

Medical Officer, Dr B
A Rural Hospital

A Report by the
Health and Disability Commissioner

(Case 04HDC00656)



Health and Disability Commissioner
Te Toihau Hauora, Hauātanga

Parties involved

Mr A	Consumer (deceased)
Mrs A	Complainant/Consumer's wife
Dr B	Provider/Medical Officer
A rural hospital	Provider/Hospital
Dr C	General Practitioner
Ms D	Practice Nurse
Dr E	Medical Officer
Dr F	General Practitioner
Dr G	Locum Medical officer
Ms H	Registered nurse
Ms I	Registered nurse
Mr J	Chief Executive Officer
Dr K	Medical Officer
Dr L	Emergency medical specialist
A first public hospital	Hospital
A second public hospital	Hospital

Complaint

The Commissioner received a complaint from Mrs A about the services provided to Mr A by medical officer Dr B, an employee of a rural hospital. The following issues were identified for investigation:

- *The adequacy of the diagnosis and treatment Dr B provided to Mr A, following his admission to a rural hospital on Day 1 with chest pain. In particular, whether Dr B:*
 - *adequately diagnosed and treated Mr A*
 - *appropriately discharged Mr A*
 - *adequately documented the care he provided to Mr A.*
- *The timeliness and adequacy of Dr B's response on Day 2 to Mr A's abnormal blood test results.*

An investigation was commenced.

Information reviewed

- Letter of complaint from Mrs A dated 15 January 2004
- Responses from Dr B dated 8 June 2004, 14 June 2005 and 15 January 2006
- Responses from the rural hospital, dated 31 March, 11 June 2004, 25 November 2005 and 31 January 2006, including Mr A's medical records
- Letters from Dr C dated 29 March and 2 June 2004
- Letter from Ms D dated 19 May 2004
- Letter from a medical centre dated 14 March 2004
- Information from ACC Medical Misadventure Unit
- Letter from Dr E, dated 19 June 2005

Independent expert advice was obtained from Dr Kingsley Logan, physician, and Dr Tim O'Meehan, cardiologist.

Information gathered during investigation

Overview

On Day 1 Mr A was admitted to a rural hospital for overnight observation, following an episode of severe chest pain. He was discharged the following morning with a diagnosis of gastro-oesophageal reflux disease (GORD) and returned to his farm to work. Tragically, Mr A died a short time later after suffering from further chest pain.

Chest-pain episode

Mr A was 54 years old, a smoker, and had no relevant past medical history. On Day 1, at approximately midday, Mr A suffered a 30-minute episode of severe central chest pain, with numbness down both arms, and developed a grey and sweaty complexion. Mrs A contacted the rural hospital (having first been unsuccessful at contacting the medical centre) and was advised to bring Mr A to the hospital by ambulance. Dr F, at the medical centre, was unsure why the on-call medical practitioner (who has since left the country) was unable to be contacted. Dr F stated:

“[Mr A] was acutely unwell with chest pain that could have been of cardiac origin (category 2 or 3). My phone advice would have been the same as [Mrs A] was given by [the rural hospital] staff. That is to call 111 to get an ambulance urgently. The ambulances are equipped with oxygen and a defibrillator. As [Mr A's home] is 35km from [the town] it was most appropriate to transport him to hospital via an ambulance as soon as possible.”

Mrs A rang 111 for an ambulance. She recalled:

“My husband was taken to [the rural hospital] by ambulance and examined, he was asked how much pain he had out of ten and indicated that it was an eight. [The ambulance record stated 8.5.] He had blood taken, was X-rayed and given an ECG. The pain had subsided but they decided to keep him in overnight for observation and monitoring.”

The ambulance staff noted that Mr A had a pallid colour with severe heart pain, which eased when he was sitting upright and was given oxygen. Mr A was provided with a soluble aspirin and urgently transferred to the rural hospital. During the transfer, Mr A was monitored with an ECG (electrocardiogram).

Admission to the rural hospital

Mr A arrived at the rural hospital emergency department (ED) at 1.45pm and was assessed by a locum medical officer, Dr G, in accordance with the rural hospital chest pain protocol. Mr A’s pain had eased and was recorded on arrival as 1/10. Mr A did not have any shortness of breath. Mr A had a number of investigations, including a chest X-ray, ECG and a blood sample taken for cardiac enzyme testing.

A qualitative troponin T “spot” blood test to evaluate whether Mr A had any evidence of cardiac damage was reported as negative at approximately 2pm. These tests are taken by hospital staff and are available 15 minutes after applying a blood sample to a rapid test kit. Mr A’s creatine kinase (CK) enzymes were not elevated.¹ Mr A’s ECG was recorded as normal. The chest X-ray was later reported as clear (although possibly indicative of a hiatus hernia). Mr A was admitted overnight for observation and monitoring with a working diagnosis of a hiatus hernia or gastro-oesophageal reflux disease (GORD). A further ECG was done at 6.08pm.

Ms H was the registered nurse responsible for the night shift on Day 1. Ms H reported that Mr A slept soundly through the night, with no further report of chest pain and normal observations at 6am.

A registered nurse, Ms I, was assigned the care of Mr A on the morning of Day 2. Mr A informed her that he felt well, had no discomfort, and was extremely keen to go home as soon as possible. Ms I recalled:

“[Mr A] was of natural colour and was able to move around without any effort. His breathing was normal.”

¹Troponin levels are used in patients who have chest pain to see if they have had an acute myocardial infarct (heart attack) or other heart damage. With a heart attack, heart muscle cells die and release their contents, including troponin and CK. Generally, troponin T and troponin I results both indicate a similar amount of heart damage; different laboratories typically will use only one or the other test.

Review and discharge by Dr B

Dr B, medical officer, reviewed Mr A at 8.50am on Day 2. He recorded that Mr A had had no further chest pain, felt well and his pain was “mostly epigastric and lower chest”; the probable diagnosis was GORD. Dr B requested a further ECG and cardiac enzyme testing with a view to discharge if the results were normal. (A chest X-ray was also requested to exclude other causes of chest pain.)

The ECG was performed by a technician at about 9.34am. The technician reported that Mr A was insistent on leaving, but she advised him to wait until Dr B had seen him.

Blood samples were taken at approximately 9.20am for further “spot” qualitative (troponin T) and quantitative (troponin I) cardiac enzyme testing. The spot test was returned negative at approximately 10am. The troponin I test was couriered to the city for testing and reporting by a medical laboratory.

Ms I took the results of the repeat ECG test, negative troponin T “spot” blood test and chest X-ray to Dr B for review. (The chest X-ray was reported as normal.) Dr B considered that the ECG finding was equivocal. He stated:

“As [Mr A] had no on-going chest pain, I felt it was reasonable to discharge him before the troponin I result came back. This was our normal practice up until this event.”

Dr B considered that Mr A met the usual criteria for discharge. The rural hospital chest pain protocol (designed by Dr B, apparently for Emergency Department rather than inpatient use, and introduced on 19 September 2003) requirements for discharge were as follows:

“Normal ECG
Normal Cardiac Enzymes
No dyspnoea
Not diabetic
No change in pattern of chest pain if history of previous pain
Chest pain atypical of ischaemia
No more than two risk factors for IHD [ischaemic heart disease].”

Dr B saw Mr A briefly prior to his discharge to inform him that his blood tests were normal, and to advise him that he could go home. Dr B recalled:

“His cardiac enzymes the previous night and that morning were normal and his ECG was essentially normal, having only an inverted T-wave in standard lead three and lead aVF [providing a voltage reading from the limbs], which is an equivocal finding. As he had a history of previous dyspepsia, a single episode of chest pain and no abnormal results, he was discharged with no new medication. He was strongly advised to give up smoking immediately.

...

[Mr A] was told that I thought he probably had GORD. I stressed to him that he should give up smoking. I also stressed that any further chest pain should be investigated and that he should see his GP for this unless he was acutely unwell.

...

As [Mr A] was in a hurry to leave he did not wait for his discharge summary. This was done later that day and posted to his GP, with a copy posted to [Mr A's] address. As it was done before the abnormal troponin I result came back, no mention of that result was made."

Dr B's discharge report dated Day 2 (addressed to Mr A's general practitioner, Dr C) states:

"Presenting complaint

Chest pain? GORD

Progress

[Mr A] presented to the emergency department by ambulance with a main complaint of chest pain of two hours duration. The pain was in his lower chest and epigastric and he had subjective paraesthesia of both arms. His pain had completely settled on arrival. He has no history of previous ischemic heart disease, but he smokes two packets of tobacco a week.

[Mr A] was admitted overnight for observation. Serial ECGs and cardiac enzymes remained normal. He had no further chest pain and was discharged the following morning. Should he have similar bouts in the future, a trial of losec may be helpful.

Medicine on discharge

Nil."

The rural hospital's "Medical Manual" states that patients may only be discharged upon the written order of a medical officer, and that a discharge summary is to be provided. Dr B made no entry in the medical records about Mr A's discharge or any instructions given. He explained:

"We normally allow a 'discharge if [further results are normal]' to be sufficient authorisation for discharge. The nurse in charge of a patient will always check verbally with the doctor that the pending results have been received and that the discharge has been approved. A further note would only be made if a result was abnormal and required some form of follow-up."

Mr J, Chief Executive Officer of the rural hospital stated:

“[Mr A] was advised to wait and see the doctor prior to leaving on the morning of [Day 2]. He did not wait and as such received minimal instructions on discharge other than the knowledge of his condition at the time (GORD/Hiatus Hernia) with a recommendation that he stop smoking immediately.

Had [Mr A] waited to see the doctor prior to leaving the hospital he would have been given the proper documentation and explanation that one test was outstanding. I’m also sure that the information would have been given to him that the test was to rule out any cardiac involvement with his chest pain. He would have been advised to continue with minimal activities until the test results were known.”

Mr J reported that nurse Ms I provided the following account of what occurred:

“Upon completion of the testing, [Ms I] indicated that she brought the test results to [Dr B] for review which he did and he indicated that [Mr A] could be discharged.

[Ms I] indicated that [Mr A] was very eager to leave and was standing in the doorway of his room awaiting the test results. When told that [Dr B] had approved his discharge, [Mr A] and his wife left without speaking to the doctor.

[Ms I] indicated that she did not complete the discharge checklist and get the patient to sign it and doesn’t recall giving the patient any further advice believing the doctor would do this prior to [Mr A] leaving.”

The rural hospital’s nursing protocols require nursing staff to complete a patient discharge checklist, which includes ensuring that a patient discharge summary has been written and provided.

Mrs A confirmed that no information was provided by staff about the outstanding troponin I test result. Mrs A recalled:

“On Monday morning at about 8am he [Mr A] rang me and said that I could pick him up and take him home, he had a further blood test, ECG etc, before leaving at about 11am with the reassurance he had a Hiatus Hernia/GORD and there was nothing wrong with his heart.”

Review of troponin I result

A fax of Mr A’s outstanding test result indicating a troponin I level of 0.5 (normal is considered less than 0.2) was received on the evening of Day 2, by the afternoon shift registered nurse who contacted Dr B (who was at home) at approximately 7.30pm.

Dr B considered that the result represented a marginal increase and did not warrant immediate follow-up. He did not return to the hospital that night to review the relevant notes and commented that (in retrospect) it was unlikely that he would have recalled Mr A that night. He stated:

“No immediate follow-up was planned on the evening of [Day 2], after I was informed about the abnormal troponin I result. This was because the elevation was marginal (although still significant) and because the patient lived a considerable distance away (about 40 minutes drive). It was my intention the next day to telephone [Mr A’s] GP, [Dr C] and ask him to follow this up.”

On the morning of Day 3, medical officer Dr K reviewed Mr A’s notes and discussed the troponin I result with Dr B. (Dr K explained that it was her practice to review patient notes when sighting an abnormal result of a patient she was unfamiliar with.) Dr K stated:

“In light of his history of chest pain and his slightly elevated troponin I result I reviewed his ECGs closely. I felt that his serial ECGs showed very subtle inferior T-wave changes.

My impression at that time was that it was very likely this man’s chest pain was in fact cardiac in nature. I felt that he needed to be followed up and I asked [Dr B] to arrange this since he had cared for him the day before. I believe I talked to [Dr B] late in the morning of [Day 3] but do not recall the exact time. We discussed that, at a minimum, he needed to be commenced on treatment (aspirin, beta-blocker), blood test for fasting lipids arranged and a cardiologist consultation (by phone).

[Dr B] elected to contact [Dr C] to get him to arrange all this. I do not know what time he contacted [Dr C]. Later that same day, I hand wrote a note on his typed discharge letter (in his medical notes, with a copy faxed to his GP) as I noted that there was no written record in his notes of this subsequent development.”

The handwritten margin note by Dr K stated:

“[Day 3]

Notes reviewed due to Trop I (2nd) 0.5

Serial ECGs shows developed some subtle T-wave changes inferiorly

— [Dr B] informed of same, who arranged GP follow-up. Needs Aspirin/ Betaloc/check lipids/stop smoking etc.”

Dr B stated:

“The following morning was busy and I had not yet phoned [Dr C], when [Dr K] came to me with the abnormal troponin I result. She pointed out the progressive nature of the inverted T-waves in the inferior ECG leads and we agreed that the two together were significant. I telephoned [Dr C] immediately after this conversation (around 11.30 or so) and told him what happened. He agreed to follow up [Mr A], urgently.”

Referral to general practitioner care

Dr B recalled that he provided Dr C with a full description of events and requested urgent follow-up with aspirin and beta blockers to be commenced immediately, as well as an urgent referral for specialist cardiology treatment. He also advised Dr C that Mr A was under the impression he had GORD. Dr C agreed to contact Mr A immediately. Dr B explained that he did not document his conversation with Dr C apart from writing “phoned [Dr C]” on Mr A’s troponin I result form.

In contrast, Dr C recalled that Dr B contacted him at approximately 4pm and advised that Mr A had a slightly positive troponin test. There was no mention of ECG results and no urgency expressed. He stated:

“I was informed that [Mr A] was discharged with Gastro-oesophageal reflux the day before, but that his blood test for troponin I had come back slightly positive and that [Dr B] wanted to know if I could follow up [Mr A] to investigate and treat his ischaemic heart disease.”

Ms D (Dr C’s practice nurse) confirmed that Dr C asked her to phone Mr A at approximately 4pm. Mrs A provided the following transcript of the answerphone message:

“Hello, a message for [Mr A]. This is [Ms D], practice nurse at [the medical centre]. Just a call on behalf of [Dr C], he’s just wondering if you would be happy to come and see him sometime tomorrow. Just a follow-up of what you have been experiencing at [the rural hospital]. If you would give us a call that would be great, thanks very much.”

Mrs A checked her answerphone at 5.30pm and received the above message (left at 4.40pm).

Dr C’s surgery received a faxed copy of Mr A’s discharge summary and troponin I test result at 5.40pm that evening.

Subsequent events

Due to poor weather on Day 2, Mr A remained at home. However, on Day 3 Mr A returned to his usual farm work (working until 10pm). On a following night, Mr A was woken by severe chest pain. He decided to take a walk and returned to bed at about 4.30am. At approximately 5.00am Mr A went into cardiac arrest and died. Mrs A recalled:

“At about 3am [Mr A] woke with a very bad chest pain and decided that a walk might help, he walked around the yard for a period, then came back in and sat in the kitchen

before coming back to bed around 4.30am. At about 5am I heard a noise like he was snoring, but different.

I checked [Mr A] and thought 'he is dying', I rang 111 and administered CPR until the ambulance arrived. They took over but were unable to resuscitate him; the local GP, [Dr C], arrived and confirmed he was dead. [Dr C] filled in the death certificate stating Mr A had died of a heart attack.

The undertaker arrived, took [Mr A] away and arranged for the funeral."

Mrs A met with Dr C the following month at his rooms (supported by the local health and disability consumer advocate). Dr C advised Mrs A that there was no doubt of the cause of death because of the information he had received from the rural hospital. Mrs A stated:

"It was then revealed that the phone call on the Tuesday afternoon asking [Mr A] to come in and see him was as the result of a call [Dr C] had received at 4.20pm on that same afternoon from [the rural hospital], telling him that they had made a mistake and there was an elevation in the T-cell [troponin I test result] that indicated that [Mr A] had had a heart attack on the Sunday when he was brought into [the rural hospital]. [The rural hospital] also told [Dr C] that the ECG readings had been read wrong and there were indications of abnormal findings.

When the blood test results were faxed to [Dr C] it appears that they were received by [the rural hospital] about 11.30am on [Day 2] and it took until 4.20pm on [Day 3] before they rang the GP and advised him to get his patient to [the first public hospital] for immediate treatment.

When [Dr C] received the phone call from the emergency services on the Wednesday morning he had no doubt what was happening to [Mr A] as he had received the information from [the rural hospital] just twelve hours previously. [Dr C] didn't need to arrange an autopsy to find out the cause of death, because he knew."

Comment by Dr B

Dr B commented that Mr A's ECG readings were "essentially normal", although he observed an equivocal finding of T-wave inversions in standard lead III and lead aVF. These T-waves became more pronounced and deeper in subsequent ECGs, which Dr B considered was due to the different positioning of the ECG leads. Dr B commented:

"[Mrs A] seems to be under the impression that I misread her husband's ECG. However, the ECG changes were both subtle and non-diagnostic. Even the notoriously over-sensitive diagnostic algorithm in the ECG machine (which I resolutely pay no attention to) gave [Mr A's] ECG as 'normal'. It was only in light of the raised troponin I that the subtle changes became more significant (although the significance was still uncertain).

Recent research has shown that even low levels of troponin I are significant in terms of mortality. However, it still seems the practice of our local cardiology department to

treat such levels as non-urgent, especially when not accompanied by frequent bouts of chest pain. Although angiography is indicated, it is often done at the next convenient time, rather than immediately.

Having said the above, I should note that, had the troponin I result been available immediately, [Mr A] would not have been discharged at all. It is our practice to keep all patients with positive troponin results until definitive angiography (or at least a cardiologist's opinion that angiography is not required)."

Dr B advised that since these events all troponin test results are now immediately discussed with a cardiology registrar at the first public hospital when they are received, and patients are also contacted immediately. Dr B explained:

"I should point out that this is the first time I have ever seen an elevated troponin I result following a negative troponin T spot test, normally the opposite occurs — the spot test is mildly positive and the troponin I lab test is negative."

Information from the rural hospital

The following information was provided by Mr J, Chief Executive of the rural hospital, in response to questions asked by my Office:

"1. What advice/guidance, and when, did [the rural hospital] provide its Medical Officers on the relative sensitivity of the bedside troponin T tests versus the laboratory troponin I test?

[The rural hospital] medical officers are afforded a variety of training in laboratory tests and testing methods and each test result clearly indicates whether the testing is within range or not and any specific information the testing laboratory feels is appropriate. From the range of opinions even expressed within this investigation, it is not an exact science as to whether either 'T' and/or 'I' tests should be used.

2. What was the expected turnaround time for troponin I testing during normal working hours?

During normal working hours, troponin I specimens leave [the rural hospital] in the afternoon courier service and testing is provided in [a medical laboratory] in [the first city]. For any abnormal results, fax notification is sent as soon as the results are available and nurses on duty alert the medical officers. All results in normal range would be delivered electronically the next morning to the [medical laboratory] computer in [the rural hospital], printed off and delivered to the nursing office. Medical officers review all testing completed the previous day.

3. What arrangements were there for troponin I tests to be carried out after hours/during weekends and what was the expected turnaround time?

Troponin T rapid testing can be done on site at all hours and all medical officers receive training in how to perform these tests by [the medical laboratory]. Troponin I

tests cannot be processed after hours or on weekends. In acute emergency cases, the medical officer would have to determine if the patient requires transfer to [the first or second public hospital], in conjunction with medical registrars at those locations should they need this type of testing.

4. Why were troponin I tests sent to [the first city] rather than [the second city]?

[The rural hospital] contracts with [the medical laboratory] to provide all laboratory testing services. [The medical laboratory] regional testing centre is located in [the first city].

5. What cardiac rhythm monitoring facilities were available at [the rural hospital], including:

a) was [Mr A's] cardiac rhythm monitored during admission?

[The rural hospital] has a Propaq Model #242 Cardiac Monitor that is typically reserved for use in our HDU room for acute emergency patients. It does not have telemetry connections to either monitor ward patients or transmit to other hospitals. [The rural hospital] also has an ECG machine. Mr A's cardiac rhythm was not monitored during his admission in the ward.

b) were [the rural hospital] nursing staff trained in arrhythmia recognition?

[The rural hospital] nursing staff [are] trained in arrhythmia recognition in a number of ways including:

- cardiopulmonary resuscitation training and updates are compulsory and this test is completed yearly for all nurses.
- All registered nurses have training in recognising cardiac arrhythmias.
- Every 2 years we ensure that all nurses attend a study session with a test relating to cardiac arrhythmias.
- All Emergency Depart[ment] registered nurses have trained and passed the defibrillation certificate. This is updated every 2 years.
- All Emergency Department registered nurses hold the triage certificate which has a large cardiac component.
- Two senior registered nurses hold the advanced cardiac life support certificate and two other registered nurses hold the certificate of emergency nursing. These nurses educate the other RNs [registered nurses] in cardiac arrhythmias on a casual and formal basis.

6. Was exercise treadmill testing available for chest pain patients at [the rural hospital]? If so, please include an explanation of timeframes for when patients could expect to receive treadmill testing.

No treadmill testing is available for chest pain patients at [the rural hospital]. There are only two providers in the area that handle this service, [a clinic] in [the first city] and [the second public hospital] in [the second city]. [The clinic] provides outpatient clinics in [the town] on a monthly basis.

7. Did Medical Officers have any regular training or education from a cardiologist or physician on the assessment of acute chest pain/acute coronary syndromes?

All medical officers undertake supervision regularly and in particular [Dr B] was taking part in [a medical school] postgraduate diploma programme on geriatric medicine as part of his CME. [The rural hospital] believes this programme did provide some of this training for him.

8. How many Medical Officers were employed by [the rural hospital] to cover the roster, including:

- a) how stable was the workforce (how long did Medical Officers usually remain in their position)?

At the time of this event, [the rural hospital] employed 3 full time medical officers. One has been on staff from 10 April 2000, [Dr B] was employed from 4 March 2002 and the third was employed on 24 November 2003 (replacing a MO who was hired on 10 June 2002 and left on 25 November 2003). [The rural hospital] has enjoyed a relatively stable work force for a small rural hospital.

- b) how often was the roster filled with locum medical officers unfamiliar with the hospital?

Once or twice a year, [the rural hospital] must utilise a locum agency to provide weekend on-call coverage of medical officers. Other than this, long serving locums provided weekend coverage on weekends that our own medical officers did not cover. In particular, [Dr G] who initially saw [Mr A] on the weekend in A&E has provided weekend coverage for [the rural hospital] since March 2000 and is completely familiar with all [the rural hospital's] policies and procedures.

9. When did [the rural hospital] decide that [the rural hospital] had appropriate facilities and infrastructure for the assessment and management of patients presenting with acute chest pain, including:

- a) was there any external advice or comment sought from a cardiologist or consultant physician on the requirement of a chest pain assessment?

[The rural hospital] is a level 2 ED and as such is designed to treat, stabilise and transfer any serious acute chest pain presentations in conjunction with specialists at secondary and tertiary centres. Since [the second public hospital] does not have cardiologists, most normal communication [is] with specialists or registrars at [the first

public hospital]. We strive in all situations where there is acute chest pain to follow our chest pain protocol, involve the correct specialists and follow their recommendations. Where, in the opinion of the medical officer, there is not cardiac involvement, the patient can be admitted to [the rural hospital] according to our protocol. In some cases, a patient is admitted on the advice of a specialist for observation or due to lack of resources in the tertiary centre.

[The rural hospital] purchased its Propaq monitor system in 2002 in conjunction with advice from regional cardiologists.

b) was there any external review of the chest pain protocols?

[The rural hospital] understands that [Dr B] completed the chest pain protocol as a part of this post graduate diploma in geriatric medicine. As such we believe that he received guidance and comment on its preparation and applicability during his studies. Subsequently, the protocol has been reviewed specifically for the use of thrombolytics.

...

Our other understanding and, at the time a point of comfort in [Dr B's] abilities, is that [Dr B] in his CV indicates that he was the clinical director of emergency services at [the second public hospital] from 1996 to 2000 with responsibilities of managing the resources available including staffing, pharmaceuticals, diagnostic testing and imaging, ensuring the training and development of medical staff as appropriate, determin[ing] and monitor[ing] the clinical standards of care with the service and ensuring they are in line with international best practice. He participated in front-line medical officer duties from 1995 to 2001 at [the second public hospital]. [Dr B] also was certified in APLSC, EMST and has a post graduate diploma in Primary Emergency Care. We believe that the protocols he developed were in line with those that he wrote while at [the second public hospital] in the positions above.

Since [Dr B] developed and wrote the protocols it is assumed that he was thoroughly knowledgeable on their contents and what was required in chest pain presentations.

10. What is the approximate size of the population served by [the rural hospital]?

[The rural hospital] services an area estimated at 20,000 people"

Review of discharge policy

Mr J advised that the rural hospital's 'Chest Pain Discharge Criteria' policy (dated 19 September 2003) has been reviewed and amendments are currently being considered by the rural hospital's Quality Committee and Clinical Governance Committee. The proposed changes will allow for discharge prior to receipt of outstanding results only in certain circumstances, in which case the patient must be informed of the outstanding result(s) and contacted when the result(s) are available. Mr J provided the following draft protocol (written by Dr B) in relation to discharge information provided to patients presenting with chest pain:

“Discharge information

- A patient may be discharged prior to all laboratory results being available, based on the Medical Officer’s clinical judgement. The patient **MUST** be informed of the outstanding blood results and that contact will be made when results available.
- Upon discharge the patient is advised who to contact if they have any further chest pain eg GP, Ambulance, nearest hospital. This is documented in the patient’s medical record.
- Discharge information must include instructions on activity, diet, follow-up arrangements, medications and other issues relevant to [a] patient’s condition. The instructions are to be documented in the patient’s medical record and on the discharge letter, which is given to the patient prior to discharge.”

Mr J commented that given the distance between the rural hospital and main centres, and the length of time to receive blood test results, this policy will allow for clinical judgement and patient preference to be adequately addressed.

He noted that all Mr A’s tests taken prior to his discharge indicated no cardiac involvement in his presenting chest pain symptoms.

ACC findings

ACC accepted Mrs A’s claim for medical misadventure on the basis of medical error. ACC found that Dr B did not observe a reasonable standard of care and skill, based on independent expert advice obtained from emergency physicians Drs Scott Pearson and Andrew Swain.

Drs Pearson and Swain considered that Dr B misinterpreted Mr A’s ECG results. Dr Pearson stated:

“A repeat ECG [taken shortly prior to [Mr A’s] discharge] was, in my opinion, misinterpreted as normal. The previously upright or flat T-wave in lead aVF was now inverted.”

Dr Swain stated:

“On my review of the ECGs I note that the trace recorded by ambulance staff reveals biphasic T-waves suggesting coronary artery ischaemia. More significantly, abnormal Q-waves and T-wave inversion consistent with ischaemia were present in lead III on admission to hospital, they persisted on the evening of [Day 1], and became more marked with extension into lead aVF by the morning of [Day 2]. These changes cannot simply be attributed to the location of the ECG electrodes. The abnormalities may have appeared subtle or equivocal to [Dr B] but they raised concern on the part of [Dr K] and they should have reinforced the need to detain [Mr A] until the final troponin-I report came through.

...

His [Mr A's] electrocardiograms revealed ischaemic changes affecting the inferior part of the heart and these changes increased and became more extensive in the sequence of electrocardiograms recorded.”

Dr Swain considered that the quantitative troponin I test was the preferred option for confirming whether damage to the heart had occurred (primarily because of the endorsement by the medical laboratory). In these circumstances, discharging Mr A before receiving the results of this test was a breach of the rural hospital discharge protocol. He stated:

“I do not understand why [Mr A] was discharged from hospital before the result became available. Even the chest pain pathway employed at [the rural hospital] states that patients must have normal cardiac enzyme levels prior to discharge and it is well known that these levels may not rise until at least six hours after myocardial infarction.”

Dr Pearson agreed that the troponin I laboratory test was a more reliable marker of myocardial damage than the troponin T ‘spot’ test:

“The troponin I test is known to be more sensitive. In my opinion, [the rural hospital] should discuss the implications of this with their nearest cardiologists ([at the first public hospital]). It may be that patients should not be discharged until a troponin I result is obtained. Conversely, a positive troponin T would allow early referral to the tertiary centre.”

Drs Pearson and Swain also considered that Mr A's troponin test result was of serious concern and required immediate action. Dr Swain stated:

“He [Dr B] did not think an immediate response was necessary. However, the troponin I level was 0.5ug/L, which is more than twice the upper limit of normal quoted by [the medical laboratory]. In my opinion, any elevation of the troponin level is normally considered abnormal. The patient is then referred promptly for medical or cardiology

opinion. (Certain disease or conditions can cause an unexpected rise in the troponin level but these were not present in the case of [Mr A].)

[Dr B] states that [Mr A] lived a 40 minutes drive from [the rural hospital]. In my opinion this should have prompted him to earnestly recall the patient as the further [Mr A] lived from hospital, the less would have been his chance of surviving a serious disturbance of heart rhythm. Cardiac arrhythmias are the commonest cause of death following a myocardial infarction and they most frequently occur during the first 24 hours.”

Drs Pearson and Swain considered the transfer of responsibility to the general practitioner for follow-up treatment was inappropriate in the circumstances. The combination of results warranted immediate action including readmission and specialist intervention by a cardiologist. Dr Pearson stated:

“Despite [the] best efforts of medical practitioners, a minority of patients with chest pain will initially be assigned an incorrect diagnosis. However, risk of adverse event or sudden death must be minimised by hospital processes and cautious management.

...

[Mr A] had clearly suffered a small myocardial infarction, confirmed by the elevation in troponin I and ECG changes.

...

I believe it is inadvisable and inappropriate *in this situation* to delegate the responsibility of contacting [Mr A] to his GP. This is because [Dr B] had all the relevant information, was familiar with the patient and was familiar with the urgency that the situation required.”

Independent advice to Commissioner

Physician advice

During the investigation I obtained expert advice reports from consultant physician Dr Kingsley Logan (dated 10 October 2004, 30 December 2004 and 12 August 2005).

The following expert advice was obtained from Dr Logan on 10 October 2004:

“Expert Advice Required

Do the ECG recordings of [Day 1] taken in the ambulance show an abnormality? Please explain.

There are a number of discrepancies [...]. Time of onset of symptoms is given in the handwritten ambulance note as at 12.00, the first ECG strip is dated [Day 1] 11.55. Aspirin is given according to the handwritten note at 12.30 and observations are recorded from 13.00 hours.

There are 3 rhythm strips one of these show biphasic T-waves in lead II it is dated [Day 1] 11.55 but has a different ID number from the other 2 strips that have been photocopied onto the sheet.

The 2 strips show quite considerable baseline artefact and cannot be easily interpreted. These have not been referred to in any of the clinical notes either in A&E or the admission notes and one of the issues therefore is whether this was available or transferred to the admission notes and were part of the hospital record.

Lead placement in patients will often vary in patients and in particular the so called limb leads will show a truncated appearance if the leads are all placed on the torso, this is the normal position of lead placement in monitor leads. The follow up ECGs at [the rural hospital] did not show the changes evident in lead II on the trace [with the different] ID number.

Did [Dr B] undertake an appropriate assessment of [Mr A] on [Day 2]?

[Dr B] consulted the notes, which suggested that the pain was non cardiac, no specific anti-anginal therapy had been commenced, and there had been no recurrence of pain. [Mr A] was ambulant and all of the blood counts were normal. This included representative CPK blood tests which until recently was the blood test done to exclude myocardial damage. There was no supportive evidence to suggest he had symptomatic IHD [ischaemic heart disease]. Unfortunately there is no test that can predict sudden death.

A negative troponin T or I at 18 hours would normally be sufficient to exclude myocardial damage and in many centres a troponin I is only done when the screening spot troponin T is in the indeterminate range.

The risk factors for sudden death include age, hypertension, left ventricular hypertrophy, intraventricular conduction block, elevated serum cholesterol, glucose intolerance, decreased vital capacity, smoking, relative weight, and heart rate identify individuals at risk for sudden cardiac death.

Smoking is an important risk factor. In the Framingham study, the annual incidence of sudden cardiac deaths increased from 13 per 1000 in non-smokers to almost 2.5 times that for people who smoked >20 cigarettes per day. Stopping smoking promptly reduced this risk, which may be mediated by an increase in platelet adhesiveness, release of catecholamines, and other mechanisms. Elevated serum cholesterol appears to predispose patients to rupture of vulnerable plaques, whereas cigarette smoking predisposes patients to acute thrombosis.

Impaired left ventricular function appears to be the most important predictor in men. The following 4 variables identify patients at increased risk of sudden cardiac death: (1) syncope at the time of the first documented episode of arrhythmia, (2) NYHA class III or IV this relates to dyspnea or shortness of breath with minimal activities or at rest, (3) ventricular tachycardia/fibrillation occurring early after myocardial infarction (3 days to 2 months), and (4) history of previous myocardial infarctions.

There were therefore no high risk features and indeed his TIMI risk score on presentation was in the order of 1 which carries an overall risk of 3% that is for death or myocardial infarction in a patient presenting with unstable angina and an ECG that does not show an acute myocardial infarct. This is at the lowest level of risk going up to a TIMI risk of 19% in those who have a score of 6 or 7. The maximum is a score of 7.

[Mr A's] presentation and his profile on a TIMI risk score was 1/7 at the lowest end of the scale and tragically demonstrates that no tool can accurately predict sudden death.

Did [Dr B] make appropriate recommendations in relation to [Mr A]?

Appropriate recommendations were given in that the cause of the pain was thought to be epigastric and gastro-intestinal. [Mr A] was not regarded as having IHD, he was not given aspirin or anti-thrombotic therapy. He was however given instruction to report promptly with any recurrence of chest pain.

The issue is one of risk. There is always a risk in patients of this age group who are smokers having evidence of symptomatic IHD should usually always [be] considered in the differential. Risk stratification prior to hospital discharge in any patients presenting with chest pain is now seen as the standard of care. This is not achieved in the majority of hospitals in NZ and for many reliance is made on potential risk. [Mr A] was not recognised to have evidence of overt IHD at the time of discharge.

Do ECG recordings taken on [Day 1 and Day 2] show any abnormality? Please explain.

The ECG recordings showed T-wave inversion in leads III and flattening of the T-wave in aVF in the initial 12 lead ECG. The ECG taken later in the day showed 0.5mm T-wave inversion in aVF. The ECG on [Day 2] showed similar features with more pronounced T-wave change in lead III the T-wave inversion in aVF reaching 1mm.

Isolated T-wave change in lead III is often regarded as being normal and therefore the interpretation is in aVF. The T-wave change is subtle [and] could be overlooked by non-specialists.

Although the electrocardiogram is an invaluable aspect of every cardiovascular examination, this rarely permits establishment of a specific diagnosis.

The range of normal electrocardiographic findings is wide, and the tracing can be affected significantly by many noncardiac factors, such as age, body habitus, and serum electrolyte concentrations. T-wave changes are variable and nonspecific and do not form the sole basis for the diagnosis of IHD. The changes were however in anatomical contiguous leads.

I would regard the changes as being subtle; the ECGs did not show evidence of an obvious myocardial infarct.

Did [Dr B] interpret [Mr A's] ECG recordings appropriately?

[Dr B] could not have interpreted [Mr A's] ECG recordings as being indicative of pending sudden death. The changes are subtle but in keeping with ischaemic heart disease.

The establishment of a correct and complete cardiac diagnosis often requires the use of several methods of examination: (1) history (2) physical examination (3) electrocardiogram (4) non-invasive graphic examinations (echocardiogram, radionuclide and other non-invasive imaging techniques) and occasionally (5) specialized invasive examinations, i.e., cardiac catheterization, angiocardiography.

ECG interpretation therefore is seen in the context of the patient's presentation and the confirmatory blood tests.

Was it appropriate for [Dr B] to discharge [Mr A] following further ECG and 'spot' cardiac enzyme testing and before obtaining his outstanding troponin I result?

Yes it was appropriate for [Dr B] to discharge [Mr A] following further ECG and 'spot' cardiac enzyme testing. He had more than spot cardiac testing, there are 3 CPK tests in the notes and whilst it can take 8 hours for the CPK to become positive none of these

were positive and it is important to note that the CPK taken at the time of the troponin I was again low. The classic cardiac enzymes were normal.

The troponin I in this situation can be difficult to interpret. There is a range of false positives that occur with any test and it has been the experience of many institutions to regard a level of 0.5 as being indeterminate. At our institution we do not regard this level of troponin I as being diagnostic and our tertiary referral centre has requested that we confirm any troponin I level below 3.0 with a troponin T done by a laboratory analyser. We would even consider doing a CPK to confirm a troponin I result in this range.

My understanding is that because of the variability of this test [the first public hospital] has stopped doing the troponin I. Our regional cardiology referral centre has also requested that only the troponin T is performed in the evaluation of these patients given the variations we have seen with the troponin I.

I have had discussions with the cardiologists at [the first public hospital] re the significance of a troponin level of 0.5. A result in this range would have been regarded as being in the indeterminate range.

The troponin test is a relatively new test and many of the laboratories had established analysers with considerable capital expenditure invested that would then dictate whether a troponin I or T would be done. Depending on the analyser availability, the regional laboratory would do a troponin I or T. [The first public hospital] has changed their analyser so that that they are now only doing the troponin T.

Please explain the diagnostic value of a troponin T ‘spot’ blood test when compared with a troponin I blood test, including how this may change over time.

The troponins are now regarded as a marker of cardiac damage. The value becomes positive from 3 hours but is regarded by most as being confirmatory at 12 hours. The level can stay elevated for as long as 14 days. Many centres would regard a 12-hour level as being representative of the index pain.

The troponin T spot test is highly regarded if this is negative, when the test is in the indeterminate range confirmation is required using an analyser and depending on the regional laboratory troponin T or troponin I. The troponin T has increasingly been accepted as the standard by cardiologists in NZ. There are many centres that would not request a confirmatory test when the screening spot troponin T is negative. As with all tests there is a range we accept as being positive, indeterminate and negative. In addition false positives occur in both assays; the following is contained within the product information brochure accompanying troponin I.

It is well recognised that fibrin can result in low level false positives. This occurs particularly where samples do not clot adequately before centrifugation. Samples must be free of fibrin or other particulate matter. In addition Heterophilic antibodies in human serum can react with reagent immunoglobulins, interfering with in vitro

immunoassays. Patients routinely exposed to animals or to animal serum products can be prone to this interference and anomalous values may be observed.

It has been the experience for many centres to regard a low level of troponin I as being indeterminate and requiring confirmation.

[Mr A] was discharged without being informed about his outstanding blood test result. Was this appropriate?

It is unusual that a troponin I was requested in patients with normal cardiac enzymes and normal screening troponin T. In our institution we only request a confirmatory troponin when the troponin T level is in the indeterminate range. Nonetheless patients should be informed of outstanding blood results and arrangements should be made to discuss/address these. There has to be a formalised process where outstanding results are actioned or handed on.

Was it appropriate that [Dr B] did not recommend follow-up or review after he was informed of the results of [Mr A's] elevated troponin I result?

[Dr B] did not initially consider that the troponin I was relevant given the normal cardiac enzymes and troponin T. The ECGs and blood results were reviewed by Dr B and his colleague the following morning, they then concurred that the chest pain should be regarded as being cardiac. The issue at that stage was a series of negative cardiac enzymes negative troponin T and a troponin I in the indeterminate range.

Reviewing the ECGs the changes do not show evidence of transmural or wall thickness myocardial infarction. The ECG recordings showed T-Wave inversion in leads III and flattening of the T-wave in AVF in the initial 12 lead ECG. Isolated T-wave change in lead III is regarded as being normal and therefore the interpretation is in AVF. The T-Wave change in AVF only just reached 1mm on [Day 2]. These changes are in an anatomical contiguous area and in the context of chest pain should be regarded as being indicative of ischaemic heart disease.

The blood results were not diagnostic and whilst in retrospect all these factors weigh heavily, the isolated troponin I of 0.5 falls into an indeterminate group. Given that [Dr B] considered that [Mr A] did not have a diagnostic ECG it was not unreasonable to initially discount the isolated troponin I that became available on the evening of [Day 2].

When, in your view, did [Mr A's] test results indicate myocardial infarction? Please explain, including what would be the most appropriate medical response?

The issues related to troponin I and T have been detailed. Creatine phosphokinase (CK) rises within 8 to 24 hours and generally returns to normal by 48 to 72 hours, except in the case of large infarctions, when CK clearance is delayed. Lactic dehydrogenase (LDH) rises later (24 to 48 hours) and remains elevated for as long as 7 to 14 days.

Risk factor assessment intervention and risk stratification either by angiography or at the least by exercise testing should be done in patients who present with chest pain thought to be cardiac. Ideally this is done prior to Hospital discharge [for patients] who are troponin negative. When the troponin is positive risk stratification by angiography prior to discharge has now become the standard. A recent survey of all NZ hospitals — private and public — has shown that only 39% of hospitals are able to do this.

[Mr A's] presentation and his profile on a TIMI [thrombolysis in myocardial infarction] risk score was 1/7 — this is at the lowest end of the scale and tragically demonstrates that no tool can accurately predict sudden death.

[Dr B] stated that he advised [Dr C] at 11.20am that [Mr A] required immediate follow-up (although [Dr C] has said that urgency was not conveyed to him). Would [Dr B's] actions on [Day 3], as he has described them, have been appropriate?

Patients presenting with chest pain who are thought to have a so-called non ST elevation myocardial infarct (non STEMI) by definition are those who present with a diagnostic troponin rise with or without specific or non-specific ECG change are normally given the benefits of anti-coagulants, cholesterol lowering drugs and Beta Blockers together with aspirin viz risk factor assessment intervention and the risk stratification by angiography.

The issue is that [Mr A] was not considered to have a so-called non STEMI on admission and whilst it was recognised eventually this was cardiac he did not fall into the obvious high risk group of patients who are likely to suffer sudden death.

It becomes a question of opinion as to how the urgency of the situation was conveyed. All communication regarding patients should summarise the situation, as the clinician perceives it and provide advice as to what should happen if circumstances change. A written record of this communication should follow this communication.

This case emphasises the need for the hospital medical record to be available to health professionals across the continuum of care. There are now web browsers that can access hospital records from a remote location whether by the referral centre, small hospital or general practitioner via password encrypted access.

Results of test can be rapidly accessed and actioned, rapid electronic confirmation of discussions can be made and provides the foundation for safer medical practice. Many practices do not have a secure email network that allows for direct patient information to be sent via email without password or encryption — again there are now electronic systems that allow for this.

Do you consider that the procedures at [the rural hospital] for patients suffering potentially cardiac symptoms met required guidelines and standards?

There are several considerations that need to be considered in patients presenting with chest pain and the issue that needs to be faced at [the rural hospital] is the procedures they follow in risk stratification.

The traditional criteria set forth by the World Health Organization (WHO) to diagnose acute myocardial infarction (AMI) in patients presenting with suggestive symptoms include specific changes in the electrocardiogram (ECG), chest pain, and the appearance of the enzyme creatine kinase (CK) or in modern terms troponin and ECG changes have been paramount in the diagnosis of AMI.

Patients presenting with chest pain who are thought to have a so-called non ST elevation myocardial infarct (non STEMI) by definition are those who present with a diagnostic troponin rise with or without specific or non-specific ECG change are normally given the benefits of anti-coagulants, cholesterol lowering drugs and Beta Blockers together with aspirin viz risk factor assessment and intervention. Risk stratification either by angiography [or exercise testing] ideally should be done prior to Hospital discharge. Inpatient exercise testing is now done as a routine in most hospitals where doubt exists and is done usually under the supervision of a physician or cardiologist. There is a considerable outpatient waiting list in most centres. The issue of inpatient angiography and the management of these patients in NZ was addressed in a nationwide audit by Dr Chris Ellis, GD Gamble, JK French, G Devlin, JM Elliott, S Mann, P Matsis, M Williams, HD White for the NZ ACS (acute coronary syndrome) Audit Group in 2002. Only 39% of patients in NZ were able to do this within current guidelines.

Do you consider that the procedures at [the rural hospital] for troponin testing were appropriate?

Troponin T is used as bedside analyser. There are major cost implications for doing the analyser troponin and for many hospitals in NZ the sample has to be couriered to a regional centre. [The rural hospital] has a very robust system of confirmation. It is unusual [for] a troponin I to be requested in a patient with normal cardiac enzymes and normal screening troponin T. In our institution we only request a confirmatory troponin when the troponin T level is in the indeterminate range.

Several studies have clearly shown that elevations in troponin I and troponin T are associated with greater short-term mortality (30-40 day mortality rates). Others have also demonstrated that increases in the troponin and CK-MB in the presence of ischemia on the ECG are associated with a greater number of cardiac adverse events within the first 72 hours following admission. The issue becomes one of the level accepted as being significant and that the CPK was always within normal limits.

...

Do you consider that the actions undertaken by [the rural hospital] since this event have adequately addressed any concerns in relation to procedures?

Yes. The investigations and procedures are comprehensive and the concerns have been addressed.

What are the relevant professional standards relating to this complaint and did [Dr B] and/or [the rural hospital] comply with these? If you consider that relevant standards were not met, was the departure minor, moderate, or major?

The outcome of course was major with the death of the patient. Relevant standards were met and whilst we know the epidemiology of sudden cardiac death to a great extent parallels that of coronary heart disease, unfortunately, most of these more stable risk factors lack sufficient sensitivity, specificity, and predictive accuracy to pinpoint the patient at risk with a degree of accuracy that would permit using a specific therapeutic intervention before an actual event.

Patients who present for the first time with chest pain usually need further tests to establish the likelihood of underlying coronary disease and to guide appropriate therapy. The evaluation of chest [pain] should be the first step in an integrated approach to the management of coronary disease, they do need further outpatient management. The issue is then seen in the context of perceived risk and the availability of resources. [Mr A's] ECGs at no stage showed ST depression or elevation.

Sudden cardiac death can be considered an electrical accident because, although many individuals have anatomic and functional substrates conducive to developing a life-threatening ventricular tachyarrhythmia and many patients have transient events that could predispose to the initiation of ventricular tachycardia or ventricular fibrillation, only a relatively small number of patients actually do develop sudden cardiac death. It is this interplay between the anatomic and functional substrates, modulated by the transient events that perturb the balance, and the impact of all 3 on the underlying potential arrhythmia mechanisms possessed by all hearts that precipitates sudden cardiac death.

The issue of inpatient angiography and the management of these patients in NZ was addressed in a nationwide audit by Dr Chris Ellis, GD Gamble, JK French, G Devlin, JM Elliott, S Mann, P Matsis, M Williams and HD White for the NZ ACS (acute coronary syndrome) Audit Group in 2002. A survey of all patients admitted with chest pain in 36 hospitals in NZ from 13/5/02 to 26/5/02 was performed. The information became available in 2003.

There were a number of issues raised that are relevant to this case and indeed reflects practice in New Zealand at that time.

There was considerable variation in the troponin tests — in NZ there were 5 troponin methods used:

Roche Lab T	(16 hospitals)
Roche 'Rapid' T	(3 hospitals)
Abbott Lab I	(13 hospitals)
Bayer Lab I	(3 hospitals)
Ortho Lab I	(1 hospital)

In (61%) of patients presenting with STEMI + Non-STEMI patients neither an exercise treadmill test or (coronary) angiogram to 'risk stratify' patient was done. In similar fashion neither an echocardiogram or (LV) angiogram for LV systolic function assessment was done in 58% of cases.

The conclusion from the group was that optimal treatment of ACS patients across NZ is probably limited by the current provision/structure of the service & probably also by low funding.

Are there any other matters which you believe to be relevant to this complaint?

Bedside tests for cardiac-specific troponins are highly sensitive for the early detection of myocardial-cell injury in acute coronary syndromes. Negative test results are associated with low risk and allow rapid and safe discharge of patients with an episode of acute chest pain from the emergency room. (N Engl J Med 1997; 337:1648-53.)

They investigated 773 patients using the cardiac troponins measured troponin I or troponin T using assay kits for cTnI and cTnT. The monoclonal antibodies used in each assay kits are different, as are the reference standards and can make interpretation difficult in the comparison to the laboratory analysers.

Nonetheless this is one of the papers that has allowed for management of patients presenting with acute chest pain with normal troponin levels who may safely be managed as an out-patient.

There were important exceptions; patients with ST-segment depression or those with ST elevation. [Mr A's] ECGs at no stage showed ST depression or elevation.

Of the 47 patients in whom an acute myocardial infarction subsequently evolved in the absence of ST-segment elevation, 44 (94 percent) were identified by a rapid troponin T assay and all 47 (100 percent) by a rapid troponin I assay. The prognosis of patients with normal values for either troponin T or troponin I was quite good – only 1.1 percent and 0.3 percent, respectively, died of cardiac causes or had a nonfatal myocardial infarction during 30 days of follow-up.

The risk however among patients with a convincing clinical history and a negative troponin test was not evaluated. At no stage did [Mr A] show evidence of ST depression or elevation and he was not thought to have a convincing clinical history.

Patients who present for the first time with chest pain usually need further tests to establish the likelihood of underlying coronary disease and to guide appropriate

therapy. The evaluation of chest [pain] should be the first step in an integrated approach to the management of coronary disease and need further outpatient management. Many of these patients [need] to stay in hospital until they have angiography, in some centres this can be a matter of 5 to 10 days depending on the waiting time in regional hospitals. The patients given the highest priority are those who show dynamic ECG change that is ECG depression that resolves or those who continue to have pain and of course those who are shown to have a diagnostic rise in Cardiac Enzymes or troponins.

The urgency of the situation is very rarely reflected in the tragic outcome we have seen. Given his presentation subtle ECG with normal/indeterminate blood results, there were indicators that Mr A had ischaemic heart disease. He was however able to return to work and partake of fairly physical activity [and this] does emphasise the variability of the disease and limited predictive ability we have in assessing these patients.

Re ECG recordings of [Day 1] taken in the ambulance:

The ECG tracings have not been referred to in any of the clinical notes either in A&E or the admission notes and one of the issues therefore is whether this was available or transferred to the admission notes. It is the practice of some hospitals to keep the A&E notes separate from the admission notes so these are not easily available. Transfer of results and documentation such as ECG tracing taken by the ambulance service needs to be formalised if these are to be regarded as part of the medical record.

This case emphasises the need for the hospital medical record to be available to health professionals across the continuum of care. There are now web browsers that can access our hospital records via a remote location whether by the referral centre viz small hospital or general practitioner. Password encrypted access to these records will allow for timely intervention and provides the foundation for effective communication that will meet the expectations of the medical workforce and public of New Zealand.”

Additional advice

Dr Logan provided the following expert advice on 30 December 2004:

“Re: Concerning the difference of opinion between myself and Drs Pearson and Swain.

I have read the report from Drs Swain and Pearson and would agree that the medical management does constitute medical error as provided in their ACC reports. I am unaware of any emergency physician working in the smaller or remote hospital in New Zealand but hopefully this will change as they become aware of this need. To date the emergency physicians function in larger centers usually tertiary centers with a triage role and ready access to full specialist support. They are not responsible for ongoing management of patients apart from those units which have a short stay admission/24 hour ward where patients are seen and evaluated and discharged.

Cardiac enzymes

1. It is not my considered opinion that the troponin I quantitative test is more sensitive or even more specific than the troponin T. This issue has been recently addressed at our regional cardiac conference where the strong recommendation was that troponin T should be used as the universal standard.

I have also indicated the report from Chris Ellis has highlighted the variance in practice that followed the introduction of the troponin Tests. I have enclosed a copy of the reference. This was published in November 2003.

NZMJ 7 November 2003, Vol 116 No 1185

Inadequate service provision for the management of patients admitted with an acute coronary syndrome in New Zealand: a comprehensive nationwide audit of practice in 2002. C J Ellis, G D Gamble, J K French, G Devlin, J M Elliott, S Mann, P P Matsis, M Williams, H D White, for the NZACS Audit Group. University Department of Medicine, Auckland Hospital, Auckland.

Background Remarkably little is known about patients admitted to a New Zealand hospital with an acute coronary syndrome (ACS). Consequently, sensible policies to guide patient management do not exist. We aimed to collect, assess and disseminate data on patients who presented with an ACS, to improve care through a better understanding of demographics, management and outcomes, throughout NZ. We also aimed to establish a nationwide group of clinicians who could repeat the audit on a regular basis to explore and report on changes in ACS management over time.

Methods All 36 hospitals in NZ admitting ACS patients were approached and a local clinician identified to lead the audit (nil declined). The audit form comprised four pages and collected patient demographics, clinical presentation, investigations undertaken, management given and procedures performed until discharge.

Results For 14 days (00.00 13 May 2002 to 24.00 26 May 2002), there were 930 patient admissions, with 'suspected or definite ACS'. Median patient age was 70 years (range 21-102), 58% male, 81% Caucasian, 7% Maori, 13% other ethnicity. Risk factors included smoking (59%), hypertension (47%), hyperlipidaemia (35%) and diabetes (17%). Investigation rates for the cohort (n = 930) were low (chest X-ray 85%, echocardiogram 20%, exercise treadmill 20%, cardiac angiogram 21%) and 586 (63%) patients had neither a treadmill nor angiogram. By admission diagnosis, and using a standard troponin 'cut-off', 42% patients were troponin positive. Patient presentations were ST-elevation myocardial infarction (STEMI) (11%), non-STEMI (31%), unstable angina (UA) (36%), 'other diagnosis unknown' (22%). Medical treatment of non-STEMI patients (n = 285) included enoxaparin (54%), Daltapalin (12%) and unfradionated heparin (9%).

Revascularisation rates were also low (percutaneous coronary intervention: STEMI (13%), non-STEMI (8%), UA (4%); coronary artery bypass surgery: STEMI (4%), non-STEMI (2%) and UA (4%).

Conclusions A collaborative network of clinicians can audit ACS patient admissions to NZ hospitals. Optimal management of patients is hampered by a limited provision of service.

The considered opinion from our own area is that the troponin T is a more reliable test than the troponin I given that we have seen several false positive tests in patients in the indeterminate range and has led to unnecessary angiography.

The troponin T is now the standard at [the first public hospital] where the hospital has stopped doing the troponin I.

I would also re-emphasise that the troponin T was negative at a representative time that is more than 24 hours after presentation. The CPK is now historical but it is important to note that serial CPK and troponin T were negative when the troponin I was found to be mildly elevated.

It is not the policy at our hospital to do a confirmatory troponin I when the troponin T is negative. We use the same troponin T test, this protocol has followed extensive investigation by our laboratory and has been sanctioned by the Pathology Department at [a tertiary hospital].

The troponin I result is in the indeterminate range and in the setting of normal troponin T and CPK would normally hold little significance.

The significance of an indeterminate troponin I in the context of two hours chest pain and subtle ECG change is of course important and I am unsure whether an additional troponin I would have affected management and the decisions that were made at that stage.

[Mr A] succumbed from presumably a fatal myocardial infarct [a few] days following his initial presentation and more than 36 hours following the final interpretation of the blood results. The report from Chris Ellis and associates highlighted the discrepancies in practice with 63% of patients neither having a treadmill nor angiogram despite being part of a survey and has contributed to our current practice where patients are kept in hospital until they have risk stratification via angiography. This is deemed in all patients who are shown to have a representative troponin elevation.

The subtle changes observed on [Mr A's] ECG were only made once the troponin I result was obtained. [Mr A] was considered after initial assessment in A&E and on the following day on the ward round as having reflux /Upper GI symptoms and was not given the standard treatment of Heparin, aspirin, Beta Blockers and Cholesterol lowering drugs that is normally prescribed in patients suspected as having an acute

coronary syndrome. The doctors did not reach a stage prior to [Mr A's] discharge to consider the troponin I and ECG change.

This must of course address the issue of patients being discharged whilst results are pending. It is unlikely that remote access/electronic access to laboratory results were available at that time in [the first public hospital]. Certainly their current computer system does now allow for that and indeed the 10 hour delay and faxing of result after hours that seem to have been the pattern in 2003 should no longer apply. Doctors and patients can have results of blood tests electronically accessed via a web link and therefore can access these results remotely and can action these in a more timely manner.

ECG results

The ECG changes were subtle and indeed was mentioned by both emergency physicians that they would have regarded the initial ECGs as being normal. Unfortunately the graph showing biphasic T-wave change taken in the ambulance never became part of the medical record. It is not recorded or referred to in the medical notes and is incorrectly labelled and timed.

The Q wave in lead III was never at a diagnostic level, normal guidelines suggest that there should be at least 25% of the ensuing R wave or equivalently 25% of the ensuing R wave, a Q wave in III is often regarded as being normal and usually has to be accompanied by a Q wave in II or aVF to be significant.

T-wave inversion can be non-specific, but in this particular case with involvement of anatomical contiguous leads, I believe the correct diagnosis should have been ischaemic heart disease.

2. [Dr B] should have sought confirmatory result and advice from a cardiologist concerning [Mr A's] condition once he considered that the presentation was one of an acute coronary syndrome. The issue of course at the time of discharge was that [Mr A] was felt to have chest pain on the basis of reflux and was not thought to have significant heart disease. Even with the reflection of the ECG and troponin I result, his opinion seemed to favour that of outpatient investigation and assessment of ischaemic heart disease rather than acute coronary syndrome.

Acute chest pain accounts for approximately 20-30% of acute medical admissions (UK data). However, of these admissions, approximately half will not have acute coronary syndrome (myocardial infarction or unstable angina) as a diagnosis. The interpretation of chest pain given the variability and nature is difficult and the following highlights the dilemma practitioners have to face.

Chest pain symptoms (Lee et al, 1985):

MI (Myocardial Infarct) USAP (Unstable Angina Pectoris).

'Burning/ indigestion' as the main feature –

MI	9.6%	(10/104)
USAP	6.3%	(9/143)

Other diagnosis	6.9%	(24/349)
‘Sharp/ stabbing’ as the main feature –		
MI	7.7%	(8/104)
USAP	18.9%	(27/143)
Other diagnosis	35.0%	(122/349)

Lee et al commented that there was no better indicator of low-risk than a normal ECG in the Emergency Department. However, 6 of 114 patients (5.3%) with normal ECGs had a final diagnosis of acute coronary syndrome. A further 56 patients out of 222 (25.2%) with non-specific or non-diagnostic ECGs had a final diagnosis of acute coronary syndrome.

It is estimated that 1-5% of patients with acute chest pain that are directly discharged from US Emergency Departments have missed myocardial infarction.

The short term mortality in the mistakenly discharged group is not insignificant 26% (9/35) with missed myocardial infarction died within 72 hours, compared with 12% (13/105) of those admitted with myocardial infarction (Lee et al, 1987).

The report from Dr Ellis highlighted the practice in New Zealand at that time and indeed the figures suggest that the vast majority of patients were not having timely intervention by cardiologists.

Practice has changed with all patients suspected as having an acute coronary syndrome being kept in hospital until they obtain inpatient angiography. Contrary to the opinion that the patient would have simply been transferred to a tertiary centre for angiography, the waiting lists in most of these centres are a matter of some days/weeks for angiography. This does of course necessitate that they remain in their community/secondary hospital until an angiogram is performed. Thus, patients usually remain at the peripheral hospital until the day prior or on the day of angiography when they are transferred to the tertiary centre.

Discharge

There is always a differential diagnosis in patients who present with chest pain and whilst it was considered that [Mr A] had reflux, [Dr B] should have considered that [Mr A] may have had ischaemic heart disease. Indeed all differentials of chest pain must consider ischaemic heart disease. Current practice would dictate that formal risk factor assessment should be done in all patients who present with chest pain. The report from Chris Ellis suggests that only a proportion of these patients were being given preventative therapy. Beta blockers are usually given to patients who are felt to have evidence of overt ischaemic heart disease and, again, in this setting these would normally be prescribed. Although the use of beta blockers is widely accepted as beneficial in the treatment of Q wave infarction, a benefit with non-Q wave infarction has not been proved. At least three separate studies have evaluated the use of beta blockers in acute non-Q wave infarction, with conflicting results. On

the basis of limited and conflicting evidence on the use of beta blockers in non-Q wave infarction, the American College of Cardiology has given beta blockers a class IIb designation (usefulness/efficacy is less well established by evidence/opinion) in the setting of non-Q wave infarction.

Dr Swain felt that chest pain protocol had been breached on two occasions, at the time of discharge [Mr A] was felt to have normal cardiac enzymes and ECG was [not] considered to be indicative of an acute coronary syndrome. It is only following discharge that the chest pain protocol was breached.

[Dr B] has indicated that he gave advice for GORD. [Mr A] was referred back to the general practitioner given that the patient was living 40 minutes from the hospital and the general practitioner was in closer proximity to the patient. The issue is then one of responsibility of care and as this was handed back to the general practitioner, whether all of the relevant information was also handed over [to] the general practitioner.

Even with the reflection of the ECG and troponin I result, [Dr B's] opinion seemed to favour that of outpatient investigation and assessment of ischaemic heart disease rather than management of the acute coronary syndrome and indeed is reflected by the fact that the general practitioner delegated responsibility to his clerical staff to contact [Mr A] and arrange an out-patient appointment. There is no formal record of this interaction.

[Mr A] did not have a clear understanding of the consequences of further chest pain, given that he did not contact an ambulance service/the local hospital or his own doctor, when he had a further recurrence of pain. Clearly this would have been affected by direct instruction interaction with the patient both verbal and written.

1. [Dr B] suggested a trial of Losec given that at the time of discharge he felt the patient had reflux. He was not able to make the correct diagnosis at the time of discharge but given that he felt this was reflux it was appropriate that he suggest a trial of Losec.
2. The medical manual may state that discharge instructions are to be documented in the medical record and on the discharge letter. Most hospitals do have a documentation audit and it would be important to ascertain whether this was a single breach of their policy or indeed whether this policy was strictly adhered to. Stylized instructions for patients with chest pain when they leave hospital can be easily made available and it is almost always emphasized that if there is a recurrence of chest pain, then they should report promptly for medical attention.
3. It was not appropriate that [Mr A] was not provided with a discharge summary when he left [the rural hospital]. The quality and detail in the discharge summary would form part of a basic audit in most hospitals.

Follow-up

1. Having considered the bloods tests available and the diagnosis of simple reflux with what was considered normal ECG and troponins at discharge, his management was appropriate.
2. I am unaware of the follow up arrangements at [the rural hospital] and as to whether outpatient follow up clinics are maintained and whether specialist clinics [and]/or supervision is available to support the MOSS [Medical Officer Special Scale] positions.

There are many patients who were referred via the outpatient system for assessment and management of ischaemic heart disease. It becomes part of the collective responsibility for all those to assess and to stratify those patients who seem to be at greatest risk.

The many risk factors present in patients with ischaemic heart disease do not result in the electrical event of so-called sudden death. To date we really do not have any predictive model. We know, however, that patients who present with a non-ST elevation MI — and that ultimately was the presentation [Mr A] had on this occasion — are advantaged by an early invasive strategy.

Additional matters

1. The first of the TIMI study group thrombolysis in myocardial infarct web site reference is: www.timi.org. There the risk factors in TIMI calculator that has evolved in so called non STEMI unstable angina patients relate to the following:
 - i Age > 65 years.
 - ii Coronary artery disease risk factors. These include sub factors in at least three of these. Family history of high cholesterol, hypertension, diabetes mellitus, active smoker.
 - iii Known coronary artery disease (stenosis >1= to 50%).
 - iv Prior chronic Aspirin use.
 - v > 1 episode of rest angina over the last 24 hours.
 - vi Elevated cardiac markers.
 - viii ST deviation >1+ to 0.5mm.[Mr A] did not therefore fulfil the criteria on ST deviation and as I emphasised, cardiac markers were non-concordant. The risk was addressed in the TIMI IIB trial 14D risk, [Mr A] would go at maximum to a score of 3 accepting that the troponin was positive, which takes it from death from myocardial infarct to a risk from 3% with a score of 0 or 1 to a death from myocardial infarct risk of 5%. That is the risk of developing death of myocardial infarct over the next of 14 days with the risk of developing death by myocardial infarct or needing urgent revascularization by 14 days going from 5% in 0–1 risk to 13% to those with a score of 3.

2. The high risk features have been shown in the TIMI risk profile as above.

[Mr A] presented the risk factors of being middle aged male and a smoker. The TIMI risk score was developed from the TIMI IIB where patients had to have a clear history of ischaemic pain at rest, documented by clinical findings, ECG changes or positive cardiac markers. It has not been tested in patients with chest pain of unclear etiology. The higher the number of these risks factors, the greater risk the patient has of having a cardiac event. In addition it is emphasised in the ACC AHA unstable angina non STEMI guidelines, the high risk patients would also benefit more from more aggressive anti-thrombotic treatment and invasive strategies.

The issue therefore becomes one of assessing risk and whilst I can agree with the opinions of Drs Swain and Pearson that if [Mr A] had been re-admitted and if he had been managed as having an acute coronary syndrome and if he had managed to have an angiogram in a timely manner, that the course may have been different. We are of course presuming that he had a further coronary event rather than a dissecting aneurysm pulmonary embolus or alternative catastrophic event. Myocardial rupture, pulseless electrical activity are possibilities in patients who die suddenly. We know, however, that the vast majority have a shockable rhythm and that if patients are in a situation where this can be effectively diagnosed and treated, that their chances of survival are greatly enhanced. ...”

Responses to provisional opinion

In response to the provisional opinion, Dr B submitted that the care he provided to Mr A was appropriate in the context of a non-specialist, working in a small rural hospital. In these circumstances, he could not be expected to have made a diagnosis of ischaemic heart disease on the basis of Mr A’s ECG recordings prior to his discharge. He submitted that the discharge information he provided to Mr A was appropriate and that it was reasonable for him to elect to transfer Mr A’s follow-up care to his general practitioner. Dr B accepts that his medical notes were not adequate.

Dr E, a medical officer at the rural hospital, also provided a submission in support of his former colleague Dr B.

Diagnosis and discharge

Dr B emphasized that the purpose of Mr A’s hospital admission was to exclude a cardiac cause for his chest pain. He stated:

“[Mr A] was in hospital *in order to exclude a cardiac cause to his chest pain*. That is precisely why he was having serial ECGs and cardiac enzyme blood tests. Similarly, the only reason he was allowed out of hospital was that I had satisfied myself that ischaemic heart disease was unlikely.”

Dr B submitted that it is unreasonable to expect a medical officer to diagnose ischaemic heart disease on the basis of very subtle ECG readings, as this requires the expertise of a specialist. He stated:

“... [T]he ECG software analysis said the ECG was ‘normal’ and this program is notorious for ‘over-reading’ ECGs and reporting abnormalities even where there are none. For this software to miss the abnormality, it must be subtle indeed.

In the face of a normal ECG (in my opinion at that time), normal cardiac enzymes (up to that time), a single non-recurrent episode of chest pain and only one known risk factor (smoking), an urgent cardiology appointment at this time would have been inappropriate, as would preventative therapy (barring the suggestion to stop smoking, which was given).”

Dr B submitted that the expert advice obtained by ACC from emergency physicians Drs Swain and Pearson was not directly applicable to a medical ward situation. Even the tiniest ECG change would result in caution when considering discharge of a patient with chest pain from an emergency department, and all blood test results would be obtained and reviewed. However, Mr A had been observed for nearly 24 hours in the medical ward with multiple ECG recordings and normal cardiac enzymes, and had experienced no further chest pain. In these circumstances, his discharge was appropriate.

Discharge instructions

Dr B reiterated that he provided Mr A with appropriate discharge information and disputed that he gave the impression that Mr A’s chest pain was conclusively non-cardiac. Mr A left hospital before he received his written discharge summary. It was unreasonable to expect him to provide a full explanation of Mr A’s condition, including the significance of outstanding blood tests, when “he [Mr A] elected to leave the hospital at once”. Dr B stated:

“[Mr A] was extremely keen to get back to his farm and did not wish to wait until I was available to sit down with him and discuss his diagnosis more fully.”

Dr B emphasised that Mr A was advised to seek medical assistance for any recurrence of chest pain and subsequently elected to disregard this advice. (Dr B observed that Mr A was also non-compliant with his advice to stop smoking.) Dr B submitted that his omission to inform Mr A of the outstanding troponin test was not particularly significant as he would have told Mr A it was expected to be negative.

Overall, Dr B submitted that his “brief explanation at the bedside” included all the relevant information.

Dr E doubted that more “far reaching” advice could have been provided to Mr A by Dr B, given the diagnosis of GORD at the time of discharge, and noted that the differential diagnoses for chest pain are not limited to ischaemic heart disease. (Dr E has also suggested that, given the outcome for Mr A, it is easy in hindsight to contend that further advice before discharge should have been given.) Dr E commented:

“I write and type these discharge letters myself and can say that ... a discharge letter would not be available at 11.00 when the negative troponin T result [be]came available.”

Dr E observed that the rural hospital discharge letter template does not have an “advice for the patient section” and, in addition, there is no handout sheet of instructions available for chest pain patients at the rural hospital.

Follow-up

Dr B submitted that it was appropriate for him to discuss the situation with his colleague, Dr K, and then organise a cardiology appointment through Mr A’s general practitioner. Dr B disagreed with the views of Drs Swain and Pearson (and subsequently of Dr O’Meeghan) that it was inappropriate to transfer responsibility to Dr C, and commented that the rural hospital staff have often asked general practitioners to follow up unexpected abnormal results. His request was not unusual, in a rural context. Dr B submitted:

- Mr A’s abnormal results still indicated a low risk situation and (rather than re-admitting Mr A) it would be sufficient to provide follow-up care on an outpatient basis;
- preventative medication (such as beta blockers) and an urgent cardiology appointment could be arranged effectively by Mr A’s general practitioner; it would be much easier for Mr A to visit Dr C, than to return to the rural hospital;
- Dr C had previously been reliable with follow-up of patients discharged from the medical ward.

Dr B emphasised that Dr C understood that beta-blockers and aspirin should be commenced immediately, and that the cardiology registrar should be consulted. Dr B stated that Dr C agreed to follow his instructions and “he presumed he [Dr C] would contact [Mr A] immediately”.

Dr B also informed me that his rural hospital colleague was present on the medical ward during his telephone conversation with Dr C. Dr B’s colleague was contacted by my Office and confirmed that Dr B contacted Dr C before midday. Dr B’s colleague was not able to recall the substance of the conversation but stated that Dr B probably requested follow-up that day.

Dr B explained that an outpatient appointment for a formal exercise test was ordered (as a matter of routine) but cancelled when Mr A died. The rural hospital has since confirmed that it holds no record of an outpatient appointment for Mr A.

Follow-up physician advice

On 12 August 2005 Dr Logan provided further advice regarding the issues raised by Dr B:

“Concerning the submissions by [Dr B] and the additional comments by [Dr E].

...

1 In light of his training and experience as a medical officer rather than a specialist should [Dr B] have recognised [Mr A's] ECG as being diagnostic?

...

It remains my opinion that the ECG changes were subtle and could be overlooked by non-specialists. The changes were non diagnostic as regards an acute myocardial infarct (STEMI), there was no ST elevation, certainly there were no changes at any stage to indicate thrombolysis.

The TIMI protocol suggests that the ECG changes necessary in entering patients into the risk calculation in patients who have an acute coronary event, a so-called non-STEMI event, the ECG change should only include 0.5mm ST depression. At no stage was this the case.

It should be emphasized that at no stage were [Mr A's] ECG changes diagnostic for an acute coronary syndrome, but review suggested that the cause of his chest pain was due to ischaemic heart disease.

There were of course no ECG changes at any stage that [indicated] this patient would die suddenly.

Cardiac arrest due to ventricular arrhythmias may be due to chronic scar or to acute MI/ischemia. A chronic infarct scar can serve as the focus for re-entrant ventricular tachyarrhythmias. This can occur shortly after the infarct or years later. Many studies support the relationship of symptomatic and asymptomatic ischemia as markers of myocardium at risk for arrhythmias. In post-mortem studies of people who have died from SCD, extensive atherosclerosis is the most common pathologic finding. In survivors of cardiac arrest, coronary heart disease with vessels showing greater than 75% stenosis is observed in 40-86% of patients, depending on the age and sex of the population studied. Autopsy studies show similar results; in one study of 169 hearts, approximately 61% of patients died of SCD, and more than 75% stenosis in 3 or 4 vessels and similar severe lesions were present in at least 2 vessels in another 15% of cases. No single coronary artery lesion is associated with an increased risk for SCD. Despite these findings, only approximately 20% of SCD-related autopsies have shown evidence of a recent MI. A greater proportion of autopsies (40-70%) show evidence of a healed MI. Many of these hearts also reveal evidence of plaque fissuring, hemorrhage, and thrombosis.

There are many patients who present with chest pain that are considered to be at relatively low risk because they do not have diagnostic ECG change or troponin elevation and are referred and managed as outpatients. In similar fashion there are many patients who have been identified as having ischaemic heart disease who require to wait on definitive investigations and management, the urgent outpatient lists in the

hospital system is measured in months rather than days. In attempting to risk stratify patients who are admitted with chest pain the TIMI score assists us in determining the risk of re-infarction or death at 14 days. I have indicated that calculating [Mr A's] risk was seen to be relatively low, viz.

TIMI Risk Factor Stratification

Recent severe angina not considered at time of presentation by admitting doctor but given the outcome I would consider the pain was ischaemic **yes**

Serial cardiac markers	no
ST deviation >0.5mm	no
Age > 65	no
3 conventional Risk Factors	no
Known CAD	no
Aspirin within 7 days	no

A higher TIMI risk score correlated significantly with increased numbers of events (all-cause mortality, new or recurrent MI, or severe recurrent ischaemia requiring revascularization) at 14 days

- Score of 0/1 – 4.7 percent
- Score of 2 – 8.3 percent
- Score of 3 – 13.2 percent
- Score of 4 – 19.9 percent
- Score of 5 – 26.2 percent
- Score of 6/7 – 40.9 percent

10% of the general population will die suddenly and whilst the recognised risk factors are well summarised in Circulation 1992 Myerburg et al, we know however that the vast majority of these patients who die suddenly will not have any of these established at the time of death given the issue of plaque rupture as the main cause for sudden death.

...

ECG interpretation suggest just that, the changes are interpreted by the reader in conjunction with the information to hand. Changes that could be regarded as being

sinister in some patients can be discounted in others, subtle changes become very obvious as additional information comes to hand and then of course experience weighs heavily in accurate ECG interpretation.

We now have modern facilities that allow for accurate transmission of ECGs to tertiary or specialist centres. ECG transmission is crucial to doctors working in isolation, they need the right equipment and as with laboratory data, need modern electronic support.

The reflective report from [Dr E] raises a number of important considerations. Whilst I indicated in my initial report [Mr A] was not thought to have a convincing clinical history and did not show evidence of ST depression or elevation, the history of chest pain as we now know it, in a patient who has succumbed to IHD, appears far more sinister than what appears in the Hospital notes. The admitting doctor who initially reviewed matters did not consider the pain or the situation to be indicative of IHD and whilst [Mr A] was admitted for observation he was not managed as having an acute coronary syndrome.

In light of his training and experience as a medical officer [Dr B], as many other doctors, would have difficulty in recognising [Mr A's] ECG as being diagnostic of an acute coronary syndrome. He did however recognise these changes as being indicative of IHD with the combination of the troponin I result and once the ECGs were reviewed with his colleague. His approach and management continued to regard this as a relatively low risk situation and to be treated as an outpatient basis; urgency was not communicated to the general practitioner given the sequence of events.

2 Was the information [Dr B] gave to [Mr A] on discharge appropriate?

Appropriate recommendations were given in that the cause of the pain was thought to be reflux. [Mr A] was not regarded as having IHD, he was not given aspirin or anti-thrombotic therapy. He was however given instruction to report promptly with any recurrence of chest pain.

...

There is always a differential diagnosis in patients who present with chest pain and not only ischaemic heart disease but other demanding medical conditions that also present in this way viz. acute vascular catastrophe e.g. pulmonary emboli, dissecting aneurysm and it would be quite inappropriate to give advice on all of these matters at the time of discharge. Fundamentally, however the major concern to patients at discharge with chest pain is whether they have demanding symptomatic ischaemic heart disease.

The deficiencies exposed with this discharge process have been highlighted in the Commissioner's provisional report. In many hospitals it is the role of the Cardiac Nurse Educators to educate and inform patients, this is not a delegated responsibility and it is still the ultimate responsibility of the supervising doctor to adequately inform

patients at time of discharge, to provide them with appropriate information and explanation.

3 Given the circumstances and available resources was it appropriate for [Mr A's] care to be transferred to his general practitioner?

It remains my opinion, that given the circumstances and available resources, that it was appropriate for [Mr A's] care to be transferred to his general practitioner. This does of course presume that all relevant information, that is relevant ECGs, hospital notes and blood results are made available to the general practitioner. I was unaware in my initial report that an appointment had been made by [Dr B] with a Cardiology Registrar, [and] it is not clear that the case was discussed with the Cardiology Registrar.

My understanding is that there is no formal outpatient follow-up at the rural hospital by the Medical Officers for patients discharged from the ward and that follow-up is maintained via the general practitioners or specialist services that attend outpatients on a periodic basis.

The opinion from the emergency physicians that the patient should have been transferred to [the first public hospital] bears further consideration. Emergency physicians do not have any ongoing responsibility for patients who are either treated and discharged back to the patient's own practitioner or are referred onto one of the specialty units in their secondary or tertiary centre. Smaller and rural hospitals in NZ do not have this facility of simply referring on all patients to secondary or tertiary centre and the medical officer's function as generalists covering many acute aspects of medical practice.

The issues that relate to [Mr A] were seen as a low risk situation and in that context; it would be usual practice for hospital doctors to refer patients back to their general practitioners who very adequately perform this function. Many of the issues of ongoing education, behavioural change depends fundamentally on the relationship that has developed between a doctor and their patients and it is very appropriate that [Mr A] was referred back to his own general practitioner.

There are further comments I wish to make.

It is very difficult to comment on what may have happened, and indeed I was asked to provide medical opinion on the sequence of events as they unfolded. Hindsight always obtains perfect vision and whilst one has the strong feeling [Mr A] would not have easily returned to hospital on a simple phone call and there was far more likelihood that he would respond to the intervention given by his own practitioner, this is a matter for speculation and really should not form part of any report.

I do not believe transferring blame to other practitioners is appropriate and it is not my intention to revise the action and opinion of the admitting medical officer. It is most unfortunate that the response from [Dr B] is now levelled at the other practitioners involved in the care. There is little question of the responsibility that lies with [Dr B] as

the medical officer discharging the patient and that this responsibility is completed when all relevant medical information is transferred or handed onto the patient's general practitioner.

There does not seem to have been any sense of urgency transferred to [Dr C] given the phone message left by his receptionist and [I] would again emphasize that [Mr A's] presentation was seen as being relatively low risk, and indeed was continued to be managed in this way.

Prevention of sudden death in patients with known IHD presents a unique challenge and to date the impact has been limited with the major effort directed at survivors of sudden death. We know that only approximately 20% of SCD-related autopsies have shown evidence of a recent MI. A greater proportion of autopsies (40-70%) show evidence of a healed MI. This represents therefore a high proportion of cardiac patients being managed as outpatients.

...

Hospital doctors can only function adequately and safely in the setting of adequate diagnostic facilities viz laboratory and imaging. In effect, these do not simply complement the facility, but define [it]. Accurate laboratory investigation, vital for patient care and safety, should be readily available.

Electronic access to laboratory results is now also becoming a defining quality for care in NZ hospitals. Electronic information needs to be shared by these institutions and indeed across the continuum of care to the primary providers. Medical and public expectation is such [that] these facilities should now be available. There is web based technology that is now available where patient information can be obtained in a secure manner by all providers responsible for the care of their patients.

This in turn raises the issue of troponin results and interpretation. It seems that the method utilised depends on the machines and contracts that are available in various DHBs and laboratories.

There does need to be further education in the acute management and assessment of patients with chest pain and interpretation of the troponin result. The presumption that was made throughout [Mr A's] admission related to his troponin being negative. Clearly, it could not have been positive within minutes or a few hours of arrival and indeed is regarded as being most representative at the 12-hour stage. We are still left with the clinical dilemma in this patient of a positive troponin I in the face of serial negative troponin T and CPK that clearly dominated the clinical decision process.

The case again emphasizes the need to adequately define those patients who can be safely assessed and admitted to smaller hospitals. It also emphasizes the need for adequate risk stratification prior to hospital discharge.

There has been considerable improvement in understanding and management of patients in NZ. The study was completed in 2002 and published in November 2003. Many of the issues highlighted in the Ellis report have been addressed by the institutions involved in the study and emphasizes the need to audit our medical practice.

Until recently there has been no training or structured career for medical officers working in isolated or rural hospitals. There is now a diploma in Rural Medicine and a vocational pathway that is being developed.”

Additional independent advice to Commissioner

Cardiology advice

In light of the complex and conflicting advice on this case, I obtained additional expert advice from cardiologist Dr Tim O’Meeghan.

Dr O’Meeghan provided his initial report on 14 December 2005:

“Thank you for your recent correspondence and copies of the initial report by the Commissioner on this case. I wish to make the following comments as per your request.

The complaints relate to a consumer, [Mr A], who presented with chest pain to [the rural hospital] on [Day 1] and subsequently died.

[Mr A] developed severe prolonged chest pain and presented to hospital via ambulance. His symptoms subsequently settled and initial investigations did not show any significant abnormality. He remained quite well throughout his hospital stay and the initial impression of the medical staff treating [Mr A] was that the symptoms were not cardiac in origin, but it was felt appropriate to keep him in hospital to further evaluate that possibility. The following morning he was quite well and eager to be discharged, according to information received. He was discharged prior to the result of the second laboratory troponin I being available.

[Dr B] did review [Mr A] prior to discharge, the morning following admission, and had the same impression as [Dr G] (who admitted [Mr A]), that the symptoms were gastrointestinal in origin. [Mr A] did not appear to want to spend much time with medical staff going over matters and was very keen for an early discharge. [Dr B], in the knowledge of the serial troponin T point-of-care qualitative test being negative, elected to proceed with [Mr A’s] discharge. Under these circumstances, [Dr B] was in a difficult situation, having to make a decision without all results being available. [Dr B] probably did not appreciate the potential limitations of the troponin T point-of-care qualitative test in terms of its limited sensitivity at low levels of troponin. This is an important mitigating factor as the limitations of the troponin T point-of-care qualitative

test are now widely known to those not familiar with the test and the result is presented to the clinician as a 'negative' without an accompanying comment as to its sensitivity.

Some hours later [Dr B] was advised that [Mr A's] second laboratory troponin I result was abnormal. I note at that point that 2 serial troponin T point-of-care qualitative tests were negative and the initial laboratory troponin I result was negative but the second laboratory troponin I level was significantly elevated, over twice the upper limit of normal. [Dr B] at that point did not attach too much importance to the second laboratory troponin I.

The following morning the notes were reviewed by a colleague and following discussions with [Dr B] the correct interpretation was applied to the clinical situation.

To summarise:

1. [Mr A] had presented with severe chest pain.
2. There had been a serial change in his ECG, which had been discussed with [Dr B] (inferior T-wave inversion which I accept is quite subtle, but this was indeed identified).
3. Laboratory troponin I tests which had shown initially normal level and then a significant abnormal result.

At that point on Tuesday morning [Day 3] (approximately 24hrs after [Mr A] was discharged), [Dr B] had the correct diagnosis; that of an acute coronary syndrome (non ST elevation myocardial infarction). The presence of symptoms, evolving ECG changes and evolving troponin I changes leaves little doubt as to the diagnosis.

It is not clear from the information received why [Dr B] did not discuss this further with a cardiologist/cardiology registrar at [the first public hospital]. [Dr B] was the author of the clinical protocols for chest pain management and in that document, there are clear provisions for abnormal troponin results to be discussed and advice sought.

The usual treatment for a non-ST elevation myocardial infarction (NSTEMI) is admission to Hospital, usually to a Coronary Care Unit type facility, or sometimes a medical ward, but the patients usually have their cardiac rhythm monitored in the Coronary Care Unit. The patho-physiology is usually an unstable coronary plaque with thrombus present and the initial mainstay of treatment is aspirin and heparin usually in the form of subcutaneous low molecular weight heparin with the aim of steadily dissolving the thrombus. Other anti-thrombotic agents are sometimes used in addition to the above agents depending on the clinical assessment of the treating clinician. Statins and beta-blockers are also started when appropriate in the acute phase and other agents are sometimes added as appropriate. Patients usually remain in hospital for at least 2-3 days while treatments and assessments are undertaken. Depending on the clinical course angiography is often considered and discussed with a tertiary centre. A variety of factors are taken into consideration when considering angiography, in particular as noted in previous submission, the components of the TIMI risk score often

form part of the discussions along with the clinical progress, and ECG changes. I note the TIMI risk score in this situation was quite low. An important consideration in any patient is any occupational issues such as vocational driving or occupations involving heavy physical tasks. I note [Mr A] was likely to have a physical job and that would normally be given some emphasis when angiography has been an issue in treating many of these patients in New Zealand. Access was not an issue for [Mr A], as a request for angiography was not made. It is not clear from the information received whether there were particular access issues for angiography in the region at that time.

Following discussion with his colleague [Dr B] elected to delegate the further management of this case to the patient's GP, [Dr C]. Further communications with Mr A were subsequently delegated to [Dr C's] nurse who left an answerphone message at [Mr A's] house. At no point was [Mr A] or his wife made aware of the diagnosis prior to his death. [Dr C] informed [Mrs A] after her husband's death of the diagnosis. [Dr C] was sufficiently certain of the cause of death and signed the death certificate to that effect without discussing with the coroner.

[Dr B] indicated that in his view the usual cardiological advice in these circumstances would be for the patient to have an outpatient assessment. Given the above description of the usual management of patients with acute coronary syndromes (non STEMI), I would be surprised if that would have been the advice and there was no information received to confirm that that would have been the case. In particular, I would not have expected advice from a tertiary centre to the effect that the patient should await an outpatient assessment and continue unrestricted with respect to physical activities in the absence of any further inpatient evaluation including exercise testing.

The decision by [Dr B] not to seek further advice on this case and to delegate further follow-up arrangements to the general practitioner was not appropriate. In previous submissions, it is stated that it is quite common in the rural setting to delegate the responsibility to the General Practitioner for a variety of follow-up issues. This may be appropriate for many conditions. In this particular situation however, given the acute nature and significance of the problem [Dr B] should have sought advice from a cardiologist (as required by the protocols) and communicated directly (or at least attempted to) with [Mr A]. Any delegation of these tasks inevitably results in delays, a loss of emphasis and priority, which this case illustrates. The delegation to [Dr C] was an important clinical issue. This was not documented contemporaneously and there is now conflict between [Dr C] and [Dr B] over the timing, content and degree of urgency expressed during those discussions.

It has been noted that two serial troponin T point-of-care qualitative tests were negative along with the first laboratory troponin I but the second laboratory troponin I result was abnormal. The explanation for the difference between the second troponin T point-of-care qualitative test being negative and the second troponin I test being positive lies in the fact that the troponin T point-of-care qualitative test is relatively insensitive at low levels of troponin elevation. These troponin elevations are important and the troponin T point-of-care qualitative test is simply not sensitive enough to detect these troponin

elevations reliably. In that situation the troponin T point-of-care qualitative test cannot be used as a reliable rule-out test. This is a very important difference between the two assays. The troponin T point-of-care qualitative test is really only useful as a rule-in test, the advantage being that if positive, decisions can be made about patients with a significantly raised troponin more promptly, particularly if there are significant delays in obtaining a definitive laboratory troponin result. (Note that the troponin T point-of-care qualitative test should not be confused with the laboratory troponin T quantitative test the latter being very sensitive particularly for low levels of troponin.)

There was clearly some awareness of this issue as even though both troponin T point-of-care qualitative tests were negative a confirmatory result was sent to the laboratory, although initially [Dr B] did not accord the second laboratory troponin I result with any degree of significance.

I had sought clarification of what advice the medical staff received from the laboratory on the strengths and in particular limitations of the troponin T point-of-care qualitative test but no specific details on this was received. I enclose a copy of advice distributed in [a city] region on the troponin T point-of-care qualitative test outlining this issue.² Ideally the medical staff should have been formally advised of this by the laboratory and they [may] well have been.

Ideally in future a further reminder of the limitations of the troponin T point of care qualitative test should be placed on any result the laboratory issues. ... This is particularly important in circumstances where the tests are used infrequently and it is possible medical staff may overlook this particular detail, which is critically important. This will be of particular assistance to any locum medical staff employed in the future who may be unfamiliar with this issue.

One of the most critical aspects in the assessment of acute chest pain is the timely provision of a laboratory troponin result. In this particular circumstance a laboratory troponin I (Bayer) was used and this is acceptable. It is important to note that there are a variety of different troponin I assays available by different manufacturers and they all have slightly different reference ranges. Any comments on troponin I results must be specific to the assay system in question as the individual levels are not interchangeable between assay systems. For this reason there is a preference for a quantitative laboratory troponin T (not the point-of-care assay) to be used in acute chest pain assessments as there is only one reference range for a quantitative laboratory troponin T and this is the same world-wide and this therefore minimises the risks of any confusion between the various different assay systems. While there is a preference for a laboratory troponin T [it] is certainly quite acceptable to use the Bayer troponin I as was used in this circumstance and in my opinion it provided an accurate and appropriate result.

² See Appendix A.

The whole system of providing a laboratory based troponin assay (as opposed to a point-of-care assay) needs further review. There are considerable limitations in the provision of these results simply through the geography of the environment. While the preferred contractual arrangements have the blood samples couriered to [a city], this introduces a significant delay in the provision of the result illustrated by this case. Most patients have two blood tests to look at their serial troponin levels during the assessment of chest pain. Technical factors sometimes necessitate further sampling in some patients, which obviously introduces further delays. In addition according to information received from [Mr J] (also noted by [Mr E]) this assay is not available after hours or at weekends placing considerable limitations on the assessment of patients who present outside of normal working hours. I have had further discussions with [the medical laboratory] who indicate that quantitative laboratory troponin I results are available after hours including weekends and they have an 'on call' service and will provide a result after hours if requested to do so.

Laboratory troponin results need to be available in a timely manner, for without this it places the medical staff in quite a difficult circumstance, often being asked to make decisions in a particular timeframe without all the results being available. A review of where the laboratory troponin requests should be sent should be undertaken promptly with preference being given to a site that can give a result in an acceptable timeframe, consistently, and available beyond normal working hours. These issues should take precedence over any preferred contractual arrangements for laboratory testing.

Undertaking assessments of acute chest pain in [the rural hospital] presents a number of significant challenges. Firstly the acute chest pain assessments are undertaken by medical officers. This is because [the rural hospital] is very small and the size of the hospital does not justify employment of specialist physicians. In the vast majority of hospitals in New Zealand assessments of acute chest pain are normally undertaken by the physicians participating in a general medical roster who are frequently exposed to this problem, or in some of the larger centres cardiologists will see a significant proportion of these patients. In these larger centres the medical staff would be supported by an on-site laboratory and exercise testing facilities. Undertaking chest pain assessments in [the rural hospital] would require the medical officers to have support and be able to discuss individual patients with the appropriate specialist, in this case a cardiologist, on a case by case basis.

Following admission with a suspected or confirmed acute coronary syndrome, patients are usually cared for in a more specialised facility, often in a coronary care unit for a period of time or in a medical ward with appropriate monitoring. From the advice received from [the rural hospital] the monitoring facilities would appear limited. While many of the patients admitted with acute chest pain will have a stable clinical course, a few will have significant arrhythmias and detection of these is very important in their clinical management.

Experienced nursing staff is extremely invaluable in the assessment of these patients. Following admission to hospital and after a period of settling in, patients will often

volunteer to nursing staff particularly important aspects of their symptoms that are not volunteered in the initial history taken by the medical staff. I note the comments from one of the nursing staff caring for [Mr A] on the evening following admission, that she felt the symptoms were cardiac in origin. Nursing observations, particularly during symptomatic episodes along with ECGs will frequently provide valuable supporting information for reaching a diagnosis and developing a management plan. Nursing staff also need to have an education programme so that they are familiar with the important aspects of chest pain assessment.

Some patients presenting with chest pain will require exercise testing to further evaluate their situation. This is preferably undertaken prior to discharge and there are no facilities for treadmill exercise testing in [the rural hospital]. Any review of the systems of care around chest pain assessment will need to define how patients who are identified as requesting treadmill testing can have these tests undertaken with a minimum of delay. Inevitably, given the geography of the situation, there will be a compromise in the provision of these tests. They are however, very useful and particularly important in the rural environment when many patients will often be undertaking heavy physical tasks as part of the occupation. The timely provision of treadmill testing will allow for some patients to be discharged and an early return to work and advised where testing proves to be satisfactory. Conversely, the treadmill tests will assist in determining those patients requiring further evaluation and appropriate activity levels advised.

Medical officers employed in this setting will have a variety of training and experience. [The rural hospital] would appear to have relatively stable work force of medical officers at present. It would be appropriate to have regular educational sessions for the medical officers on the assessment of acute chest pain (and for that matter a variety of other acute medical problems) and this would serve as the basis for good clinical liaison between [the rural hospital] and other specialist physicians in [the first and second public hospitals] depending on the specialist service. ...

It would be a matter of some priority for [the rural hospital] to seek input into the systems of care around acute chest pain assessment from [the first public hospital] cardiology service. In particular, the chest pain protocol should be reviewed by the cardiology service. The current protocols have been written by [Dr B] but the only external input into these protocols apparently has been the supervisors of his geriatric medicine course he is undertaking [at a medical school]. I do not believe this is appropriate external review and advice for these protocols, and more specific and locally based advice would be far more appropriate. A particular deficiency in the protocols is the lack of detail and clarity around the various troponin assays, in particular, the limited sensitivity of the troponin I point-of-care qualitative test, and how laboratory troponin results are obtained after hours. The time frame of 6 hours (noted in the algorithm appendix 3) for the second troponin test would also be a point of further review. This time frame of 6 hours is in conflict with the laboratory advice accompanying the troponin I result which advise a minimum of 12 hours between the onset of symptoms and the subsequent troponin sample that is used to rule in/out an

acute coronary syndrome. The timing of the serial troponin tests is a critical factor in any protocol for chest pain assessment.

In summary, there are a variety of individual clinician issues as well as system and organisational factors that have contributed to an adverse outcome:

1. Delays in obtaining definitive troponin results.
2. Limited appreciation of the insensitivity of the troponin T point-of-care qualitative test at low levels of troponin elevation that are clinically important.
3. Limited appreciation of the clinical response to abnormal troponin level.
4. Lack of contemporaneous documentation of the delegation of care to the GP.
5. Inappropriate delegation of care.
6. Chest pain protocols with insufficient external input.
7. No structured education programme for medical officers with visiting cardiologists on the assessment and management of chest pain including ECG reading.

Additionally, the following factors, while not playing a significant role in this case, will need further review:

1. Chest pain protocols that have conflicting advice with the laboratory on the timing of serial troponin tests.
2. Lack of clarity as to how definitive laboratory troponin results are obtained after hours.

In constructing this report, I have had discussions with the following clinicians:

1. [A chemical pathologist] — troponin assays.
2. [A physician].
3. [A cardiologist].
4. [A pathologist at the medical laboratory] — availability of troponin assays after hours.”

Responses to cardiology advice

A copy of Dr O’Meeghan’s initial report was forwarded to Dr B and the rural hospital for comment. Their responses are summarised below.

Dr B

Dr B reiterated his view that he provided Mr A with appropriate discharge information. It was not reasonable to expect him to provide a full explanation of Mr A’s condition, including the significance of outstanding blood tests, when “he [Mr A] elected to leave the hospital at once rather than wait for me to have the time to see him properly”. Dr B also stated:

“In view of my election of the GP to follow up this matter, it also would be unfair to have expected me to contact him with the updated information (his positive blood test).”

Dr B commented that it was normal practice to transfer responsibility to the patient’s general practitioner, but only in a rural setting. He stated:

“It is of no surprise to me that [the HDC physician advisor] Dr Logan, who works in a medium-sized rural hospital, should accept this method of follow-up, while [the HDC cardiology advisor] Dr O’Meeghan, who works in a tertiary facility, does not ... this practice is only normal in rural setting, where distances are large.”

Dr B explained that although he had stated that Mr A was likely to require outpatient treatment, he did not intend to imply this would have been routine. He believed that Mr A would have been given an urgent treadmill appointment within one or two days. He explained that, at the time, the wait for urgent angiography was five to seven days.

Dr B submitted that the care he provided to Mr A was appropriate in light of accepted practice at the time of these events:

“Although there is no doubt that, in terms of our understanding today, [Mr A] would be considered as having a non-ST elevation myocardial infarction and in need of an urgent cardiology consultation, this was by no means clear [at the time]. At that time, we were only just aware that troponins above 0.03 were considered to represent ‘myocardial damage’ but the significance had not been spelt out, just noted on the lab forms. We certainly had no information from [the first or second public hospital] suggesting any changes to our normal procedures.”

Dr B commented that the rural hospital chest pain protocol was intended for use in the emergency department. There was no protocol for inpatient use at that time. He agreed with Dr O’Meeghan that the rural hospital protocols should be reviewed by a cardiologist.

The rural hospital

The rural hospital provided a report from emergency medical specialist Dr L, who provides supervision for the rural hospital medical officers and is employed at a hospital in the region. Dr L stated:

“Thank you for asking me to review the provisional HDC report. I am left with serious concerns as such a finding will have a dramatic negative result on assessment and care of chest pain for patients other than those within rapid access of a tertiary centre and further seems to put emphasis on tests such as exercise ECG’s that are not regarded by most clinicians, including cardiologists, as useful in the acute assessment of patients suspected of having an acute myocardial infarct (AMI) or acute coronary syndrome (ACS).

1. Wide variation in cardiology opinion.

I respect Dr Tim O’Meeghan as an excellent cardiologist but I do not accept all of his recommendations as they are not consistent with most hospital practices. Nor are they consistent with the recommendations of the NZ ACS Guidelines Group led by Dr Harvey White (published in the NZMJ in Oct 2005), Dr Kingsley Logan (expert physician advisor to the HDC), and the tertiary cardiology group in [the first public hospital] that [the rural hospital] refers to. In fact physicians and cardiologists throughout the world do not agree on a number of factors with considerable variations in hospital guidelines and practice. If the HDC is to start somewhere it will have to be the lowest common denominator of these many opinions ... to find a hospital in breach.

2. This case occurred in 2003.

One must also realise that this assessment of [Mr A] took place [some time ago] and the case must be considered against recommendations current then, not the ideal of a single cardiologist today.

3. Troponin T or I (Laboratory vs POC/point of care testing)

It is recognised that there is approximately a 3 to 10 fold greater sensitivity of the quantitative laboratory test as opposed to the qualitative POC test strip. Despite this difference in sensitivity the POC test will pick up excess of 99% of infarcts if tested serially over a period of hours (time period discussed under point 5). Consequently most physicians including Dr Kingsley Logan (page 3 final sentence in para 5 of his report), the ACS Guidelines Group, and ([The chemical pathologist] — document on POC testing with troponin 2003) accept this as a valid test and able to stand alone in many cases. I do not support the ‘only if positive’ statement of Dr O’Meeghan.

4. Availability of Quantitative Testing.

No small hospital in NZ has access to acute quantitative testing for cardiac enzymes other than POC bedside troponin T. I suspect even [the second public hospital] and many other base hospitals do not have routine cardiac enzyme assays 8am until 5pm, for 7 days per week. (They mostly batch the samples once per day.) The verbal statement quoted from the laboratory for [the town] simply is not correct. They cannot get urgent after hours quantitative troponins as they have to be sent to [the first public

hospital] and that is done the next day by courier as in this case. [A number of rural hospitals] all get their quantitative troponin results the next afternoon from a morning take (only Monday to Thursday in most cases).

5. 4, 6, 8, 10 or 12 hours for testing of cardiac enzymes

It has long been recognised that both ECG changes and cardiac enzyme changes, though these may be present early in large infarcts, they commonly are more reliable if tested serially and after a period of hours from the onset of the pain. Up until the past 2 years the generally accepted standard was 4 hours (*from onset of pain – NOT from when first assessed*) but if the clinician was concerned then a longer period of observation and testing should be given. The standard currently in [the first public hospital] is 8 hours. The ACS Guidelines Group recommend 6–8 hours (note these are 2005 recommendations). [The chemical pathologist] recommends 10 hours based on scientific data on enzyme testing. Our own hospital recommends 4 hours at present but is seriously considering changing to 8 hours. Dr O’Meeghan’s 12 hour recommendation has high cost for a small gain though appropriate if the clinician is suspicious despite negative results earlier.

6. Exercise ECG testing

This is a useful assessment tool (semi-urgent) only for assessment of angina not ACS or AMI. The cardiology department [at the first public hospital] would be hard pressed to offer a service within a week of request. Most small hospitals do not have this service. [Another rural hospital] has the service once per month by a visiting Cardiologist and I suspect [another rural town] has a similar service. [A larger rural town] does have a resident Physician who does them weekly.

7. Monitoring

The only real weakness in [the rural hospital’s] observation/assessment capability is their lack of centralised monitoring facility as I agree it is essential. This does need to be addressed recognizing that most small hospitals are only now upgrading to this level.

8. Registrar vs MOSS [medical officer special scale] assessment

Most MOSS’s are far more experienced than most registrars at assessment of chest pain and I absolutely disagree with Dr O’Meeghan’s view. I have little to dispute with [Dr B’s] assessment of this case acutely and this is supported by Dr Kingsley Logan.

9. Guidelines

[The rural hospital] Guidelines are exactly in keeping with most hospitals in NZ in 2004 and even these cannot be seriously criticised today as many hospitals still use this standard. They were followed explicitly by [Dr B] on the day in question.

10. Cardiologist opinion

Seeking a cardiologist opinion is only valid if you have a patient in hand. It is not particularly relevant if you cannot contact the patient. Therefore I am unable to criticise [Dr B] for not doing this.

11. Angiography

Acute angiography is extremely difficult to obtain. Even in [the first public hospital] it is difficult and this case would not have a chance of receiving this service from [the rural hospital]. Again without the diagnosis and the patient it would be impossible. You cannot validly criticize [Dr B] for not trying.

12. Other drugs

Reference by Dr O'Meeghan to 'Statins' and 'B-Blockers' are not relevant in this case as the diagnosis of non STEMI was not made until the following day without a patient to give medicine to.

13. Attempt at patient contact

From my understanding of the case [Dr B] or the nurse did try to contact the patient the following day. If unsuccessful it is not unreasonable to try the GP who has a better understanding of the patient's normal activities and has a better relationship with the patient to get an appropriate response. The GP is also an experienced rural GP and if he was willing to take on this task he becomes fully responsible. It is not unreasonable of [Dr B] to leave it with such an experienced person who would well know the consequences of the diagnosis and to prove this the GP was so confident of the cause of death not to request a coroner's inquest.

...

Opinion

I cannot find fault with [Dr B's] acute management in that he followed a guideline which was appropriate [at the time]. His initial interpretation of the elevated troponin I was erroneous but he did discuss it with a colleague the next morning and came to the correct conclusion. If he did attempt to contact the patient himself or via the nurse that was appropriate and then more than reasonable to pass on the task to the patient's General Practitioner for the reasons given above and we have done exactly the same in near identical circumstances. I agree leaving only a recorded message in this situation is inadequate. Lack of documentation is unacceptable. I cannot see any vicarious liability in [the rural hospital's] service in this case.

The HDC's comment that [the rural hospital] may not be suitable to assess chest pain does not take into account the needs of the people in the area or in fact the needs of all rural people in NZ. ... [P]atients will simply not travel or have to take an extra 45 minute trip and then wait hours for assessment or more likely instead die at home in much larger numbers. Therefore stopping [the rural hospital] from assessing patients with chest pain is not a reasonable option from the patient's perspective. Advancing [the rural hospital's] ability is possible through obtaining a central monitoring system.

I do not see the laboratory service offering or being able to offer quantitative cardiac enzyme testing on a 7 day urgent basis in the near future. The next generation of POC testing may be more sensitive. So until then [the rural hospital] needs to amend its guideline for POC troponin T testing to at least 8 hours post onset of chest pain.

Achieving a visiting Cardiologist for [the rural hospital] again is unlikely given the shortage of Cardiologists in the country especially [this region].”

Mr J made the following submissions on behalf of the rural hospital:

“Date of Incident

The incident occurred [some time ago]. This timing needs to be considered along with the recommendations of Dr O’Meeghan.

- I have spoken to [the second public hospital] through their Quality Facilitator and secured a copy of the ‘Acute Management of Chest Pain and Acute Coronary Syndrome Procedure’.³ It was noted that this document includes a risk stratification of presenting patients. It was also noted the document was drafted and put into effect in July 2005 ... Upon cursory inspection, if [the rural hospital] had used this risk stratification in [Mr A’s] case, he would have been considered low risk and could have been discharged earlier from ED (without hospital admission) with a follow up appointment for stress testing. Only upon receipt of the final TNI would he have been raised to intermediate risk [level] requiring hospital admission. It would appear that management of [Mr A’s] case per our protocol [at the time] yielded a more conservative approach (hospital admission) than what he might currently receive at the secondary centre.
- I have asked for the same information from [the first public hospital] but ... have been unable to secure it. However by reference to items in [Dr L’s] letter, I believe the same timing of drafting procedures would be found.
- [The rural hospital at the time] appears to have been ahead of the curve by having a chest pain protocol.
- We believe it is unfair to be judged in hindsight on 2005 recommendations for incidents occurring [in ...], especially when it appears we were more advanced with written protocols and they (the protocols) were more conservative than at least the secondary hospitals in our DHB area are now.

³ See Appendix B.

Variety of Opinions

[Dr L] points out that even in the specialist ranks there is quite a difference of opinions on clinical and treatment issues. He also rightly notes that one specialist opinion must be tempered with what is happening on the whole in the particular region being investigated.

- We note there is disagreement within opinions sourced for this review.
- We note on [Dr L's] letter that there is disagreement on the use of and effectiveness of stress testing.
- We note considerable disagreement on testing and testing procedures — specifically with labs.

Laboratory Testing

There seems to be a major issue regarding the question of where the troponin testing is done and its availability.

- As a rural hospital, we are simply not in the position to provide round the clock access to a number of tests including quantitative troponin testing. As such, this must be handled in policy and procedure on whether we can admit the patient or must send them through to base hospital where the testing may be more readily available.
- [Dr L] also correctly points out hospital labs normally will [do] batch testing as well — indicating that even at base centres results may not be immediately available.
- In response to [the medical laboratory] comments on availability — I'm sure that if we wanted them to do a stat test for us on evenings, weekends and holidays, they probably do have people in the major city areas on duty to handle it. The problem then becomes logistics to any area ([the first city or the second city]) — how do we get a sample there for testing? There are no couriers, no taxis, no bus service etc., to make it happen.
- In the new Lab proposal that the combined [first and second public hospitals] are planning, one lab will do the testing for both DHB areas. We have been told that the current set up we have will remain in place under this new contracting initiative. Therefore, even with Secondary and Tertiary DHBs organising the laboratory testing for us from some point in the future, no plans exist to change the basic structure which exists for [the rural hospital] or any other Rural hospitals in [the surrounding] areas.
- We believe that the issue of lab result location, especially [at the time of these events], is an unfair criticism at this point.

Without belabouring the points much further, [the rural hospital] does not believe that the future adverse comments are required. We have shown a willingness to accept new information and put that into effect on a rapid basis as evidenced by our provisional comments.

[The rural hospital] does fully realise that no medical protocols can remain stagnant and there is always room for improvement. I am wondering, as a practical solution to this issue, if you would be willing to consider the following in lieu of further adverse comment. [The rural hospital] proposes to undertake a project to review its protocol for Acute Management of Chest Pain with a view towards improvement of current practices. I have attached a project brief⁴ which has been developed for this project. I think you will find that, while some disagreement still remains about clinical treatment, the brief seeks to determine what will be best for the local area taking into account the community needs, best medical practice and development of protocols that are consistent with and approved by local physicians/cardiologists.”

Follow-up cardiology advice

Dr O’Meeghan provided a follow-up report on 2 March 2006:

“In response to comments by [Mr J]:

Firstly I fully support the further review of the systems of care around patients presenting with acute chest pain. It is critical there is involvement of [the first public hospital] cardiology service.

I do not agree that [Mr A] would have been classified as low risk and discharged.

He had greater than 20 minutes of chest pain.

He had evolving T-wave changes on his ECG.

The laboratory troponin-1 was elevated.

I would also note that the reference range for the troponin 1 used in [the second public hospital] may be different from the troponin 1 used by the laboratory in [the first city]. It is not clear from [the second public hospital] protocol which assay system is used and therefore comparison of the results of the troponin 1 levels between the two assay systems may not be valid.

I therefore do not agree with the view that a secondary or tertiary Hospital would have discharged [Mr A] as either of the first two factors alone would have put him in a group that would require admission.

In response to comments by [Dr L]:

[Dr L] acknowledges the greater sensitivity of the laboratory test. It has been the practice in [rural hospitals] and other Hospitals to support the use of a point of care test with a confirmatory laboratory test at some point. This is also recommended by [the

⁴ See Appendix C.

chemical pathologist] in his recommendations on point of care testing. I would recommend this practice continues. To further emphasise this point the diagnosis of an acute coronary syndrome was not suspected in [Mr A] until the receipt of the laboratory troponin.

I acknowledge the logistical difficulties in obtaining troponin results and the fact that laboratory organisation is under review but I would recommend regular discussions with providers of laboratory services on how troponin results can be provided in the most timely fashion.

The time frame recommended for repeat troponin analysis following the onset of symptoms, as I indicated in my initial report, will be a critical factor for any guideline developed for the assessment of acute chest pain. My comments as to the time frame were not necessarily definitive for any individual patient or for guideline, however it would be potentially confusing for medical officers to have a guideline recommending one time frame and the laboratory report recommending a different time frame without some explanatory comment in the guideline. I should also point out that the non-ST elevation acute coronary syndromes NZ Management Guidelines, NZ Medical Journal Volume 118, 7 October 2005 recommend repeat troponin at 6–8 hours and also note that some patients will develop elevated troponins up to 12 hours after symptoms onset. To further clarify this the guidelines recommend:

‘The clinical history, examination findings, electrocardiographic changes, and blood levels of cardiac marker and troponins are all critical factors in determining risk. Risk assessment should be considered a dynamic process and patients should be assessed when first seen, after several hours, 6–8 hours, 24 hours and prior to discharge.’

Obviously the extent of evaluation of any individual patient will take into account a number of factors and clearly this is a clinical decision. In my comments about timing of troponins, I just simply wanted to point out that not all patients will have completed their assessment at 6 hours.

Development of the guidelines will include recommendations on the timing of troponin levels [and] will need to take into account the fact that:

- the point of care assay is less sensitive, (but the capacity to identify patients’ abnormal troponin levels may be improved if sampling is delayed to allow levels to increase into the detectable range);
- there will be delays in obtaining laboratory troponin results;
- the capacity to easily repeat laboratory troponins is limited.

Additionally the skill of the medical officers in evaluating a patient’s history and reading ECGs will not necessarily be at the same level as that of a consultant physician or cardiologist and hence the troponin level will have greater emphasis in this setting. All of these factors would tend to suggest a more conservative approach to the timing of the troponin sampling in this particular setting.

I indicated in my previous submission that treadmill testing does form part of the assessment of some patients presenting with acute chest pain; and would not normally in the first instance be part of the acute assessment of patients presenting with an acute coronary syndrome. I readily acknowledge the logistical difficulties here but simply emphasise the need to outline how these tests might reasonably be available to patients, obviously with some delay. I was not necessarily recommending that they be available in [the rural hospital] itself. The comment in my previous submission in respect of [Mr A] having an inpatient evaluation and exercise testing with a specific issue in relation to his particular occupation. I had previously outlined the usual treatment of heparin, aspirin, beta blockers, statins with subsequent consideration of angiography and revascularisation; depending on progress and the treatment strategy, an exercise test and the timing of this would have been a particular consideration in view of his physical occupation and the quite possible need to drive heavy vehicles (LTSA requirements are for a satisfactory stress test before resuming driving with special licences).

In response to point 8, I indicated in my submission that assessments of patients presenting with chest pain admitted to a medical ward would normally be undertaken by a consultant physician or cardiologist. Obviously registrars do participate in this process and in many cases will see the patients in advance, on behalf of the consultant physician with subsequent review by the consultant. Medical officers, who are carrying out these tasks, without the training or experience of a specialist consultant, will need to have appropriate guidelines and educational support in recognition of this.

I do not agree with point 10 that it is impossible to discuss a patient when they are not at hand. [Dr K], a colleague of [Dr B], was able to discuss the patient with him the following day and the correct diagnosis was reached without the patient at hand.

In regard to point 11, I do not agree that angiography is always extremely difficult to obtain. The consultant process in developing the guidelines will clarify the access to acute angiography at [the first public hospital].

In response to point 12, 'other drugs' I was simply outlining the usual initial medical management for patients with acute coronary syndromes.

Overall I believe it is quite possible for [the rural hospital] to come up with an improved system of care for these patients with further input."

Code of Health and Disability Services Consumers' Rights

The following Rights in the Code of Health and Disability Services Consumers' Rights are applicable to this complaint:

RIGHT 4

Right to Services of an Appropriate Standard

- 1) *Every consumer has the right to have services provided with reasonable care and skill.*
- 2) *Every consumer has the right to have services provided that comply with legal, professional, ethical, and other relevant standards.*
- ...
- 5) *Every consumer has the right to co-operation among providers to ensure quality and continuity of services.*

RIGHT 6

Right to be Fully Informed

- 1) *Every consumer has the right to the information that a reasonable consumer, in that consumer's circumstances, would expect to receive, including —*
 - a) *An explanation of his or her condition; ...*

Opinion: No Breach — Dr B

Hospital admission

On Day 1, Mr A was admitted to the rural hospital for observation, following a bout of severe chest pain. Dr B reviewed Mr A at 8.50am on Day 2. Mr A had slept soundly through the night and had reported no further discomfort. His test results (including a chest X-ray, ECG and cardiac enzymes) were reported as normal. Dr B ordered further ECG and cardiac enzyme testing (troponin T and troponin I) with a view to discharging Mr A if the results were satisfactory. At approximately 10am, Dr B reviewed the test results (excluding Mr A's troponin I test, which was couriered for reporting that evening). Dr B told Mr A that he probably had gastro-oesophageal reflux disease (GORD) and that he could be discharged.

Dr B stated:

“His cardiac enzymes the previous night and that morning were normal and his ECG was essentially normal, having only an inverted T-wave in standard lead three and lead AVF, which is an equivocal finding. As he had a history of previous dyspepsia, a single episode of chest pain and no abnormal results, he was discharged with no new medication. He was strongly advised to give up smoking immediately.”

Mr A left the hospital late in the morning of Day 2, without waiting for his discharge summary.

Electrocardiogram interpretation

My physician advisor, Dr Kingsley Logan, reviewed Mr A’s ECG recordings and considered that there was no sign of an obvious myocardial infarction. Dr Logan emphasised that Dr B could not have interpreted Mr A’s ECG results as being predictive of sudden death.

However, Dr Logan considered that there were subtle ECG changes and the correct interpretation in this context was ischaemic heart disease (IHD). Dr Logan emphasised that although an ECG is an invaluable tool it rarely permits the establishment of a specific diagnosis. ECG interpretation is to be seen in the context of the patient’s presentation and any other relevant tests. Dr Logan stated:

“T-wave inversion can be non-specific, but in this particular case with the involvement of anatomical contiguous leads, I believe the correct diagnosis should have been ischaemic heart disease.

[Dr B] should have sought [a] confirmatory result and advice from a cardiologist concerning [Mr A’s] condition once he considered the presentation was one of an acute coronary syndrome.”

Emergency medicine physicians Drs Swain and Pearson, who advised ACC, considered that Mr A’s ECG results were misinterpreted by Dr B.

Dr Pearson stated:

“His [Mr A’s] electrocardiograms revealed ischaemic changes affecting the inferior part of the heart and these changes increased and became more extensive in the sequence of electrocardiograms recorded.”

In response to the provisional opinion, Dr B submitted that it is unreasonable to expect him to have diagnosed IHD on the basis of Mr A’s very subtle ECG readings and noted that there was no indication from the very sensitive ECG software analysis programme of an abnormal result.

Dr Logan was asked to provide further comment and agreed that, in light of his training and experience, Dr B (and many other doctors) would have had difficulty in recognising Mr A's subtle ECG changes as being diagnostic of an acute coronary syndrome. He stated:

“It remains my opinion that the ECG changes were subtle and could be overlooked by a non-specialist.”

My cardiology advisor, Dr Tim O'Meehan, agreed that the inferior T-wave inversion, later identified on Mr A's ECG recordings, was quite subtle.

Troponin tests

Dr B discharged Mr A following the receipt of a second negative “spot” troponin T blood test result for Mr A (at approximately 10am). A further troponin I test was due to be reported later that evening.

As noted above, Dr Logan explained that the ECG on its own is rarely diagnostic of IHD and needs to be considered in conjunction with the nature of the chest pain, together with serial blood tests. He stated:

“These classically have involved measurement of cardiac enzymes and for many years the CPK [creatinine phosphokinase, the former name for creatine kinase] was regarded as the gold standard and is still used to judge infarct size. It is invariably raised when there has been significant myocardial damage.”

Dr Logan commented that although troponin testing is relatively new, it is now regarded as being the best marker of cardiac damage:

“The troponin T spot test is highly regarded if this is negative, when the test is in the indeterminate range confirmation is required using an analyser and depending on the regional laboratory troponin T or troponin I. The troponin T has increasingly been accepted as the standard by cardiologists in NZ. There are many centres that would not request a confirmatory test when the screening spot troponin T is negative.”

ACC expert advisors Dr Swain and Dr Pearson considered that of the tests available, the laboratory troponin I was the preferred option for confirming whether damage to the heart had occurred.

Dr O'Meehan commented that the troponin T point-of-care qualitative test is not particularly sensitive at low levels, and therefore is not a reliable “rule-out” test. Dr O'Meehan considered that Dr B would not necessarily have been aware of the limitations of this test, and laboratories do not provide any explanatory comment with the test results. Dr O'Meehan stated:

“The troponin T point-of-care qualitative test is really only useful as a rule-in test, the advantage being that if positive, decisions can be made about patients with a significantly raised troponin more promptly, particularly if there are significant delays in obtaining a definitive laboratory troponin result.”

In his submission in support of the rural hospital, Dr L acknowledged that the laboratory test is recognised to be more sensitive, but disputed that that a negative spot test requires confirmation.

Discharge

Dr B submitted that he took reasonable steps to exclude a cardiac cause for Mr A's chest pain, prior to the decision to discharge him. Dr B emphasised that the reason for Mr A's hospital admission was to exclude a possible cardiac cause for his chest pain and he did not discharge Mr A until he was satisfied that an IHD diagnosis was unlikely. He stated:

“In the face of a normal ECG (in my opinion at that time), normal cardiac enzymes (up to that time), a single non-recurrent episode of chest pain and only one known risk factor (smoking), an urgent cardiology appointment at this time would have been inappropriate, as would preventative therapy (barring the suggestion to stop smoking, which was given).”

(Dr B also explained that an outpatient appointment for a formal exercise test was ordered, as a matter of routine, but cancelled when Mr A died. The rural hospital could find no record of the appointment.)

Dr Swain considered that Mr A should not have been discharged and that the rural hospital chest pain protocol was breached. In contrast, Dr Logan commented that Dr B's decision to discharge Mr A was appropriate and the chest pain protocol was only breached following Mr A's discharge (see discussion below). He stated:

“Having considered the blood tests available and the diagnosis of simple reflux with what was considered normal ECG and troponins at discharge, his management was appropriate.

...

Yes it was appropriate for [Dr B] to discharge [Mr A] following further ECG and 'spot' cardiac enzyme testing. He had more than spot cardiac testing, there are 3 CPK tests in the notes and whilst it can take 8 hours for the CPK to become positive none of these were positive and it is important to note that the CPK take at the time of the troponin I was again low. The classic cardiac enzymes were normal.”

Dr O'Meeghan commented that Dr B was in a “difficult situation”, with a patient who appeared to be “very keen” for an early discharge, and having to make a decision without all the results being available.

Dr Logan noted that, even if Mr A had been diagnosed with IHD, he may have been managed as an outpatient. However, if the diagnosis had been made, Mr A should have received preventative therapy and, ideally, risk assessment and stratification prior to discharge. He stated:

“There is always a differential diagnosis in patients who present with chest pain and whilst it was considered that [Mr A] had reflux, [Dr B] should have considered that [Mr A] may have had ischaemic heart disease. Indeed all differentials of chest pain must consider ischaemic heart disease. Current practice would dictate that formal risk factor assessment should be done in all patients who present with chest pain. ... Beta blockers are usually given to patients who are felt to have evidence of overt ischaemic heart disease and, again, in this setting these would normally be prescribed.

...

Risk factor assessment intervention and risk stratification either by angiography or at the least by exercise testing should be done in patients who present with chest pain thought to be cardiac. Ideally this is done prior to hospital discharge [for patients] who are troponin negative.”

Dr Logan also commented that smoking and age are important risk factors and that a diagnosis of ischaemic heart disease should always be considered in patients of Mr A’s age group who are smokers. He stated:

“The many risk factors present in patients with ischaemic heart disease do not result in the electrical event of so called sudden death. To date we really do not have any predictive model. We know, however, that patients who present with a non ST elevation MI — and that ultimately was the presentation [Mr A] had on this occasion — are advantaged by an early invasive strategy.”

Conclusion

Mr A was admitted in accordance with the rural hospital chest pain protocol for the purpose of ascertaining whether there was a cardiac source for his severe chest pain episode. He received examination and review, ECG testing and cardiac enzyme testing, which were not considered to be indicative of a cardiac diagnosis during his admission to the rural hospital. He did not receive any preventative therapy or formal risk assessment for his condition prior to his discharge.

Dr Logan has explained that, with the benefit of hindsight, Mr A’s ECG recordings were indicative of IHD. Dr O’Meeghan commented that the inferior T-wave inversion (detected later) was subtle. Dr Logan considered that, without specialist training, Dr B would probably not have been expected to have diagnosed IHD (although medical officer Dr K was able to interpret Mr A’s ECG recordings correctly the following morning). I also note that Dr Logan considered that the ECG recordings could not have been interpreted as predictive of Mr A’s subsequent death.

There is a discrepancy of opinion between Drs Logan and O’Meeghan (and emergency physicians Drs Swain and Pearson) about which type of troponin test provides the most reliable result. I accept that the troponin T ‘spot’ (point-of-care) test can be very useful in guiding initial care, and in many cases will provide some basis for making reasonable clinical judgements, particularly where there is no laboratory troponin T or I test immediately available. However, the troponin T or I laboratory test is generally regarded

as more sensitive. I conclude that it was not, in itself, an error of judgement for Dr B to rely upon the cardiac enzyme and troponin T test results (notwithstanding his request for confirmatory laboratory tests), particularly in light of his view that Mr A's ECG recordings were satisfactory. Mr A also appeared to be well, with no further report of chest pain, and was keen to return home.

I acknowledge that preventative therapy and risk stratification may have been to Mr A's advantage, although it cannot be known whether these measures would have prevented his death.

Overall, I consider that as a medical officer at a rural hospital Dr B provided satisfactory care to Mr A during his admission and is not culpable for his failure to interpret the ECG results as indicative of ischaemic heart disease. In these circumstances Dr B did not breach Right 4(1) of the Code.

Opinion: Breach — Dr B

Circumstances of discharge/ information provided

Mrs A states that Mr A left the hospital "at about 11am with the reassurance he had a Hiatus Hernia/GORD and there was nothing wrong with his heart". Dr B disputes that he gave Mr A the impression that his chest pain was conclusively non-cardiac. However, it is agreed that Mr A was not provided with a written discharge summary when he left the hospital (contrary to the rural hospital medical manual discharge policy, at the time) and was not informed of his outstanding troponin I test result.

Dr B explained that he saw Mr A briefly to inform him of his normal results and that he could go home. Dr B stated that Mr A was keen to return home, and elected to leave hospital without a full explanation of his condition. Ms I confirmed that Mr A left immediately after being informed of his test results, before his discharge summary was available.

Dr B submitted that his omission to inform Mr A of the outstanding troponin test was not particularly significant as he would have told Mr A that it was expected to be negative. He stated:

"[Mr A] was told that I thought he probably had GORD. I stressed to him that he should give up smoking. I also stressed that any further chest pain should be investigated and that he should see his GP for this unless he was acutely unwell."

My physician advisor commented that Mr A should have been informed of his outstanding blood test result and arrangements made to discuss it. He stated:

"[Mr A] did not have a clear understanding of the consequences of further chest pain, given that he did not contact an ambulance service, the local hospital or his own doctor

when he had a further recurrence of pain. Clearly this would have been affected by direct instruction interaction with the patient both verbal and written.

...

Advice was given at discharge that was appropriate to the presumed diagnosis of chest pain as a result of reflux. It is very clear that [Mr A] did not have an understanding of the consequences of further chest pain given that he did not contact an ambulance service / the local hospital or his own doctor when he had a further occurrence of pain.”

I am not able to determine precisely what information Dr B provided to Mr A concerning any further chest pain prior to his discharge, as there is no supporting documentation. However, it is clear that the discussion between Dr B and Mr A was relatively brief, and that Mr A left shortly after he was informed that he was to be discharged. It appears that Mr A was under the impression that his pain had been non-cardiac in nature — he returned to his farm work when the weather had cleared. In addition, his action in not immediately seeking medical assistance when his pain recurred suggests that he may not have been fully aware of the risks associated with further chest pain.

Mr A presented with sufficient risk factors to be advised that any recurrence of chest pain should be treated very seriously indeed. I find it doubtful that Dr B so advised him. It is also important to inform patients of any outstanding test results, even if the results are expected to be reassuring. Clearer instructions — including advice that one laboratory test result was still awaited — may have prompted Mr A to contact an ambulance immediately when he experienced the onset of a further episode of chest pain.

In my opinion Dr B breached Right 6(1)(a) of the Code by not providing clear instructions on discharge to Mr A about the risks associated with further chest pain, and not informing him that one laboratory test result was still awaited.

Actions following receipt of abnormal test result

At approximately 7.30pm on Day 2, Dr B was informed by nursing staff of Mr A’s troponin I test result of 0.5. Dr B took no immediate action as he considered the result indicated a marginal increase and was not significant. Dr B commented that at that time, the significance of an elevated troponin level was not fully known.

Dr Logan advised that it was reasonable for Dr B to consider that Mr A’s troponin I result was not significant. He stated:

“The troponin I in this situation can be difficult to interpret. There is a range of false positives that can occur with any test and it had been the experience of many institutions to regard a level of 0.5 as being indeterminate.

...

Given that [Dr B] considered that [Mr A] did not have a diagnostic ECG it was not unreasonable to initially discount the isolated troponin I that became available on the evening of [Day 2].”

Dr Logan commented that when viewed in the context of two hours of chest pain and subtle ECG changes, the troponin I result becomes more important. However, at the time the result was received, Mr A was thought to have a normal ECG.

Dr O’Meeghan noted that the second troponin test was significantly elevated, and was over twice the upper limit of a normal level. Dr B did not initially “attach too much importance” to it and demonstrated a limited appreciation of the appropriate response to an elevated troponin I result. Dr L also noted that Dr B’s initial interpretation of the troponin result was erroneous.

Drs Swain and Pearson considered that Mr A’s troponin I test result was significant, consistent with myocardial tissue injury (particularly when viewed in the context of his ECG readings and chest pain), and an indication that immediate action by Dr B was warranted.

Review of Mr A’s results, Day 3

Mr A’s results were reviewed the following morning by medical officer Dr K, who decided to review his serial ECG recordings. She formed the view that the “very subtle inferior T-wave changes” were cardiac in nature. This was discussed with Dr B, who decided to contact Mr A’s GP, Dr C, and ask him to arrange follow-up. Dr K noted the change in diagnosis on the previously completed discharge report (which was faxed to Dr C that evening together with the troponin I test result).

Dr Logan considered that an indeterminate troponin test in the context of Mr A’s presentation (including the subtle ECG changes) was significant, and an indication that Mr A might have ischaemic heart disease. Dr Logan stated that even with the benefit of hindsight Mr A’s presentation placed him at a low risk of sudden death. However, following the diagnosis of ischaemic heart disease, Mr A would have benefited from timely follow-up and preventative treatment. Dr B should have sought confirmatory results and advice from a cardiologist concerning Mr A’s condition once he considered that the presentation was an acute coronary syndrome. Dr Logan stated:

“The issue is then one of responsibility of care and as this was handed back to the general practitioner, whether all of the relevant information was also handed over [to] the general practitioner.

Even with the reflection of the ECG and troponin I result, [Dr B’s] opinion seemed to favour that of outpatient investigation and assessment of ischaemic heart disease rather than management of the acute coronary syndrome and indeed is reflected by the fact that the general practitioner delegated responsibility to his clerical staff to contact [Mr A] and arrange an out-patient appointment. There is no formal record of this interaction.”

Drs Swain and Pearson considered that the transfer of responsibility to the general practitioner for follow-up treatment was inadequate. The combination of results warranted immediate action including readmission and specialist intervention by a cardiologist. Dr Pearson stated:

“I believe it is inadvisable and inappropriate *in this situation* to delegate the responsibility of contacting [Mr A] to his GP. This is because [Dr B] had all the relevant information, was familiar with the patient and was familiar with the urgency that the situation required.”

In response to the provisional opinion, Dr B explained that the decision to refer Mr A to general practitioner care was an effective way of arranging follow-up in a rural hospital context and was a common occurrence. His colleague, Dr E, also submitted that there was no reason why Mr A should not be followed up by his GP.

Dr Logan accepted that it was “very appropriate that Mr A was referred back to his own general practitioner”. However, he reiterated that Dr B had a responsibility for ensuring that Dr C had all the relevant information about Mr A’s condition. He stated:

“This does of course presume that all relevant information, that is relevant ECGs, hospital notes and blood results are made available to the general practitioner.

...

There is little question of the responsibility that lies with [Dr B] as the medical officer discharging the patient and that this responsibility is completed when all relevant medical information is transferred or handed onto the patient’s general practitioner.

There does not seem to have been any sense of urgency transferred to [Dr C] given the phone message left by his receptionist ... [Mr A’s] presentation was seen as being relatively low risk, and indeed was continued to be managed in this way.”

Dr O’Meeghan considered that the failure to seek cardiology advice and Dr B’s decision to delegate follow-up arrangements to the general practitioner was not appropriate. He stated:

“In previous submissions, it is stated that it is quite common in the rural setting to delegate the responsibility to the General Practitioner for a variety of follow-up issues. This may be appropriate for many conditions. In this particular situation however, given the acute nature and significance of the problem [Dr B] should have sought advice from a cardiologist (as required by the protocols) and communicated directly (or at least attempted to) with [Mr A]. Any delegation of these tasks inevitably results in delays, a loss of emphasis and priority, which this case illustrates. The delegation to [Dr C] was an important clinical issue. This was not documented contemporaneously and there is now conflict between [Dr C] and [Dr B] over the timing, content and degree of urgency expressed during those discussions.”

Dr B claimed that he recommended urgent cardiology follow-up, with the commencement of immediate preventative therapy (aspirin and beta-blocker). Dr B stated that he phoned Dr C at approximately 11.30am on Day 3 and this has been confirmed by Dr C's colleague. (Dr C incorrectly recalled that he was phoned at 4pm, shortly after he instructed his practice nurse Ms D to contact Mr A.) Dr B stated that Dr C agreed to follow his instructions and "presumed he [Dr C] would contact Mr A immediately".

In contrast, Dr C disputes that urgent follow-up was recommended. Neither Dr B nor Dr C documented the telephone discussion and, accordingly, I am unable to determine precisely what information Dr B provided to Dr C (see *Record-keeping* below).

(Dr L submitted that he understood Dr B attempted to contact the patient directly, or via the GP's practice nurse, before passing on responsibility to the GP. However, there is no supporting evidence that this occurred.)

Dr Logan and Dr O'Meehan both consider that a medical officer in Dr B's rural hospital setting should have sought cardiology opinion after the review of Mr A's test results and the change to a cardiac diagnosis. In Dr Logan's view, delegation to the GP was only acceptable if appropriate information had been provided to the GP, conveying a sense of urgency.

In my view, Dr B acted unwisely in transferring Mr A's care to his general practitioner, without first seeking the input of a cardiologist. I do not share Dr L's view that seeking cardiology opinion is "only valid" if the patient is "in hand". Dr B was in possession of a significant amount of clinical data about Mr A's condition, and was well placed to discuss his management with a specialist.

In any event, Dr B clearly had a responsibility to ensure that the GP knew that urgent follow-up was required, on that day. Dr C did not advise his practice nurse to contact Mr A until approximately four hours after his discussion with Dr B. The telephone message for Mr A (left by Ms D at 4.40pm) did not convey any sense of urgency about Mr A's condition. Dr C's actions suggest that he was not under the impression (from Dr B) that Mr A required urgent follow-up.

In addition, Dr C did not receive the faxed information about Mr A's reviewed diagnosis until approximately 6pm that evening. This was significant clinical information that should have been provided to him earlier. Dr C was therefore totally reliant on his conversation with Dr B to make a judgement concerning the urgency of follow-up.

I accept that Mr A would not necessarily have been re-admitted to hospital and may have been managed as an outpatient. However, he may well have benefited from the immediate institution of preventative therapy, and reduction in physical activity pending further evaluation.

In these circumstances Dr B breached Rights 4(1) and 4(5) of the Code by delegating follow-up management to Mr A's GP without (a) first seeking cardiology opinion; (b) promptly faxing updated discharge information, including the abnormal test results; and (c)

stressing the urgency of the situation and the need for immediate steps by Mr A. Dr B, by his omissions, did not treat Mr A with reasonable care and skill, and did not co-ordinate his care effectively with his GP.

Record-keeping

Any significant clinical decision made in relation to a patient's care must be clearly documented, to ensure health professionals involved in treatment can ascertain what care has been provided and ideally what care was considered. The Medical Council of New Zealand (the Council) issued the following statement on the 'Maintenance and retention of patient records' in August 2001:

“1. Maintaining patient records

- a) Records must be legible and should contain all information that is relevant to the patient's care.
- b) Information should be accurate and updated at each consultation. Patient records are essential to guide future management, and invaluable in the uncommon occasions when the outcome is unsatisfactory.”

This statement from the Council is a relevant standard for the purposes of Right 4(2), which states that every patient has the right to services that comply with legal, professional, ethical, and other relevant standards.

Dr B did not specifically document Mr A's discharge details in the medical records, following his prior written instruction to nursing staff that Mr A could be discharged if his further tests were returned normal. The rural hospital policy authorised discharge of patients only on the written order of a medical officer. Dr B explained that writing “discharge if” in the medical records was normal practice.

In my view, the record of discharge was unclear and did not contain all the relevant information, including the instructions provided to Mr A and the fact that the troponin I test result was awaited. I note that the proposed new rural hospital policy requires that discharge instructions are documented in the patients' medical records, including any advice on who to contact in the event of recurrence of chest pain.

In addition, Dr B made no record of his telephone conversation with Dr C apart from a note on Mr A's discharge summary stating “phoned Dr C”. My physician advisor commented that a written record of this discussion should have been made. Dr Logan stated:

“All communication regarding patients should summarise the situation, as the clinician perceives it and provide advice as to what should happen if circumstances change. A written record of this communication should follow this communication.”

Dr B should have taken particular care to fully document his telephone conversation with Dr C, in light of the importance of the information conveyed.

Dr B accepts that his documentation in relation to Mr A, in particular with respect to the conversation with Dr C, was inadequate. In these circumstances, Dr B breached Right 4(2) of the Code.

Opinion: No Breach — The Rural Hospital

Under section 72(2) of the Health and Disability Commissioner Act 1994 (the Act) employers may be vicariously liable for any breach of the Code by their employees. Employers are responsible for ensuring that employees comply with the Code of Health and Disability Services Consumers' Rights. Under section 72(5) it is a defence for an employer to prove that it took such steps as were reasonably practicable to prevent the employee from breaching the Code.

As noted above, Dr B breached Rights 4(1), 4(2), 4(5) and 6(1)(a) of the Code. I have considered whether Dr B's employer, the rural hospital, is vicariously liable for his breaches of the Code.

Circumstances of discharge/ information

Dr B breached Right 6(1)(a) of the Code by not providing sufficient information to Mr A about his condition prior to discharge. In particular, he did not ensure that Mr A fully understood the consequences of any further chest pain and did not tell him that a test result was awaited. In my view, this is an error in judgement for which Dr B is solely accountable.

Follow-up of abnormal results

Dr B breached Rights 4(1) and 4(5) of the Code by delegating Mr A's follow-up management to his GP, without seeking cardiology advice and without providing sufficient information and advice. In my view, these omissions are also a matter of individual error in judgement, for which Dr B is solely accountable.

Record-keeping

Dr B breached Right 4(2) of the Code by not adequately documenting Mr A's discharge and his telephone conversation with general practitioner Dr C. I consider Dr B's failure to adequately document aspects of Mr A's care to be an individual failing.

Conclusion

Dr O'Meehan has made a number of criticisms and suggestions for improvement in relation to the rural hospital systems (see *Other comments* below). Nevertheless, I consider that the errors by Dr B were primarily lapses in clinical judgement, rather than limitations in the hospital systems.

Accordingly, the rural hospital is not vicariously liable for Dr B's breaches of the Code.

Other comments

Troponin testing

As previously noted, Dr O’Meeghan considered that Dr B probably did not appreciate the limited sensitivity of the troponin T point-of-care test at low levels. He stated:

“It has been noted that two serial troponin T point-of-care qualitative tests were negative along with the first laboratory troponin I but the second laboratory troponin I result was abnormal. The explanation for the difference between the second troponin T point-of-care qualitative test being negative and the second troponin I test being positive lies in the fact that the troponin T point-of-care qualitative test is relatively insensitive at low levels of troponin elevation. These troponin elevations are important and the troponin T point-of-care qualitative test is simply not sensitive enough to detect these troponin elevations reliably. In that situation the troponin T point-of-care qualitative test cannot be used as a reliable rule-out test. This is a very important difference between the two assays. The troponin T point-of-care qualitative test is really only useful as a rule-in test, the advantage being that if positive, decisions can be made about patients with a significantly raised troponin more promptly, particularly if there are significant delays in obtaining a definitive laboratory troponin result.”

Dr L (and my physician advisor Dr Logan) did not consider that the point-of-care troponin tests necessarily required confirmation. However, Dr L agreed that the laboratory test was more sensitive.

Dr O’Meeghan considered that preferred troponin test is the laboratory troponin T test (not the troponin T point-of-care test), as there is only one reference range, and this minimised the confusion caused by the various different assay systems. However, Dr O’Meeghan considered it appropriate to use the laboratory troponin I test, and that it provided an accurate and acceptable result in the case of Mr A.

The rural hospital advised that during normal hours, the troponin tests are couriered to the laboratory in the afternoon and the results are faxed to the rural hospital as soon as they become available. However, there is no provision for the processing of troponin tests after-hours. There is no proposal to change the current set-up for laboratory testing under the new combined laboratory proposal.

Dr O’Meeghan considered that one of the important areas to be improved is developing a timely method for after-hours laboratory troponin results. Dr O’Meeghan noted that the timeframe for obtaining a definitive troponin test under the rural hospital chest pain protocol was six hours, which conflicted with the laboratory advice accompanying Mr A’s troponin I result (which recommended a minimum of 12 hours). Dr O’Meeghan considered that this aspect of the chest pain protocol should be reviewed. Dr O’Meeghan also thought that it would be helpful if the rural hospital staff received information directly from the laboratory about the different troponin tests.

Dr O’Meeghan recommended that the rural hospital continue using laboratory results to confirm point-of-care testing. He accepted that there are logistical difficulties in obtaining laboratory test results but suggested that the rural hospital explore the available options to improve the timeliness of laboratory testing. In response to Dr L’s suggestion that an eight-hour timeframe for troponin testing to occur is adequate, Dr O’Meeghan stated:

“Obviously the extent of evaluation of any individual patient will take into account a number of factors and clearly this is a clinical decision. In my comments about timing of troponins, I just simply wanted to point out that not all patients will have completed their assessment at 6 hours.

Development of the guidelines will include recommendations on the timing of troponin levels [and] will need to take into account the fact that:

- the point of care assay is less sensitive, (but the capacity to identify patients’ abnormal troponin levels may be improved if sampling is delayed to allow levels to increase into the detectable range);
- there will be delays in obtaining laboratory troponin results;
- the capacity to easily repeat laboratory troponins is limited.

Additionally the skill of the medical officers in evaluating a patient’s history and reading ECGs will not necessarily be at the same level as that of a consultant physician or cardiologist and hence the troponin level will have greater emphasis in this setting. All of these factors would tend to suggest a more conservative approach to the timing of the troponin sampling in this particular setting.”

Overall, it appears that the laboratory test has greater sensitivity, particularly at low levels. It may be appropriate to seek a confirmatory laboratory result, particularly when there are other factors that may indicate possible cause for concern. I urge the rural hospital to carefully consider Dr O’Meeghan’s comments about troponin testing, and to ensure that its staff, including temporary staff, are educated and informed about troponin testing. I recommend that the rural hospital explore possible methods of improving the availability and timeliness of after-hours laboratory test results.

Education and support

Mr J explained that medical officers undertake supervision regularly. Dr O’Meeghan considered that medical officers, and nursing staff, need a structured educational programme to ensure they have appropriate training:

“It would be appropriate to have regular educational sessions for the medical officers on the assessment of acute chest pain (and for that matter a variety of other acute medical problems) and this would serve as the basis for good clinical liaison between [the rural hospital] and other specialist physicians in [the first and second public hospitals] depending on the specialist service.”

Dr O’Meeghan commented that experienced nursing staff are invaluable in the assessment of chest pain:

“Nursing observations, particularly during symptomatic episodes along with ECGs will frequently provide valuable supporting information for reaching a diagnosis and developing a management plan. Nursing staff also need to have an education programme so that they are familiar with the important aspects of chest pain assessment.”

Dr O’Meeghan emphasised the importance of providing appropriate support for medical officers undertaking chest pain assessment:

“In the vast majority of hospitals in New Zealand assessments of acute chest pain are normally undertaken by the physicians participating in a general medical roster who are frequently exposed to this problem, or in some of the larger centres cardiologists will see a significant proportion of these patients. In these larger centres the medical staff would be supported by an on-site laboratory and exercise testing facilities. Undertaking chest pain assessments in [the rural hospital] would require the medical officers to have support and be able to discuss individual patients with the appropriate specialist, in this case a cardiologist, on a case by case basis.”

The rural hospital has provided no specific information concerning the ongoing education of medical officers. It apparently occurs on an “as required” basis via the supervisory process (although I note that nursing staff receive ongoing training in cardiac arrhythmia recognition). It appears that it was not regular practice for medical officers to seek cardiology advice before making potentially significant clinical decisions about chest pain patients, even though the relevant protocols refer to physician consultation.

I acknowledge the difficulty that rural hospitals face in recruiting and retaining appropriately skilled staff. In this situation, ongoing training for medical and nursing staff is essential, together with a system to facilitate appropriate consultation with specialist physicians. Medical officers should not be placed in the position of providing acute medical care without well-established training and support mechanisms.

Resource constraints

Mr J explained that the rural hospital facilities are designed to treat, stabilise and transfer any serious acute chest pain presentations, in conjunction with specialists at secondary and tertiary centres. The rural hospital has a cardiac monitor and an ECG machine for acute emergency patients. No exercise testing or angiography is available for chest pain patients at the rural hospital.

Dr O’Meeghan noted that the rural hospital is very small, and there is no justification for the employment of specialist physicians. He stated:

“From the advice received from [the rural hospital] the monitoring facilities would appear limited. While many of the patients admitted with acute chest pain will have a

stable clinical course, a few will have significant arrhythmias and detection of these is very important in their clinical management.”

Dr L agreed that the absence of a centralised monitoring facility at the rural hospital should be addressed, and commented that most small hospitals are only now upgrading to this facility. Dr L considered that in other respects, the facilities at the rural hospital are appropriate.

Dr O’Meeghan stated that exercise testing should be available for patients prior to discharge. While he would not expect the rural hospital to offer exercise treadmill testing, he commented:

“Any review of the systems of care around chest pain assessment will need to define how patients who are identified as requesting treadmill testing can have these tests undertaken with a minimum of delay. Inevitably, given the geography of the situation, there will be a compromise in the provision of these tests. They are however, very useful and particularly important in the rural environment when many patients will often be undertaking heavy physical tasks as part of their occupation.”

(Dr L noted that exercise testing is primarily used for the diagnosis of angina rather than acute coronary syndrome. Dr O’Meeghan clarified that treadmill testing can be a helpful diagnostic tool for acute chest pain, but is not normally used in the first instance for the assessment of acute coronary syndrome.)

My physician advisor, Dr Logan, commented that if Mr A had been recognised as suffering from ischaemic heart disease, some form of risk stratification would have been appropriate:

“Risk factor assessment intervention and risk stratification either by angiography or at the least by exercise testing should be done in patients who present with chest pain thought to be cardiac. Ideally this is done prior to Hospital discharge [for patients] who are troponin negative. When the troponin is positive risk stratification by angiography prior to discharge has now become the standard. A recent [2002] survey of all NZ hospitals private and public has shown that only 39% of hospitals are able to do this.”

Dr Rothwell commented that acute angiography is “extremely difficult” to obtain. Dr O’Meeghan did not agree that this was always the case, and noted that consulting with the first public hospital will clarify the process for the rural hospital staff accessing acute angiography services at the first public hospital.

I accept that the hospital is a small rural hospital and cannot reasonably be expected to provide services such as angiography or exercise testing. However, I encourage the rural hospital to consider my expert’s comments as part of its proposed review of the management of acute chest pain. I also note that there is a consensus of opinion that the rural hospital’s monitoring equipment requires upgrading.

Chest pain protocol

Mr J explained that the rural hospital chest pain protocols were written by Dr B as part of his graduate diploma in geriatrics. He stated:

“As such we believe that he [Dr B] received guidance and comment on its preparation and applicability during his studies.”

Mr J commented that the applicable protocols were relatively conservative with regard to the threshold for admission. He argued that Mr A would have been regarded as low risk under the equivalent protocols at the second public hospital, and would not have been admitted to hospital until after the receipt of his final troponin test results. In response, Dr O’Meeghan observed that Mr A had evolving ECG T-wave changes, and had an elevated laboratory troponin level, and either of those two factors would have placed him in the category that required admission under the protocols of second public hospital.

Mr J’s submission is predicated on the hypothetical situation of a medical officer making the same judgement calls that Dr B made in applying the second public hospital protocols. This is speculative and unhelpful. I do not accept Mr J’s submission that Mr A would have been discharged if he had presented at a secondary or tertiary hospital — where it is more likely that his ECG changes would have been regarded as diagnostic and immediate action taken following receipt of an elevated Troponin I result.

Dr O’Meeghan recommended that the rural hospital seek input from the first public hospital cardiology service about the systems of care around acute chest pain assessment, and was supportive of the rural hospital’s proposal to review its protocols for the management of patients with chest pain. He stated:

“It would be a matter of some priority for [the rural hospital] to seek input into the systems of care around acute chest pain assessment from [the first public hospital] cardiology service. In particular, the chest pain protocol should be reviewed by the cardiology service. The current protocols have been written by [Dr B] but the only external input into these protocols apparently has been the supervisors of his geriatric medicine course he is undertaking [at a medical school]. I do not believe this is appropriate external review and advice for these protocols, and more specific and locally based advice would be far more appropriate.”

It is somewhat surprising that the rural hospital chest pain protocol was drafted by a medical officer (as part of Dr B’s training for a graduate diploma in geriatric medicine), without any external review or input from a cardiologist. (According to Dr B, there was actually no protocol for the management of chest pain patients who had been admitted to the rural hospital.) I am not convinced by Dr L’s submission that the protocols were adequate. They appear to lack depth and detail, particularly with regard to troponin testing. I note with concern that, according to Dr L, other small hospitals may share a similar lack of clarity in their chest pain protocols. I would expect chest pain protocols in small (rural or provincial) hospitals to be developed in consultation with a regional cardiologist, and to provide specific guidance on the sensitivity of the type of troponin test being utilised.

Overall, I consider that aspects of the rural hospital systems for the management of chest pain were not optimal. I accept that, to a degree, criticism of the rural hospital systems must be tempered by resource constraints and the variation in nationwide clinical practice in the management of chest pain patients. Nevertheless, the matters identified by Dr O’Meeghan merit careful consideration.

Ambulance records

My physician advisor Dr Logan queried whether the ambulance ECG recording had formed part of Mr A’s medical records during his admission. Dr Logan stated:

“Unfortunately the graph showing biphasic T-wave change taken in the ambulance never became part of the medical record. It is not recorded or referred to in the medical notes and is incorrectly labelled and timed.

The 2 strips show quite considerable baseline artefact and cannot be easily interpreted. These have not been referred to in any of the clinical notes either in A&E or the admission notes and one of the issues therefore is whether this was available or transferred to the admission notes and were part of the hospital record.

...

It is the practice of some hospitals to keep the A&E notes separate from the admission notes so these are not easily available. Transfer of results and documentation such as ECG tracing taken by the ambulance service needs to be formalised if these are to be regarded as part of the medical record.”

I am unable to determine whether the ECG readings taken in the ambulance were included with Mr A’s medical notes. However, I draw the rural hospital’s attention to Dr Logan’s comments. Clearly, a patient’s ambulance record, including any tests conducted by ambulance staff, must be transferred and integrated with the hospital record following admission.

Medical records

I note that the rural hospital’s proposed amendment to its “Medical Manual” requires patients to be informed of any outstanding results, and documentation of instructions given at discharge in the patient’s medical records, including who to contact if the patient has any further chest pain. These are also to be documented in the discharge letter that is given to the patient prior to discharge.

Dr Logan commented that it would be useful to establish whether staff strictly adhere to the hospital documentation discharge policy. I recommend that the rural hospital undertake an audit of current practice, and ensure that staff are complying with the new protocol.

Nursing documentation

Ms I did not complete the discharge checklist at any stage or take any action to ensure that Mr A received his discharge summary. Ms I explained that this was because Mr A left immediately after being informed of his test results and pending discharge.

The relevant rural hospital protocol stated that patients “will” receive a discharge letter (prior to discharge) and that nursing staff were required to complete a patient discharge checklist (which included the provision of a patient discharge summary).

In my view, it is important that staff complete the required discharge documentation or, at a minimum, inform or caution any patient insistent on leaving immediately that the required documentation has not yet been prepared.

Information for chest pain patients

I also note that the rural hospital does not appear to have any standard written information for chest pain patients on discharge. Dr Logan stated:

“Patient instructions for patients with chest pain are readily available and it is my opinion that this does facilitate patient understanding of the problem and compliance.”

In light of Dr Logan’s comments, I recommend that the rural hospital consider introducing an information sheet for chest pain patients on discharge.

Actions taken

The rural hospital has reviewed its chest pain discharge protocol. It has also undertaken a project to review its protocols for the management of acute chest pain, with a view towards the improvement of current practices and to deliver best possible service to the community. Mr J advised that the project has been completed and the revised protocols will be finalised and in operation shortly.

Dr B advised that all troponin test results are now discussed with a cardiologist, and patients are contacted directly on receipt of outstanding results.

Recommendations

Dr B

I recommend that Dr B take the following action:

- review his practice in light of this report
- apologise to Mrs A for his breach of the Code. The apology is to be sent to the Commissioner and will be forwarded to Mrs A.

The rural hospital

I recommend that the rural hospital complete the proposed review of its chest pain protocol, as outlined in the project brief, and report the outcomes to my Office. I also recommend that the rural hospital:

- undertake an audit of discharge documentation;
- introduce an information sheet for chest pain patients;
- review procedures for patients who leave prior to formal discharge; and
- ensure that information in a patient's ambulance record is transferred into the patient's hospital medical record.

Follow-up actions

- A copy of this report will be sent to the Medical Council of New Zealand, the Accident Compensation Corporation, and the District Health Board.
- A copy of this report, with identifying details removed, will be sent to the Royal Australasian College of Physicians, the Cardiac Society of Australia and New Zealand, the Australasian College of Emergency Medicine, the New Zealand Rural General Practice Network, and the Ministry of Health, and placed on the Health and Disability Commissioner website, www.hdc.org.nz, for educational purposes.

Addendum

A copy of the rural hospital's reviewed chest pain protocols were sent to Dr O'Meeghan for his further comment. Overall, Dr O'Meeghan considered that the proposed protocols were appropriate. However, he emphasised that the rural hospital needed to take further steps to ensure that Medical Officers are appropriately educated and supported in the provision of services to chest pain patients — by ongoing consultation with regional cardiology services to ensure rural patients receive the best possible treatment and outcomes.

Appendix A — A city region advice on troponin I point-of-care testing

“Point-of-care testing with troponin

Occasionally it may be useful to use point-of-care strips for measuring troponin T in general practices. No one should use these devices without being aware of their limitations. Users should also be aware of the protocols that accredited laboratories are required to adopt when using point-of-care testing of any type. If it is decided to opt out of these protocols the reliability of the procedures is even more limited.

The decision to use point-of-care testing should always be driven by a clinical need that cannot be met from a central laboratory. In general this means inadequate turnaround time in a situation where an early result might affect a crucial clinical decision.

Proper use of point-of-care testing involves adequate education of all users. For qualitative tests this would include having been a definite positive result. For accredited procedures there should be a register of certified users. There should be some sort of quality assurance programme. This could consist of occasional positive and negative specimens being sent from the central laboratory to the practices using the point-of-care device. Documentation of the results is necessary. Using point-of-care testing for important clinical decisions does expose people to possible medical-legal consequences and thus it would be recorded who did each test and the result should be clearly entered in the practice records.

Given the limitations of the method for point-of-care testing from troponin T, it would be advisable for all samples so tested to be sent for a confirmatory test. This would pick up the false negative tests that will undoubtedly occur.

The limitations of the troponin T point-of-care test are as follows:

1. The test is not as analytically sensitive as the laboratory test. Thus a negative result does not rule out myocardial infarction. The sensitivity quoted by the manufacturers is around 0.1ug/ml.

In actual clinical practice this is unlikely to be achieved and 0.15 is probably more realistic. The laboratory method has a quoted sensitivity of 0.01 but in actual practice is reliable only above 0.03. It should be noted that elevations above 0.03 are clinically significant but this level is well below the threshold of the point-of-care device.

2. The problems with sensitivity will be especially true if the point-of-care test is used early after a clinical episode suggesting myocardia ischaemia. Elevations of troponin are likely to be small and not detected at this early time and in some cases of definite MI the troponin T may not become positive, even with sensitive lab tests until up to 10 hours after the initial episode. This is why chest pain protocols include two tests for troponin T if the first is negative. Thus the use of the point-of-

care test for triage of chest pain patients in general practice is not very useful unless the first result is positive.

3. The best use might be in the stable patient presenting some time after chest pain especially after 10 hours.
4. Any positive test with the relatively insensitive point-of-care test should be regarded as definitely significant, even if it is very faint positive. False positives are possible but rare.

It should be remembered that while any raised troponin indicates myocardial damage, not all elevations are caused by ischemic damage. Levels can be elevated in severe heart failure, cardiomyopathy, pericarditis, pulmonary embolism with right heart failure and cardiac trauma.

[A chemical pathologist]”

Appendix B — The Second Public Hospital's chest pain protocol

Type: Procedure	Classification No. _____ Review Date: 11/07/07 Date Issued: 11/07/05
Title: Acute Management of Chest Pain & Acute Coronary Syndrome (ACS) Page 2 of 9	

Admission Decisions
Based on initial review, each patient needs to be stratified into a risk category.

- 1. High Risk**
 - Has one of the following features:
 - Accelerating rate of ischaemic symptoms over last 48 hours
 - Ongoing prolonged (>20 min) rest pain
 - New clinical findings – haemodynamic instability, LVF, new or worsening MR murmur
 - New ECG changes – pain related ST depression, new BBB, VT(sustained or non-sustained)
 - Significant TNI elevation (eg > 1.0)
- 2. Intermediate Risk**
 - Has one of the following features, and no high risk features:
 - Previous MI/CABG/PTCA
 - Vascular disease: either known or at high risk - Diabetes/PVD/CVA/TIA etc
 - Previous aspirin use
 - Prolonged (> 20min) chest pain at rest, now resolved, together with at least moderate clinical suspicion of ischaemic origin
 - Age > 70
 - New T wave inversion or Q waves on ECG
 - Minimal TNI elevation (eg TNI > 0.1 but < 1.0)
- 3. Low Risk**
 - Has no high or intermediate risk features but may have one of these features:
 - New onset anginal like symptoms, on mild or minimal exertion, with at least moderate likelihood of CAD
 - Normal or unchanged ECG during episode of chest pain
 - Normal TNI

All high risk patients should be admitted to hospital and most cases would be best managed in CCU.

Most intermediate risk patients should be admitted to hospital, unless their symptoms are obviously stable and have no features of recent progression.

Low risk patients and intermediate risk patients being considered for discharge should be kept in ED for planned repeat ECG and TNI in 6 -12 hours. Obviously if further symptoms occur in the interim they will need clinical reassessment and reestimation of their risk status. If repeat investigations at 6-12 hours still leave patient in low risk group, they can either be discharged with outpatient exercise treadmill test (ETT) arranged, and to take place within the next few days, or they can have an ETT performed before discharge. All ETT requests must be discussed with and approved by a medical registrar. All such patients should be counselled to see their GP within a few days. Further hospital follow up would depend on ETT result.

NB : If assessing patient with further chest pain within 2 weeks of AMI with TNI rise, one needs to order CK/CKMB, *not TNI*, as measure of cardiac muscle damage.

**Appendix C — The rural hospital's project brief for the review of the protocol for
Acute Management of Chest Pain**

Acute Management of Chest Pain
Risk Stratification

Project Brief

[The Rural Hospital]
21.01.03

BACKGROUND

developed its original Chest Pain Protocol in September 2003. The protocols and procedures were developed by . In a recent complaint which was considered before the Health and Disability Commissioner, expert advice given to the Commissioner from cardiologist Dr. T. Meegan indicated that further items should be addressed at in regard to patient presenting with chest pain. While believes its chest pain protocol was best practice at the time of development, we are eager to insure that all our practices remain state of the art. We therefore have authorised this project to review the practices and procedures in place with a view toward improvement.

PROJECT DEFINITION

OVERALL OBJECTIVE

wishes to develop best practice measures/procedures/protocols for the acute management of chest pain and Acute Coronary Syndrome (ACS) for use at

PROJECT SCOPE

The scope of this project includes but may not be limited to:

- Review the current Chest Pain Protocol
- Review of current Chest Pain Protocols at Hospitals
- Review of current cardiac monitoring equipment available in Hospital
- Review of current cardiac monitoring equipment in use at secondary and tertiary facilities including telemetry linking to these sites
- Discussion and agreement with secondary and tertiary centres on types of patients that can be/should be managed at and those that should be transferred.
- Modifications to practices and protocols that reflect the agreements which include external review by of documents by specialists
- Easily understandable and written documentation on stratification of risks involved
- Develop proposals and costs on any equipment modifications/updates necessary to implement the new procedures
- Review of the information/education available at about the sensitivity of all Troponin testing
- Review the timeliness of current lab testing arrangements with regard to Troponin testing
- Review current level of consultation with specialists in secondary and tertiary centres and suggest ways to increase this consultation effort.
- If required, develop education and informational offerings on Troponin testing
- If required, develop alternative lab testing arrangements for Troponin testing
- Review the level of educational sessions provided for Staff on chest pain issues
- If required, develop proposal for on-going training in cardiac best practice
- Develop protocols for provision of exercise treadmill testing for patients if unavailable at Hospital

Author:

3

EXCLUSIONS

This project is specific to chest pain protocols – other types of protocols and education are not included in the scope of this project.

METHOD OF APPROACH

_____ has appointed _____ as the manager of this project. It is anticipated that the project will last for a three to four week period. _____ will be excused from current Medical Officer duties during this period to insure completion.

ORGANISATIONAL STRUCTURE

_____ will report to CEO, _____ directly for the project. During the project, _____ may seek advice and guidance from all _____ staff and shall work in cooperation with _____ QA Coordinator.

PROJECT DELIVERABLES

The project deliverables will be a minimum of:

- A revised, written procedure for the Acute Management of Chest Pain & ACS
- A written risk stratification of patients presenting to _____ showing their associated risk level and what level of treatment that we can provide
- Written and prepared training programmes for Troponin Sensitivity
- Written memoranda of understanding with secondary and tertiary facilities over agreement of our chest pain procedures and their responsibilities upon our referral
- Written proposals for new, modifications or upgrades to current equipment
- Written proposals for changes to lab testing, if determined necessary
- Written communication/training plan to develop consultation process with tertiary specialists

QUALITY PLAN

The information developed in this project will be incorporated into _____ Quality system.

INITIAL PROJECT PLAN

RESOURCES

There are no funds in the current year budget to account for this work. All sums will be taken from reserves.

Expenditures should be moderate and take the form of one months medical officer wages, a mileage allowance for use of vehicle and visiting other centres and stationary and printing costs.

A budget of \$14,000.00 has been established to cover this work.

Author:

4