitar and writing music in a band. He did not attend weekly training sessions of football but played it on the weekend (during the season). Mr Daniels' family/genetic cardiac history was not produced to this inquest.

*2.2.2: Medical history:*

36. The medical records of Sunrise Health centre at Ngukurr disclose that he attended that clinic on 25 occasions in the five years prior to his death.
37. On 1 August 2007 he attended the clinic with a sore ankle due to a football incident. His heart rate was 90 beats per minute (bpm) and his blood pressure was 134 (systolic)/91(diastolic)<sup>21</sup>.
38. On 2 October 2007 he attended the clinic in relation to an infection. His blood was extracted in order for it to be tested.
39. On 21 November 2007 he attended the clinic in relation to a toothache. On this occasion and the previous attendance there is no record of what his blood pressure was.
40. On 25 February 2008 he attended the clinic in regard to a problem with a sore ankle that occurred after playing football. His heart rate was 92 bpm (beats per minute) and his blood pressure was 130/86.

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<sup>21</sup> "Systolic" refers to the phase of the heartbeat when the heart muscle contracts and pumps blood from the chambers of the heart into the arteries. This is the point when maximum pressure is exerted. Diastolic refers to the phase of the heartbeat when the heart muscle relaxes and allows the chambers of it to fill with blood.

41. On 19 March 2008 he attended the clinic in regard to a health check up. His medication was reported to be up to date. His exercise level was noted to be poor. His blood pressure was 110/70.
42. On 25 September 2008 he attended the clinic for another health check. On this occasion his blood pressure was recorded as 127/78 with a pulse rate of 65.
43. On 30 September 2008 he attended the clinic where the patient notes reveal that he was a heavy drinker and that advice was given to him that he must reduce his weight and alcohol use and that he was to be seen in three months. He was specifically advised to exercise for at least 30 minutes a day or at least five days a week. His heart rate was 68 beats per minute. His blood pressure was not recorded.
44. On 3 March 2009 he attended the clinic and said that he had chest pain two weeks ago. He said that he didn't know if family members had heart conditions. He said that he had "chest pain back of both shoulders area". He was not on medication. He said that when carrying a child he gets short winded and that he can't walk for long. He said that he got short of breath but that he was not an asthmatic. He said that he didn't know if family members have heart conditions. He said that he had pain in the back of both shoulders. He was not on medication. His blood pressure was recorded as 137/90 with a pulse rate of 83bpm. The assessment of him at that time is recorded as "astmatic (sic)/over weight- pressure cuasing (sic) shortness of breath". Under the subheading of "plan", it had been noted that he needs to see a doctor. He was prescribed a drug called Brufen 400 mg 1 tablet to be taken three times per day after food. Brufen is a trade name for Ibuprofen, a drug that is commonly prescribed for the relief of pain and inflammation. It is not a drug that is specifically targeted to deal with cardiac artery disease.

A specific note was made that “musculoskeletal shoulder pain was causing SOB”.<sup>22</sup>

45. On 21 July 2009 he had his blood extracted again for the purpose of tests to be conducted upon it. These were to pertain to LFT and Lipids (amongst other things).
46. In August and September 2009 the notes record that there were a number of appointments in which there was no client contact.
47. The next contact with the clinic appears to be on 2 October 2009 when Mr Daniels attended and reported pain in the right shoulder after some fighting the previous night. His blood pressure was recorded as 135/85 with a pulse rate of 112. It was noted that he had difficulty in raising his arm to shoulder level. It was noted that he had a soft tissue injury. There is no recording on this occasion of the results of his blood tests that had been conducted previously. He was prescribed Panadeine and Brufen.
48. On 3 November 2009 he was administered an immunisation (for swine flu).
49. Mr Daniels’ next appointment was on 12 January 2010 when he attended the clinic complaining of shoulder pain on both sides which had occurred for two weeks. A note was made by the treating nurse that he needs to be referred to a doctor or a specialist. His heart rate was 119 bpm and his blood pressure was 140/110. (It should be observed that any systolic rate of blood pressure of 140 or above is high and not within normal bounds). He was not prescribed any blood pressure medication or indeed any medication on this occasion. Nor is it clear from the notes when the appointment with the doctor was to occur.
50. Mr Daniels attended the clinic on 8 March 2010 again in relation to pain in his shoulder. A doctor named Dr Henwood saw him. It was specified that he

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<sup>22</sup> SOB is an abbreviation for shortness of breath.

experienced pain in his right shoulder when lifting and carrying at work and that this had been present for 6 months. Panadol and rubbing in medication did not help. On examination it was observed that he might have had a rotator cuff injury to the right shoulder. A specific note was made that because of his weight, blood pressure and proteinuria that he should be on ACE (meaning ACE inhibitor medications). It was also noted that he was very unfit and that he will need “ace to save kidneys”. He was referred to a GP again. He was prescribed Ibuprofen on this occasion and directed to have an X ray.

51. On 13 April 2010 Mr Daniels again attended the clinic. However, on this occasion it was in relation to a problem that he was experiencing with defecation. The diagnosis was stated to be unclear. His blood pressure was not recorded at this point.
52. On 29 April 2010 Mr Daniels attended the clinic in relation to a problem that occurred after he fell off a roof of a house whilst drunk. He had a very painful left wrist, a laceration under his left foot and a painful right heel. His heart rate was 110 bpm with a blood pressure of 135/81. He was prescribed Panadeine forte.
53. On 23 May 2010 Mr Daniels attended the clinics for the purpose of a review of his X ray results (regarding his shoulder) as well as discussing his kidneys. His heart rate on this occasion was 131 bpm, which was very high as was his blood pressure, which was 150/89. He again reported a sore shoulder, which he said that he hurt after throwing rocks at the roof of his girlfriend. There was no recorded discussion of the X ray results regarding his shoulder, or the blood test results. Nor is there any mention of dealing at this point with lowering his blood pressure. There was some discussion regarding police concerns that there was a male who had threatened suicide. Mr Daniels denied that he had any such intention.

54. On 24 May 2010 Mr Daniels again attended the clinic and had a musculo-skeletal examination of his shoulder. It was noted that it was tender and that it was an old injury. Little other detail was obtained on this occasion.
55. On 3 September 2010 Nurse Linklater attended the police cells at Ngukurr at 3.45am where Mr Daniels was after having been arrested by police. He noted that there was a need to deal with treatment of possible fracture/dislocation of the scaphoid.
56. On 6 October 2010 Nurse Sujin Lee treated Mr Daniels at the clinic. She noted that he presented with right shoulder pain, which was an old injury and that the client stated that it was getting worse. His heart rate was 99 bpm with a blood pressure of 144/100. She advised him to come back the next day to see a doctor for further investigation. She offered an analgesic. Regrettably, the following day, 7 October 2010, Mr Daniels did not see a doctor at the clinic.
57. The next documented attendance at the clinic was on 10 January 2011 when Mr Daniels attended in relation to a sore thumb. He was prescribed panadol for this.
58. On 21 January 2011 Mr Daniels attended the clinic in relation to a painful left shoulder. He was unable to lift his arm. His heart rate was 125 bpm with a blood pressure of 145/102. Strapping and a collar cuff were applied with an analgesic given to him in the form of panadeine forte.
59. On 24 January 2011 Mr Daniels again attended the clinic in relation to “subluxed his L shoulder fighting”. He was prescribed more Panadeine and also Brufen.
60. On 15 February 2011 Mr Daniels attended the clinic this time in relation to a headache, coughing and vomiting. He said that he was sore in the diaphragm and in the ribs. His heart rate was 128 beats per minute and his blood pressure was 142/93. A detailed assessment was made of him on this

occasion. Urinalysis revealed that he had 300 mg of protein/per dL in his urine. It was observed that he had a fever, sinusitis and a red throat. He was prescribed antibiotics in the form of Penicillin, Amoxicillin and Clavulnic. He was also given vitamin C tablets, Loratidine and Bisolvon.

61. On 18 April 2011 Mr Daniels again attended the clinic and was received a check up. It was noted that he presented with one year of intermittent pain to the left shoulder, wrist and left index finger and occasional right index finger pain. He thought that the pain could have commenced after he was involved in a fight last year but on probing of this it was noted that it could have preceded this fight. Mr Daniels said that the problems affected his ability to work and to assist in household chores. He was advised to return to the clinic if the symptoms did not improve in the next 24-48 hours. Regarding his musculo-skeletal system it was noted that he was to “See Dr re arm”. His blood pressure was recorded as 139/87 with a pulse rate of 104bpm. No advice was rendered regarding smoking, nutrition, alcohol, physical activity –perhaps because it was noted that Mr Daniels was in a hurry to leave.
62. On 22 July 2011 Mr Daniels presented in respect to a minor matter of a boil and on 26 November 2011 in regard to ear pain.
63. On 30 January 2012 Mr Daniels attended in relation to pain/swelling to his right foot, which had been injured playing football. He was treated for this with anti-inflammatory medication, local ice and advised to rest the leg.
64. A note appears in the patient notes for both 13 February 2012 and 17 February 2012 that he was to consult the clinic Doctor regarding chronic sore shoulders. A note on the records states that an attempt by the clinic was made to find Mr Daniels on 13 February 2012 without success. This was the last entry in the records prior to Mr Daniels’ death.

**Part 2.3: Analysis of the treatment of Mr Daniels between 5.45pm and 8.30pm on 19 February 2012**

65. Given that Mr Linklater did not give evidence in this inquest, the evidence of what he did or did not do at the critical time from between 5.45pm to 8.30pm is derived from evidence that emanated from five sources: firstly, the observations of Nurse Tibballs when she assisted Mr Linklater at about 6.15pm-6.30pm; secondly, the statement that Mr Linklater made to the police; thirdly, the patient notes that he typed up in this regard; fourthly, a document that described a “Debrief” meeting that was held on 20 February 2012, which was compiled by Mr Linklater<sup>23</sup>; and fifthly, the evidence of Mr Daniels’ relatives who attended that night, who were Daphne Daniels, Doreen Daniels and Beatrice Roberts.
66. Regarding the debriefing document, some attention was focused upon this in the inquest and in particular whether it was an entirely accurate and reliable document. Dr Davies and Nurse Tibballs, in particular, gave evidence that called into question the accuracy of significant parts of this document. It is unnecessary for the purpose of this inquest to enter into the intricacies of this debate. What is important is to analyse the following matters, most of which are referred to in the debriefing notes.

*2.3.1: Mr Daniels’ medical history and whether it was consulted by Mr Linklater between 5.30pm and 8.30pm*

67. There is no reference in Mr Linklater’s police statement, his patient notes, or debrief notes as to whether he looked at Mr Daniels’ medical history (which was readily accessible on the computer system at the clinic), which included persistent high blood pressure for a period of three years, a presentation of chest pain from an unspecified cause, presentations of

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<sup>23</sup> This document was tendered in this inquest having been disclosed to Counsel Assisting by Mr Linklater’s solicitor Mr Hope (who stated that the document was written by Mr Linklater).

shoulder pain (on both sides and separately), pains in the hands, persistent obesity, persistent absence of exercise, phases of shortness of breath, persistent cigarette smoking, and the presence of proteinuria. Clearly, a careful diagnostician would have looked at Mr Daniels' medical history at the time that Mr Linklater saw Mr Daniels in the clinic on 19 February 2012 to assist him in arriving at the correct provisional diagnosis. The absence of any reference in the relevant materials referred to above to consulting the medical history of Mr Daniels would tend to suggest that did not occur. Nevertheless, in fairness to Mr Linklater, in the absence of evidence from him, I cannot find that this in fact did not occur. It is, nevertheless, regrettable, that if the medical history was consulted, there was no note made of it in the aforementioned materials. I shall refer to this later in my Recommendations.

*2.3.2: The question of why a blood pressure reading was not obtained*

68. In regard to Mr Daniels' blood pressure, the debriefing notes stated that:

“BP unobtainable (? peripherally shut<sup>24</sup> down)(?Cuff size-n.b. large cuff could not be found due to a ‘tidy up’ when NL on leave) and adult large cuff missing from emergency room.”

69. It is important, firstly, to state that in order for a blood pressure reading to occur, a cuff, which is a bandage type object, is placed around the upper arm of the patient and then pumped up with air. The standard cuff is designed to accommodate most arm sizes. I accept that a very large upper arm diameter requires a large cuff to accommodate it. In this case, there is no evidence as to what was the circumference of Mr Daniels' upper arm. Mr Linklater did not describe its size.

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<sup>24</sup> “Peripherally shut down” refers to the phenomenon of the body, in an emergency situation, focusing the blood supply on the protection of key organs such as the brain and kidneys, rather than peripheral areas such as the skin. Consequently, the skin becomes cold due to reduced blood supply and blood pressure becomes difficult or impossible to obtain due to reduced blood flow.

70. It is important to observe on careful perusal of the Debrief document that the reference to BP being unobtainable is immediately followed by “? peripherally shut down”. This invites the inference that the blood pressure was the subject of an endeavour to obtain it, but was not in fact obtained because Mr Daniels’ body was peripherally shut down. It follows logically from this that a cuff large enough to accommodate the arm was employed for the purpose of effecting the process of endeavouring to obtain the blood pressure. The note in the Debrief notes that immediately follows this that refers to “n.b. large cuff could not be found due to a tidy up when NL on leave”, does not specifically state that a blood pressure reading could not be obtained because a large enough cuff could not be obtained. This note regarding large cuffs is thus not necessarily inconsistent with the aforementioned reference to “? peripherally shut down”.
71. The statement that Mr Linklater made to police contains no reference to the unavailability of large cuffs. It says, plainly, that he was “unable to obtain a blood pressure, due, I suspected to him having ‘shut down’ and having restricted blood supply peripherally, that is to his arms”.<sup>25</sup> The patient notes also contain no reference to the absence of large cuffs. They merely state that “BP unobtainable” and at 19.45 “Unable to obtain BP- still”. It is important to note that Mr Linklater was not able to insert a cannula into Mr Daniels’ arm. The patient notes say: “IVC sited at 18.30 after two failed attempts- ? peripherally shut down”. This suggests that Mr Linklater could not insert a cannula himself because he thought that Mr Daniels had peripherally shut down.
72. Nurse Tibballs says that she recalls that Mr Linklater said to her (between 6.15 to 6.30pm) that the blood pressure machine hadn’t worked. She did not say that he said to her why it had not worked. She said that she was not aware of a problem in obtaining large cuffs (which would be necessary to

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<sup>25</sup> Paragraph 3

obtain a reading for a large person –either with the machine or manually). She said that blood pressure could be obtained manually at the clinic.

73. In summary, Mr Linklater’s police statement and patient notes support a finding that Mr Daniels’ blood pressure was not obtained because his body “peripherally shut down”. Furthermore, if there was a problem with an absence of large cuffs, *which caused the blood pressure not to be obtained*, one would expect that to find its way into the patient notes and Mr Linklater’s police statement. It did not.
74. On the other hand, Mr Linklater’s statement to Ms Tibballs (that the blood pressure machine hadn’t worked) at least ostensibly provides some support for an argument that the blood pressure was not obtained because a large enough cuff was not available. The problem with this argument is that there is no evidence that this in fact was the case. Ms Tibballs certainly does not say that Mr Linklater said this to her, and the Debrief notes, as discussed, do not state this. Nor does any other written document on the subject. Indeed, as previously discussed, the reference in the debrief notes to “BP unobtainable (?peripherally shut down) supports a finding that a blood pressure reading was sought to be taken but could not occur because of the peripheral shut down. The failure of Mr Linklater to cannulate due to what he appeared to have thought was peripheral shut down lends further weight to this argument. In conclusion, the evidence supports a finding that a blood pressure reading was not taken because the peripheral system of Mr Daniels had shut down.

### 2.3.3: *The question of a pulse*

75. The debriefing notes contain a reference to a pulse being obtained and it being ‘strong’. Mr Linklater’s police statement contains no reference to obtaining a pulse. Nor do the Patient notes. However, Ms Tibballs did state in evidence that the patient did have a pulse when she put the drip in (at about 6.30pm). She could remember no further detail about the pulse. In

summary, the evidence would suggest that there was a pulse, although given the absence of any reference to the strength of the pulse in the patient notes, Mr Linklater's police statement and also Ms Tibballs' evidence, the evidence is not able to support a finding that it was a strong pulse.

*2.3.4: The physical state of Mr Daniels between 5.45p.m to 8.15pm*

76. The Debrief notes state on a number of occasions that the patient was in effect calm and in no distress prior to the cardiac arrest that occurred at 8.15pm. Specifically, the Debrief notes state that Mr Daniels: "remained awake at all times"; "patient calm and alert" (at the time of Ms Tibballs departure at shortly after 6.30pm); "patient conversant and in no distress throughout"; "patient said he felt good but got tired"(at or about the time of the football match); "patient remained calm and in no distress"; "increasingly alert", "NL advised by patient he was feeling much better and wanted to go home".
77. Ms Tibballs said that when she arrived and saw Mr Daniels at 6.15pm he looked sweaty and faint looking. When she left (at about 6.30pm), she said that he looked "quite sweaty still, pale and restless". Daphne Daniels, Mr Daniels' aunt, saw him on the evening in question. She said that she saw him under an alfoil sheet and that he was very shiny from being soaked with sweat and that his skin on his face and hands were swollen up.<sup>26</sup> Ms Daniels added that she held Mr Daniels' hand and he said "I'm alright, but I need some water".<sup>27</sup> In cross-examination, Ms Daniels agreed that Mr Daniels had said that he was ok and for her to get some party food ready for her.
78. Ms Beatrice Roberts, the wife of Mr Daniels, saw him initially in the clinic when there was no drip in his arm. His eyes were open but he initially didn't answer her. Then he did, it would appear, a short time later. She says that Mr Linklater said to her that Mr Daniels was near to death, he was so

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<sup>26</sup> Police statement of Ms Daphne Daniels, paragraph 13.

<sup>27</sup> Police statement of Ms Daphne Daniels, paragraph 14.

dehydrated, and that he might be home after he has the drip, maybe after midnight.<sup>28</sup> She was told to stay outside, as Mr Daniels needed rest. Later, she said that Mr Daniels told her to go home to get some party food ready for him for when he gets out of hospital. She came back later to the clinic and then saw Mr Daniels briefly with Mr Linklater near him, telling him to wake up. Shortly after this, she saw other medical personnel rush in.

79. Ms Doreen Daniels saw Mr Daniels on the bed in the clinic. Initially he had his eyes closed. She was told by Mr Linklater to go outside the clinic. This she did. Later she was told that Mr Daniels had passed away.

*2.3.5: When was the IV<sup>29</sup> tube placed in Mr Daniels?*

80. The Debrief notes refer to Nurse Tibballs cannulating<sup>30</sup> Mr Daniels' right wrist. The notes do not say when this occurred. Nor does Mr Linklater's police statement, which also does not state who put the cannula in. Nurse Tibballs was in fact the person that did this –at about 6.30pm, according to the patient notes, which state that an IVC<sup>31</sup> was sited at 6.30pm. She did it because Mr Linklater told her that he could not get a drip<sup>32</sup> into the patient.

*2.3.6: Could Mr Daniels be tested for Troponin between 5.45pm and 8.30pm?*

81. As previously stated, Troponin is an excellent means to determine if a patient is suffering from a cardiac arrest. A blood test is used to determine the presence of Troponin. The best means of quickly conducting a blood test for this purpose is by using an ISTAT device, which was then not available at the clinic. In the absence of an ISTAT device it was still potentially possible to test for Troponin using a test kit. However, Dr Tanya Davies, who was the GP who was on call at the clinic at the time stated in evidence that she did not know if a test for Troponin could be conducted. She said

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<sup>28</sup> Police statement of Ms Beatrice Roberts, paragraph 10

<sup>29</sup> Intravenous

<sup>30</sup> This refers to the insertion of a cannula into Mr Daniels's body.

<sup>31</sup> Intravenous drip line

<sup>32</sup> medical slang for an IV line, which was dependent upon the insertion of a cannula.

that the test kit would usually be kept in the fridge but did not know if in fact it was there at the time.

### **PART 3: FINDINGS**

#### **Part 3.1: Cause of Death**

82. As discussed previously, there is no issue in this case as to the cause of death, which has been specified in these findings, at paragraphs 11 and 12.

#### **Part 3.2: General Findings**

##### *3.2.1: Matters preceding Mr Daniels' arrival at the clinic on 19 February 2012*

83. I find that Mr Daniels smoked approximately three cigarettes at or just before the start of the football game that he played in, which, at the very least, did not assist his health. I note that Dr Heggie stated that the smoking of the cigarettes by Mr Daniels may have had two results: firstly, increasing the blood pressure, which would make the heart work harder; secondly, caused the arteries to get smaller, thus reducing blood flow. In effect, it is possible that it may have induced the coronary arrest that I find Mr Daniels suffered that occurred either during the game or immediately after it. It is not possible to find, however, that the cigarettes did in fact cause this. Nevertheless, this matter offers a severe warning, yet again, of the dangers of cigarette smoking, especially when combined with the presence of other cardiac disease risk factors.

##### *3.2.2: What probably was the cause of Mr Daniels' collapse at or about the time that he was playing football and how did this affect his presentation at the clinic?*

84. Dr Herman has provided a concise and useful explanation for what occurred in this regard, which I accept. It is as follows: on the day of his collapse, Mr Daniels probably developed an acute plaque rupture, which occurred on a pre-existing coronary atherosclerotic plaque in the left anterior descending

artery. The plaque rupture was then subsequently complicated by the development of an acute thrombus, which then occluded (blocked) the artery and resulted in a lack of blood supply to the heart muscle. Given the position of the blockage, a large amount of heart muscle would have been compromised which would then predispose Mr Daniels to ventricular arrhythmia and then cardiac arrest. Dr Hermann added that the plaque rupture probably occurred as a result of the physical stress (with increased blood pressure and pulse rate) in extreme heat in an otherwise fairly sedentary young male.

85. Dr Herman added that the discomfort and shortness of breath and subsequent dizziness experienced by Mr Daniels were probably all related to myocardial ischaemia (lack of blood supply to the anterior (front) of the heart muscle with poor cardiac output causing low blood pressure, inadequate cerebral perfusion (brain blood flow) and the other symptoms that he presented with at 5.45pm at the clinic. Again, I accept this explanation.

*3.2.3: The medical Care supplied by Mr Linklater between 5.45pm and 8.30pm*

*3.2.3.1 Summary of treatment rendered*

86. I find that, in relation to the critical time period between 5.45pm – 8.30pm, that Mr Linklater:
- a. Made a provisional diagnosis of dehydration, which he adhered to.
  - b. Did not consider an alternative diagnosis of a heart attack until the cardiac arrest occurred at 8.15pm
  - c. Said to Nurse Tibballs when she asked him (at between 6.15-6.30pm), that he would call the DMO.
  - d. Said to Nurse Tibballs when she asked him (at between 6.15-6.30pm), that he would conduct an ECG test.

- e. Was not able to obtain a recording of the blood pressure of Mr Daniels.
- f. Did not contact or attempt to contact the DMO or Dr Davies prior to the cardiac event at 8.15pm.
- g. Did not conduct an ECG test.
- h. Did not conduct a Troponin test.
- i. Did not arrange for a doctor to be contacted until 13 minutes after CPR commenced.
- j. Was not personally able to insert a cannula into the arm of Mr Daniels (the cannula was inserted into Mr Daniels' arm by Nurse Tibballs at about 6.30pm)

*3.2.3.2: Failure to call a doctor and conduct an ECG test*

87. I find that the medical treatment rendered by Nurse Linklater to Mr Daniels between 5.45pm to 8.30pm was inadequate and should have been significantly better. The evidence of Dr Ilton and Dr Heggie makes clear that in the absence of a blood pressure reading and with the persistence of the patient remaining profusely sweaty, combined with a presentation of shortness of breath and non-specific generalised pain, that an ECG reading should have been obtained and the DMO (District Medical Officer) promptly contacted by phone. Given Nurse Tibballs' evidence that Mr Linklater said to her at about 6.15pm-6.30pm that he would contact the DMO and conduct an ECG, these two matters were clearly on Mr Linklater's mind. Yet he did nothing in this regard. I accept Dr Ilton and Dr Heggie's evidence that the inability to obtain a blood pressure reading and the persistence of sweating, in combination, ought to have indicated to Mr Linklater that the problem was more serious than dehydration, and that a Doctor needed to be contacted in relation to the vital question of determining if the provisional diagnosis was accurate, and if not, what the correct differential diagnosis was. It is

relevant to note that there was no practical difficulty in contacting the DMO. This merely required a phone call.

88. Whilst Dr Ilton and Dr Heggie did not specify the precise time to do so, given the patient notes state that Mr Daniels was very tired and weak and exhausted from the commencement of his time at the clinic, and that he was experiencing extreme sweating as late as 7.15pm, I find that contacting the DMO should have occurred early on, preferably in the first half an hour (after his arrival at the clinic), given that for the entire length of this time period, there was a failure both to obtain a blood pressure and to cannulate, at a time when the patient was very tired and weak. Thus, at this critical time, when there was a provisional diagnosis operating of dehydration, this was not being attended to appropriately; and furthermore, a critical piece of diagnostic information (blood pressure) remained absent.
89. A further matter that I find occurred reinforces the argument that a doctor should have been called: this is Beatrice Roberts's evidence that Mr Linklater said to her that Mr Daniels was near to death, he was so dehydrated. In finding that Mr Linklater said this to her I note that it is not possible to say precisely when this occurred save to say that having said this remark, it clearly indicates that Mr Linklater was well aware of the extreme gravity of Mr Daniels' situation, (despite making the wrong provisional diagnosis), which ought to have emphasised the importance in his mind of calling a doctor in the circumstances as described.
90. An additional matter pertaining to this point is that Dr Herman stated that in regard to the provisional diagnosis of dehydration, (which he found could have been plausibly made), there was nevertheless, the "absence of response to effective fluid replacement therapy should have provoked alarm".
91. Regarding the ECG test, I accept Dr Ilton's evidence that an ECG test should have been conducted given the presentation of no blood pressure/or hypotension, shortness of breath, and unspecified pain. Dr Ilton emphasised

that an ECG test is a simple and easy tool to obtain a rapid diagnosis. I accept that it is. Further, Dr Ilton noted, and I accept this, that symptoms of heart attacks can sometimes be vague and that an ECG is in effect a quick way to obtain clarity regarding a diagnosis.

92. On the subject of the absence of an ECG test (prior to 8.15pm), Dr Herman commented that this was **the** most unfortunate occurrence, as a cardiac arrhythmia in the setting of acute myocardial ischemia is eminently reversible -with cardiac defibrillation being employed.
93. I find that a prudent, careful nurse, would have, in these circumstances, promptly conducted an ECG test and promptly called for assistance from a doctor-especially as there was a doctor in town, Dr Davies, (who worked for the same clinic) or by picking up the telephone and calling for urgent medical assistance, which included cardiology assistance that was instantly available. I note that Dr Heggie said that in his experience, many nurses, confronted with the scenario here, would have called for a doctor, having already obtained the ECG and Troponin results, which I find would be good practice.

*3.2.3.3: Mr Linklater's actions after the 8.15pm cardiac arrest*

94. A further matter is that once Mr Daniels suffered a cardiac arrest at 8.15pm Mr Linklater should have called for medical assistance immediately. Yet he waited for 13 minutes before attending to this. The CARPA manual and prudent nursing practice supports immediately calling for medical assistance, which, was as I have stated was close at hand.

*3.2.3.4: Mr Linklater's failure to insert a cannula and the consequences of this*

95. I have found that Mr Linklater was not, by himself, despite having a period of half an hour available to him, able to insert a cannula into Mr Daniels. It was Nurse Tibballs who did this after she arrived on the scene after being

requested by members of Mr Daniels family to check on Mr Daniels. She noted that Mr Linklater had not been able to insert the cannula and then attended to it. It is remarkable that in a case of what he thought was severe dehydration, with a man who Mr Linklater said was close to death from dehydration, that Mr Linklater had allowed 30 minutes to go by and had still not attended to what ought to be regarded as a fundamental matter in cases of dehydration: the insertion of a cannula, which would facilitate the introduction of an intravenous drip, which is a basic matter in dealing with a dehydration problem. Yet having failed over a period of half an hour to insert a cannula himself, Mr Linklater failed to ask for medical assistance in this regard. I repeat that Ms Tibballs arrived on the scene of her own volition. It is plain that the provision of cups of water to Mr Daniels (during the first three quarters of an hour) is not enough, according to the CARPA manual, to adequately attend to a dehydration problem. Any registered nurse ought to have known that, let alone an experienced registered nurse, as Mr Linklater was. In any event, this failure to again seek medical assistance, even for a diagnosis that was wrong, is consistent with Mr Linklater's broader failure, which I have referred to previously, to seek medical assistance promptly. I find that the fact that an IV drip was not inserted until 45 minutes after the presentation of Mr Daniels at the clinic represents a fundamental failure to attend to what Mr Linklater thought was the correct provisional diagnosis.

*3.2.3.5: The question of the appropriateness of Mr Linklater's provisional diagnosis of dehydration*

96. I find that the CARPA manual (volume 5), which was the manual that was applicable at the time of this death, probably did not assist Mr Linklater at that time. This is because, firstly, the manual did not contain a reference to the treatment of dehydration in adults. The section of the manual that dealt with dehydration was contained in a sub-chapter labelled "diarrhoea" that was contained within a chapter marked "Child Health". In an emergency

situation it may have been difficult for Mr Linklater to find this particular section as it was contained in child as opposed to adult health. Of course if he had have found it, it clearly refers to consulting a doctor in the case of persistent or severe dehydration.

97. I find that Nurse Linklater was also not assisted by the CARPA Manual (volume 5) as there was no specific sub-chapter in the Emergency Section of the Manual that dealt with the problem that arose here: determining the correct diagnosis of a patient who (unbeknown to the diagnostician) has in fact suffered a cardiac arrest (as Dr Heggie, Dr Herman and Dr Ilton said) and yet showed no symptoms of chest pain, but who was sweating profusely, and whose blood pressure could not be obtained. Instead, in the emergency section of the CARPA manual, it addresses the subject of heart attacks only under a sub-chapter entitled “Chest Pain”. This entirely misses the problem of heart attacks that do not show any symptoms of chest pain. This must be dealt with and I refer to this in my recommendations.
98. Dr Herman has stated that the provisional diagnosis of dehydration was not unreasonable in the circumstances as they presented to Mr Linklater- with no chest pain history specified, the playing of an active game in extreme heat, and myocardial ischaemia/myocardial infarction being extremely rare in a 24 year old.
99. I have already found that cardiac disease, which includes myocardial ischaemia/infarctions, is not extremely rare amongst Aboriginal young people in the NT. Therefore, this part of Dr Herman’s reasoning can be put to one side. However, in the absence of clear guidance from the CARPA manual (fifth edition) on this point, I find that in the circumstances it was not unreasonable why Mr Linklater might arrive **initially** at a provisional diagnosis of dehydration. I make this finding only on the assumption that Mr Daniels’ computerised medical history at the clinic was *not* consulted by Mr Linklater at the time. If it was consulted, as it should have been, the

persistence of high blood pressure, a previous chest pain presentation, persistent obesity and cigarette smoking, and previous shoulder pain problems extending on and off for some years, should have prompted an immediate provisional diagnosis of a cardiac problem-which would of itself necessitate an immediate call to the DMO and an ECG being conducted. I note also that Mr Linklater specifically treated Mr Daniels as far back at 3 September 2010, that is 17 months prior to his death, and that he might have had at least some recall of Mr Daniels. In any event, in the absence of any reference in the evidence supplied to this inquest by Mr Linklater, it would seem that it is possible that Mr Linklater did not look at the medical history at the time of the final consultation on 19 February. However, as I have noted, I cannot exclude the possibility that Mr Linklater, in the absence of evidence from him, did look at the computerised medical history of Mr Daniels' at the some point between 5.45pm and 8.15pm.

100. If Mr Linklater did not consult the computerised medical history, the provisional diagnosis of dehydration ought, nevertheless, to have been swiftly reviewed, when it became readily apparent that the blood pressure could not be obtained nor even a cannula inserted into Mr Daniels, as well at the persistence of a significant amount of sweating (despite the ingestion of water on a number of occasions). The combination of these factors, I find, ought to have made it clear to Mr Linklater that there was a proper medical basis for the view that the problem may well have been greater than dehydration alone. Dr Ilton and Dr Heggie, in effect, said this. I find, therefore, that a prudent and careful nurse in these circumstances would have, at the least, reasonably quickly sought assistance from a doctor-either by calling the DMO on the phone or calling Dr Davies.
101. In so finding, I note that it would have been helpful to a Nurse in the position of Mr Linklater if there had have been a reference in the CARPA manual in its Emergency section to the necessity of calling a doctor in cases

where the blood pressure cannot be obtained in circumstances as they presented here.

*3.2.3.6: The consequences of calling the DMO/conducting an ECG*

102. If the suggestions proposed by Dr Ilton and Dr Heggie of an ECG test and promptly phoning the DMO had occurred I accept the evidence of Dr Ilton that it is highly likely that the ECG would have picked up the blockage in the left anterior descending artery. Accordingly, it would have been quickly apparent to those treating Mr Daniels that he was suffering from a serious cardiac problem and was in imminent danger of death. Having accepted this consequence of the ECG it is not necessary to discuss in detail the other consequences of an ECG that were discussed in detail by Dr Heggie, which included picking up other more complicated aspects of heart disease.

*3.2.4: What was the likely cause of the collapse of Mr Daniels at 8.15pm?*

103. Dr Herman stated that the collapse that occurred at 8.15pm was probably as a result of a cardiac arrhythmia (ventricular fibrillation) and subsequent pulseless electrical activity that occurred in response to a large area of ischaemia (lack of blood supply to the heart muscle) subtended by the occluded (blocked) left anterior descending coronary artery. I agree with this and so find. I should add that ventricular fibrillation refers to the uncoordinated contraction of the cardiac muscle of the heart's ventricles, which makes them quiver, rather than contract properly. Ventricular fibrillation is a medical emergency that unless treated immediately will likely lead to an asystole<sup>33</sup> occurring. This in turn will lead to sudden cardiac death in a matter of minutes unless immediate medical attention is delivered.

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<sup>33</sup> This is colloquially known as a 'flatline' event, which is a state of no cardiac electrical activity.

*3.2.5: The question of the possibility of survival if a correct diagnosis had have been made*

104. If Mr Daniels' cardiac problem had have been diagnosed promptly, well prior to the 8.15pm cardiac arrest, I accept the evidence of Dr Heggie, Dr Herman and Dr Ilton that Mr Daniels might have survived, notwithstanding that he was in a position of extreme danger. I find that there was a possible avenue to survival which consisted of the near immediate ingestion of drugs designed to deal with the blockage in the left anterior descending artery (and the consequences of that blockage), as well as transporting Mr Daniels on an emergency aircraft to Darwin and thence to Royal Darwin Hospital. As Dr Herman stated, the management of Mr Daniels in this regard would have involved the administration of anti-platelet and anti-coagulant therapy. Of course, I must emphasise in so finding, that Mr Daniels may well not have survived and that the odds in favour of his survival were not great. Nevertheless, his cause was not hopeless and there was a realistic basis for his life to be saved, which was predicated upon the delivery of medical skill of a professional standard, luck and immediate attention.

*3.2.6: The risk of heart and coronary artery disease for young Aboriginal people*

105. I should add formally that I find that young Aboriginal people are particularly vulnerable to the development of coronary artery disease and atherosclerosis at a very young age (including in their late teens/early twenties). The precise scientific reasons for this, I find, are not clear.

*3.2.7: Why Mr Daniels' blood pressure was not obtained when at the clinic between 5.45pm and 8.15pm?*

106. As to why Mr Daniels' blood pressure was not obtained I refer to my observations in this regard at paragraph 68- 73 inclusive of these findings. In short, I find that the blood pressure was not obtained because Mr Daniels' peripheral system had shut down.

*3.2.8: The relevance of high blood pressure*

107. I accept the evidence of Dr Heggie that a systolic blood pressure rate over 140 is high and poses a serious cardiac risk to the health of any patient who presents with this blood pressure. I find that this risk increases the longer that high blood pressure persists without being treated.

*3.2.9: The pulse rate*

108. Regarding the question of a pulse rate, I find that a pulse was present, as Ms Tibballs has stated (at about 6.15-6.30pm), but it is not possible to determine the strength of the pulse, for the reasons that I have referred to in paragraph 74 of these findings.

*3.2.10: The physical state of Mr Daniels whilst in the clinic between 5.45pm and 8.15pm*

109. Regarding the physical state of Mr Daniels in the clinic prior to his suffering a cardiac arrest at 8.15pm, I find that Ms Tibballs is the most reliable witness in this regard and that her observation, when she left the clinic at shortly after 6.30pm, that Mr Daniels appeared “quite sweaty still, pale and restless”, can be accepted as an accurate description of Mr Daniels at that point. I so find. I note that there is evidence that suggests that Mr Daniels did at some point say to some relatives that he was ok and expressed a desire for food to be obtained for him when he returned home, but in accepting that he did say that, I do not find that this indicates that his health had in fact significantly improved or indeed, improved at all, whilst in the care of Mr Linklater. The evidence suggests that Mr Daniels’ health whilst in the care of Mr Linklater, remained parlous. The reality is that he suffered a cardiac arrest at 8.15pm, which killed him swiftly. It is obvious from this fact alone, which came on top of a previous cardiac arrest either on the football field or shortly thereafter, that he was in grave danger and in an extremely precarious situation whilst at the clinic.

110. The evidence from a number of Mr Daniels' relatives suggests, in summary, that they were very concerned for Mr Daniels when he was at the clinic, that they were told to go outside and let him rest, and that whilst he did say a positive remark at one point to them, this was in the context of his being in a delicate state.
111. I have noted the constant references in the Debrief notes to Mr Daniels stating, in effect, that he was much better, and wishing to go home. I note, however that the patient notes do not paint such a sanguine picture. For example, a reference in the patient notes at 19.00 hrs to Mr Daniels stating he felt much better, was immediately followed by an observation of "extreme sweating and skin cold to touch from 19.15". This was shortly after followed by a note that he appeared to be peripherally shutting down. Shortly after this it was noted that he was "very tired and drowsy". Then at 19.45 it was noted that he was "more awake and alert". At 19.45 it was noted that "Unable to obtain BP-still" and that "Remains tired and sleepy but conversant and oriented". The next note is at 20.15 when he suffered the cardiac arrest. I find that the Patient notes are more likely to be an accurate written record of Mr Daniels' state of health at the clinic as they are written not long after the event and are consistent with the observations of Nurse Tibballs and Mr Daniels' relatives who I find all told the truth to me. I find that the Patient notes suggest that Mr Daniels' health was delicate, and fluctuating, within an overall paradigm that suggested that his health was acutely vulnerable whilst at the clinic. The final point here is Ms Roberts' evidence that Mr Linklater said to her, when she was at the clinic, that Mr Daniels was near to death. In summary, I find that Mr Daniels' health was fragile in the extreme whilst he was at the clinic; I reject any notion that there was some sudden or general improvement in his health whilst at the clinic.
112. In contrast to this, the Debrief document, through the repetition of a theme of wellness, and an absence of reference to any of the parts of the

clinical/patient notes that suggested that Mr Daniels' health was poor, possess a self-serving flavour. I also note that the handwritten notes that Mr Linklater apparently made that evening went missing for an obscure reason. I also repeat that the accuracy of the Debrief document has been called into question by Nurse Tibballs as well as Dr Davies, both of whom were present at the debrief meeting. Taking all of these matters into account, I cannot accept what is contained in the Debrief document that pertains to this issue as constituting a reliable account of Mr Daniels' state of health in the clinic on the evening in question. It may be the case that a process of reconstruction has intruded into the compilation of this Debrief document by Mr Linklater.

*3.2.11: Whether Troponin could have been tested for when Mr Daniels was in the clinic*

113. Due to the absence of an ISTAT device at the clinic at the time the only way to test for Troponin would have been using a kit that was normally kept in the clinic's refrigerator. However, when Dr Davies, the resident GP at the clinic, was asked about this when giving evidence in this inquest, she did not know if the kit was there. In any event, there is no evidence to suggest that Mr Linklater considered this matter. I find that there is a real doubt as to whether Troponin could have been tested for when Mr Daniels was at the clinic on the night of his death.

*3.2.12: Summary of Medical treatment rendered by Mr Linklater between 5.45pm and 8.30pm on 19 February 2012*

114. Mr Linklater made the wrong provisional diagnosis, persisted with it, despite not inserting an IV drip for 45 minutes, and never obtaining a blood pressure reading in the face of a peripheral shut down. He did not consider an alternative diagnosis of a heart attack until, at the earliest, the time that the fatal heart attack that killed Mr Daniels occurred (8.15pm). Mr Linklater was not assisted by the CARPA manual but nevertheless, persisted down the

wrong diagnostic route despite, as Dr Herman put it, “an absence of response to fluid replacement therapy that ought to have provoked alarm”. In acknowledging the problems with the CARPA manual, to which detailed reference has been made, it can be understood why Mr Linklater initially made a provisional diagnosis of dehydration; however, his persistence with this diagnosis despite clear warning signs, which Dr Ilton and Dr Heggie said, in effect, should have indicated to him to do as they suggested, indicated a lack of professional judgement that was extremely regrettable. His fundamental failure to promptly call for a doctor, despite saying to Nurse Tibballs that he would do so, and moreover, saying to Mr Daniels’s wife that Mr Daniels was close to death, was, in the context of the persistence of sweating, the continued failure to obtain a blood pressure reading due to a peripheral shut down, in the circumstances, both regrettable and remarkable. His failure to conduct an ECG test, despite saying to Nurse Tibballs when she asked him that he would do so, is also, in all the circumstances both regrettable and remarkable. The extent of Mr Linklater’s failings are accentuated by the ease with which the two key matters that have been emphasised by Dr Ilton and Dr Heggie of contacting by phone a medical expert and conducting an ECG test were instantly available to be achieved. In short, at the critical time in his life when Mr Daniels needed expert medical attention he did not receive it. I find that the medical attention that he received from Mr Linklater between 5.45pm and 8.15pm fell significantly short of expert medical care. In so finding I have considered the question of whether the standard of the care that Mr Linklater delivered was at a level that warranted Mr Linklater being referred to the DPP in regard to whether a criminal charge of negligent manslaughter could be laid against him. I have carefully considered this matter. On reflection, I do not think that the conduct of Mr Linklater is sufficiently negligent to render it criminal. Nevertheless, from the perspective of Mr Daniels’ grieving relatives I can well understand why they might remain aggrieved by the conduct of Mr Linklater.

115. I do not know if Mr Linklater is considering returning to nursing in the NT. If he is, I recommend that the findings that I have made in this file regarding Mr Linklater, be referred to the NT Nurses Registration Board.

*3.2.13: The medical care delivered to Mr Daniels by the Sunrise clinic in the five years prior to his death*

116. I find that Mr Daniels did receive regular medical attention from Sunrise's Clinic at Ngukurr in the five years prior to his death. I find that it should have been apparent to those treating him, especially in the three years immediately prior to his death, that Mr Daniels had a number of serious problems that concerned, amongst other things, high blood pressure and proteinuria. As Dr Heggie said, proteinuria is an indicator that the kidney may have been damaged before and that its existence, of itself, increases cardiac risk. The persistence of these two serious health issues, together with the fact that he remained persistently obese, did not exercise, had been noted to experience chest pains and shoulder pains, and was a smoker, ought to have suggested to those treating him that Mr Daniels was at serious risk of developing cardiac disease. Mr Daniels needed to be subjected to a thorough analysis to determine if he had cardiovascular disease. It never happened; In this regard, there was thus a failure to some extent to properly diagnose and treat Mr Daniels. I find that the persistent high blood pressure readings in the final three years of Mr Daniels' life were (in the context of the other problems that I have referred to) clearly consistent with the possibility that Mr Daniels may in fact have been in the early stages of developing coronary artery disease in the three or so years prior to his death. At the very minimum the protocol referred to in the CARPA manual<sup>34</sup> needed to be activated. It is not complex. As has been said repeatedly in

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<sup>34</sup> As previously noted the Fifth edition came into operation in hard copy form in June 2010 and in electronic form in May 2011. The fourth edition was operating prior to these dates. The primary relevant difference between the two editions was that the cardiovascular risk chart on page 230 and 231 of the fifth edition was not present in the fourth edition. Nevertheless, the chronic coronary artery disease assessment and

these findings an ECG test (stress test or otherwise) and a Troponin test are easy and effective ways to do this.

117. It is not possible to say with certainty when Mr Daniels began to develop atherosclerosis but it is probable that at some point in that three year period prior to his death that he developed it. The 3 March 2009 consultation in which chest pains were raised by Mr Daniels for the first time ought to have sparked, of itself, a thorough analysis of Mr Daniels to determine if he had cardiac artery disease at that time. It never happened. Dr Sinton had said that Mr Daniels was suffering from a severe form of atheromatous coronary artery disease. This is consistent with the disease not simply developing overnight.
118. It may be that those treating Mr Daniels did not think that he was at risk of coronary artery disease given his young age and the absence of persistent complaints of chest pains- and hence did not consider coronary heart disease as a serious risk or potential diagnosis.
119. This inquest should permanently drive home to all medical personnel treating Aboriginal people in the NT that the risk of coronary disease is a live consideration, in the circumstances as they presented here, notwithstanding the youth and absence of persistent complaints of chest pains of the patient.
120. The presence of a Troponin test kit in the absence of an ISTAT device is vital. Sunrise should strive to ensure that there is no repeat of an issue that arose in the inquest: that the doctor in charge was uncertain if the troponin test kit was in fact in the refrigerator at the time of this emergency.
121. I find that in any event the Sunrise medical personnel treating Mr Daniels, should have also, in addition to conducting fundamental tests to determine if

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diagnostic analysis, which is specified at page 192- 195 of the fourth edition remained essentially the same as the fifth edition's CAD (cardiac artery disease) assessment and diagnostic analysis.

Mr Daniels' was suffering from coronary artery disease, as Dr Ilton and Dr Heggie stated, have promptly organised a plan to deal aggressively with the cardiac risk factors, when it became abundantly clear from multiple visits that Mr Daniels made to the clinic, that he had persistent high blood pressure, and the other problems to which I have referred. That plan necessarily involved putting Mr Daniels on blood pressure medication, cholesterol lowering medication and endeavouring to deal with raising his exercise levels and ceasing smoking. I accept the evidence of Dr Heggie, who said one of the key risk factors, high blood pressure, can be easily treated medically, which is an excellent and fundamental way to actively reduce the risk of dying from coronary artery disease.

122. I find that if a cardiovascular risk assessment as part of a comprehensive CAD (cardiac artery disease) differential diagnostic analysis had been implemented that it may have revealed the existence of some form of cardiac artery disease, that, if it existed, would probably have required, as Dr Ilton stated, at the very least, drug therapy to occur and the other strategies specified in the CARPA manual<sup>35</sup> to be initiated.
123. I further find that if drug and other specified therapy, as part of a comprehensive treatment plan, was delivered to Mr Daniels, subsequent to the 3 March of 2009 visit, that aimed to deal with the cardiac risk factors that had been identified, that, if it had been strictly adhered to by Mr Daniels and properly monitored, it would probably have assisted Mr Daniels by reducing his chances of dying on 19 February 2012 from the consequences of cardiac artery disease. I further find that a comprehensive treatment plan, if in place sooner rather than later, probably would have, if strictly adhered to, assisted Mr Daniels by reducing his chances of dying at a young age of the consequences of that disease.

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<sup>35</sup> Both the fourth and fifth editions refer to these matters.

124. In making these findings, I do not intend to discourage Sunrise in its efforts. I further acknowledge that the business of providing medical care is a difficult and unrelenting one. However, it is vital that Sunrise understand that its core business demands continual overview of its medical practices. Only by acting in this fashion can effective medical care be continually rendered- especially in this critical area. The general tenor of the medical care that has been demonstrated to have been rendered, as least to Mr Daniels, for the three years or so years immediately prior to his death frequently conveys the flavour of an operational paradigm that was predicated upon an absence of proactive treatment. I so find. There was merely the occasional reference, such as on 30 Sept 2008, to a suggestion being made by medical personnel to Mr Daniels to reducing his weight and alcohol use; however, no structured plan was rendered in this regard. It is in this area in particular that Sunrise must devote greater energy. Both Dr Ilton and Dr Heggie emphasised the importance of this aspect of the matter. I appreciate that a patient such as Mr Daniels must accept personal responsibility to assist himself; but he can only do so in the context of being properly advised and guided by his medical advisors as to what exactly he should do as part of a structured plan. This was all predicated upon the implementation of fundamental CAD diagnostic tests so that it could be clearly revealed to Mr Daniels and those treating him as to what exactly was wrong and therefore how exactly it could be dealt with. It is not complicated and the diagnostic tools in this area have greatly improved. Simply put, proper testing, advice and guidance was required. Because these fundamental matters were not attended to Mr Daniels was left in the dark as to the exact state of his health and what should or should not be done to attend to it appropriately.

#### *3.2.14: Medical advances in dealing with cardiac disease*

125. I must also add formally that I find that, as Dr Ilton, Dr Herman and Dr Heggie have said, cardiac artery disease can, with proper medical

management, be dealt with, its damage minimised and can be controlled so that patients suffering from it can live a lot longer than they otherwise would. With proper management it is not necessarily fatal at all. There is a great mass of medical evidence from around the world that demonstrates this. Indeed, in the last twenty years major gains in life expectancies have occurred in regard to cardiac patients. I formally accept these matters. Furthermore, the chances of sudden death or death at a young age, which was the fate of Mr Daniels, can be reduced considerably if proper care and attention is applied. All specialist doctors referred to the use of drugs such as statins, which are designed to stop or reduce the production of cholesterol in the liver as well as anti-inflammatory drugs. I find that it, in summary, is patently clear that a comprehensive treatment plan can be very effective with dealing with and controlling the effects of cardiac artery disease.

### *3.2.15: Cardiac technology*

126. In terms of technology, I accept the evidence of Dr Ilton and Dr Heggie that an ISTAT device (which is designed to rapidly deliver a blood analysis) and an ECG device that is able to transmit results digitally to on call cardiologists for immediate review and clinical support, are, in combination, the preferred means to rapidly determine (diagnostically) if a cardiac problem exists. I accept that the presence of Troponin in the blood is a reliable indication of the existence of a significant cardiac problem, and in that regard, the ISTAT device, in rapidly analysing the blood, is a most useful tool. I should add that the ISTAT device was not available at the time of the death of Mr Daniels. It follows from the proposed use of both an ISTAT device and an ECG, that there is no need to explore the installation of devices such as 3D or 4D echocardiography in remote areas in the NT, given that the provision of the latest ECG devices in combination with ISTAT devices would provide a very effective means by which a diagnosis of a cardiac problem can be obtained. There was also some discussion by the medical experts of ECMs (external cardiac monitors) in this inquest.

However, the point remains clear that the combined use of an up to date ECG device as described, as well as an ISTAT device, means that there is not a practical need for an ECM device.

#### **PART 4: FORMAL FINDINGS**

127. Pursuant to section 34 of the *Act*, I find, as a result of evidence adduced at the public inquest, as follows:

- (i) The identity of the deceased in this case was Dion Daniels born on 2 June 1987 at Katherine Hospital, Katherine, Northern Territory. Mr Daniels last resided at Lot 237, Ngukurr, Northern Territory.
- (ii) The time and place of death was 9.15pm on 19 February 2012.
- (iii) The cause of death was Coronary Artery Thrombosis with the morbid condition leading to that death being Coronary Atherosclerosis.
- (iv) Particulars required to register the death:
  - (1) The deceased was Dion Daniels
  - (2) Dion Daniels was an adult Aboriginal male
  - (3) The cause of death, being Coronary Artery Thrombosis, was reported to the Coroner
  - (4) The cause of death was Coronary Artery Thrombosis.
  - (5) Dion Daniels' parents were Margaret Mary Conway (mother) and Davis Ulubulari Daniels (father).

#### **PART 5: RECOMMENDATIONS**

128. That consideration be given by those charged with editing the CARPA manual to it being amended so that there is a specific sub-chapter, preferably in the Emergency section, that deals with the problem of the diagnosis of heart attack victims who are not displaying chest pains. This is for the specific purpose of assisting medical personnel placed in this position of making the correct provisional differential diagnosis. This

should also address how a provisional diagnosis of cardiac disease may be confused with a diagnosis of dehydration.

129. That consideration be given by those charged with editing the CARPA manual to it being amended so that there is a specific chapter that deals with dehydration that is both in a separate chapter and which deals specifically with adults.
130. That consideration be given by those charged with editing the CARPA manual to it being amended so that there is a specific chapter in the Emergency section specifying that, in an emergency presentation, an ECG test should be conducted in all cases in which blood pressure is unable to be obtained or hypotension (low blood pressure) exists. In addition, that consideration ought to be given for the manual to be amended so as to require, that in the circumstance of a nurse being placed in the position of having to make a provisional diagnosis in circumstances such as Mr Daniels' case, that the nurse be required to phone the DMO for medical assistance.
131. I acknowledge fully the efforts of the Department of Health to promote its chronic disease strategy comprising Adult Health Checks and Cardiovascular Risk Assessments. I recommend that by way of supplementing these efforts, that substantial further education be encouraged, both of medical personnel and of Aboriginal people especially in remote areas, of the reality of young Aboriginal people developing cardiac disease at a very early age. This point must be driven home to all medical personnel in the NT to assist them in making the correct diagnosis of cardiac disease, particular in the circumstances as they presented in the case of Mr Daniels. It must also be emphasised to all medical personnel and Aboriginal people in the NT, that heart attacks can occur in the absence of chest pains.
132. Having previously acknowledged the efforts of the Department of Health, I recommend that a coordinated strategy be embarked upon by the Department

of Health for the purpose of screening of heart disease in young Aboriginal people, especially in remote areas of the NT. I recommend that a proactive approach be taken in this regard and that it extend to the NGO sector and, in particular, organisations such as Sunrise Health.

133. I recommend that the Department of Health engage in a coordinated strategy to educate medical practitioners and nurses in this field to engage in proactive testing and screening for cardiac disease.
134. I recommend that Sunrise Health Service conduct a review of its own practices and staff with particular efforts being made to encourage training of staff to be proactive in screening for cardiac disease, and conducting the appropriate tests and treatment plans pursuant to the protocol specified in the CARPA manual. I encourage Sunrise Health Service to liaise with the Department of Health in this regard and also in regard to the education of Sunrise's clientele especially in regard to cardiac risk identification and avoidance. I emphasise that Sunrise advertise the point in its clinics that unless dealt with, heart disease will kill, and will strike down many young people unless steps are taken to deal with it. I would further emphasise that Sunrise promote the point that I have emphasised in these findings that Coronary Heart disease can strike without warning and without chest pains. Specifically, the importance of regular wellness checks must be emphasised by Sunrise and impressed upon its clientele.
135. The risk of smoking cigarettes and the very high rate of smoking amongst Aboriginal communities in the NT must be part of a public health campaign targeted at Aboriginal people that emphasises the grave danger to the health of all smokers and the significant risk of early death. Such a public education campaign could include a reference to the fact that the smoking of a small quantity of cigarettes prior to a football match, as occurred in this case, possibly can produce a spasm in an artery and contribute directly to causing a heart attack, leading directly to death.

136. Regarding the installation of ISTAT devices, I understand that they have been distributed to major health clinics as well as centres with populations in excess of 1,000 or so throughout the NT. I recommend, given their effectiveness, that they be installed in all medical clinics in the NT. The ability to have a quick and reliable analysis of blood cannot be underestimated. Together with an up to date ECG devices, it means that much more expensive technology is not necessary in order to promptly ascertain a diagnosis of a cardiac issue of a patient living in a remote area.
137. If an ISTAT device is not available at a clinic that clinic must ensure that it has a Troponin test kit that is available and functioning for the purpose of testing for this important enzyme.
138. I endorse the installation of the most up to date ECG devices (that are able to transmit results digitally to an on call cardiologist for immediate review) to all medical facilities in the NT. I understand from Dr Heggie that these devices are being delivered throughout the entire NT at this point and that it is expected that this will be completed in the next month or so. This is to be commended.
139. I recommend that the education of nurses emphasise that a nurse not make a provisional diagnosis of a medical problem unless there is clear support for it in the CARPA manual. Further, in terms of making a provisional diagnosis, if in any doubt, a medical practitioner must be consulted immediately. Finally, in terms of making a provisional diagnosis it ought to be emphasised that a nurse (or doctor) must consult the patient history and furthermore, make a note of what was discovered in that regard in the clinical or patient notes.
140. I recommend that the Central Australian Rural Practitioners Association consider the findings of this Inquest when preparing the next edition of the CARPA manual.

Dated this 3<sup>rd</sup> day of November 2014

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GREG CAVANAGH  
TERRITORY CORONER