Dr B

A Public Hospital

A Report by the

Health and Disability Commissioner

(Case 03HDC01502)

Health and Disability Commissioner
Te Toihau Hauora, Hauātanga

Parties involved

Mrs A	Consumer (deceased)
Mr A	Complainant / Consumer's husband
Dr B	Senior House Surgeon / Provider
Public Hospital	Provider's employer
Dr C	Consumer's general practitioner
Dr D	Clinical Director, Emergency Department, Public Hospital

Complaint

On 30 January 2003 the Commissioner received a complaint from a solicitor, on behalf of his client, Mr A, about the services provided to Mr A's late wife, Mrs A. The complaint was summarised as follows:

Dr B did not provide services of an appropriate standard to Mrs A on 30 June 2002 when she presented to the Emergency Department at the Public Hospital. In particular, Dr B did not:

- *adequately assess and treat Mrs A;*
- *diagnose the cause of her pain.*

An investigation was commenced on 13 May 2003.

Information reviewed

I reviewed information from Mr A, Dr B, the Public Hospital, Dr C and a medical laboratory.

Independent expert advice was obtained from Dr Chip Jaffurs, a specialist in emergency medicine.

Information gathered during investigation

Background
Over the weekend of 15 and 16 June 2002 Mrs A, aged 43, complained of tiredness and was slightly run down. She was still unwell over the weekend of 22 and 23 June 2002.

On 24 June Mrs A was assessed by a general practitioner in the same practice as Dr C. He recorded that she complained of severe lethargy and tiredness with no obvious cause and had a mild productive cough. Her bowel motions, chest, cardiovascular system and thyroid were normal. Dr C prescribed co-trimoxazole (anti-bacterial medication) 480mg twice a day and requested a full blood count and an iron study.

The medical laboratory reported that Mrs A's ferritin (iron compound) level was 14.00 micrograms per litre (normal range 20-200) and recorded that "iron therapy is indicated".

Mrs A had an appointment with Dr C on 26 June to discuss the results of her laboratory tests. He prescribed one Ferrogradumet tablet once daily for three months to address her iron deficiency.

Mr A recalled that on 29 June his wife felt sick and complained of chest pain. She also complained that her tongue was tingling and her neck and throat felt numb.

Dr C assessed Mrs A at 3.22 pm and recorded that she had had four episodes of chest pain earlier in the day, although by then her chest pains had gone. Mrs A also had pain in her neck and tongue. Her blood pressure recordings were 150/90 and 140/85. An ECG was normal. Dr C recorded that Mrs A had "poss[ible] reflux" but he planned to obtain the results of further laboratory tests to assist his diagnosis. Her troponin-T level was later reported as 0.03ng/ml (normal range less than 0.03ng/ml). Dr C prescribed omeprazole 20mg once per day for 10 days to treat her reflux and requested a myocardial enzyme test.

Mr A recalled that Dr C informed him and his wife that she could have a heart problem or her stomach could be affected by the iron tablets; he could not be sure until he considered the results of the laboratory tests. Dr C suggested that Mrs A return or present to the Emergency Department (ED) at the Public Hospital if she had any further chest pains after his surgery was closed.

Visit to Emergency Department

On the evening of 29 June Mrs A felt a little better but at midnight reported that she was feeling sick, vomited three times (on the third occasion her vomit had blood in it) and complained of "severe chest/stomach pains".

Mrs A presented with her husband to the ED at the Public Hospital at 1.06am on 30 June. Mr A recalled that his wife informed a female senior house surgeon that her pain was burning rather than sharp.

The triage nurse recorded that Mrs A complained of burning epigastric pain "from her tongue down" and could not lie down because of her discomfort. The nurse also recorded that Mrs A had seen her GP the previous day with epigastric pain and "?reaction to iron tabs". Mrs A's vital signs were recorded as follows:

Time	BP	HR	RR	Temp	OS
1.20am	156/93	84	12	37°C	96%
1.40am	136/86	73	NR	NR	100%
1.45am	118/76	74	12	NR	100%
1.50am	127/74	73	NR	NR	100%
2.00am	131/76	76	14	NR	98%

Consultation with Dr B
Dr B, a senior house surgeon on call that night, recalled that Mrs A entered the ED in a wheelchair while he was in the trauma bay preparing for the arrival of an acutely unwell patient. The female senior house surgeon informed him that Mrs A complained of a burning pain in her stomach after commencement on iron tablets. Mrs A was placed in a holding bay and Dr B continued to prepare for the arrival of the other patient. After he had treated that patient a nurse requested that he assess Mrs A.

Dr B recorded that the preceding week Mrs A had been diagnosed by her GP with deficient anaemia and prescribed ferrous sulphate tablets which she had taken on 26, 27 and 28 June. She developed "terrible chest pain" on 29 June and her GP prescribed omeprazole. An ECG was normal. Mr A recalled that Dr B was informed that Dr C had requested blood tests to rule out a heart problem.

Dr B also recorded that Mrs A had "chest pain", had been feeling unwell the last few weeks and was tired. She reported that her pain was sharp and burning and went "all the way from the tongue down to the tummy". The pain did not radiate down her arm. Mrs A had no other history of note, was not taking any medication, had no allergies, smoked, and drank little alcohol. In response to the complaint Dr B advised that Mrs A denied feeling short of breath, clammy or having any numbness or altered sensation in her arms. He was also informed that Mrs A's father had had angina but not heart attacks.

Dr B examined Mrs A and recorded that her abdomen was very tender in the epigastrium and there was no rebounding, guarding, masses or scars. Her bowel sounds were active, her chest was clear and her heart sounds were normal. Mrs A's jugular venous pressure was at 1cm, an apex heart beat was palpable at the fifth intercostal space midclavicular line, her pupils reacted normally to light and accommodation, and her Glasgow Coma Score was 15/15 with movement in all four limbs. Dr B recalled that Mrs A's oropharynx and tongue were black.

Mrs A was administered omeprazole 20mg and Gaviscon 20mls (both for gastric reflux) at 1.06am, prior to her consultation with Dr B. Dr B prescribed Metoclopramide 10mg intravenously (to prevent vomiting) and ranitidine 50mg intravenously (to reduce stomach

acid), which were both administered at 1.40am. Mrs A was also prescribed morphine 10gm intravenously, although is not clear at what time this was administered.

Dr B diagnosed Mrs A with gastritis. He advised Mrs A to stop taking her ferrous sulphate tablets and to increase her omeprazole from 20mg to 40mg over the next week. In response to my investigation, Dr B stated that he advised Mrs A to return for reassessment if her condition did not improve.

Mr A advised that he questioned Dr B's diagnosis by asking how his wife's iron tablets could cause such severe pain. Mr A recalled that Dr B replied that he had made his diagnosis and turned his back on him. Mr A further advised that Mrs A was very sick and was having trouble explaining her condition, and Dr B should have listened to him.

Mrs A's death
Mr A advised that after leaving the ED (at 2.21am) his wife slept until 6.30am on 30 June. When she awoke Mrs A reported that she was feeling better and Mr A went to the supermarket. He returned at about 10.30am and noticed that his wife was sleeping on the sofa. Mr A said that when his wife awoke her stomach felt a lot better and her chest pains had gone, but she appeared very tired. Shortly after midday Mrs A was relaxed and seated when suddenly her arms flew outwards and her head went back. Her whole body shuddered and she gasped. Mr A said that he checked his wife's tongue but she was not breathing and had no pulse.

Mr A said that his wife's brother rang an ambulance while he tried to resuscitate her. After they arrived the ambulance staff tried for 20-30 minutes to revive Mrs A and because she still had a pulse decided to take her to hospital. Mrs A arrived in the ED at the Public Hospital at approximately 12.49pm in cardiac arrest. Staff were unable to revive her.

Mr A provided a post-mortem report, which stated that Mrs A appeared to have congenital narrowing of the distal left anterior descending and circumflex coronary arteries. The report concluded that Mrs A died from MI, occurring approximately 12-24 hours before her death.

Subsequent events
Dr B said that because he was shocked about Mrs A's death he immediately discussed the incident with a more senior doctor and with Dr D, the Clinical Director of the Emergency Department at the Public Hospital. Dr D, who responded on behalf of the Public Hospital, advised that at the "debrief" meeting with Dr B he endeavoured to be supportive but made the following points:

- Mrs A's cigarette smoking was a risk factor for cardiac pathology
- Mrs A reported chest pain which radiated to her mouth and throat area. This was consistent with cardiac pain
- Mrs A's pain was not improving (since consulting with her GP on 29 June) and she presented in the middle of the night

- The ranitidine Dr B prescribed for indigestion did not relieve Mrs A's symptoms

- Dr B should have confirmed the result of the troponin-T test (0.03ng/ml) requested by Mrs A's GP or contacted a laboratory technician, who was available by pager throughout the day and night. The troponin-T result was considered "borderline" at the Public Hospital (and a high level by the laboratory)

- Dr B should have requested a troponin-T test

- Dr B should have undertaken an ECG. Dr D stated in an email dated 1 July 2002 to […] Customer Relations Co-ordinator of the Public Hospital, that patients presenting to the ED with upper abdominal pain should have an ECG, particularly if the pain radiates upwards. Furthermore, 50% of patients who have heart attacks initially have normal ECGs (as in this case)

- Dr B should have consulted with a more senior doctor (in this case the on-call registrar).

Dr B advised me that he confidently diagnosed Mrs A with inflammatory gastritis because her pain was sharp and burning, she indicated by pointing and pressing that it was in her upper abdomen (rather than her chest) and it was reproduced on palpation of her stomach. Dr B considered that the pain in the upper part of her body was in her mouth and pharynx but not her neck or jaw. Ferrous sulphate tablets can cause gastric irritation and he thought the pain and blackness which he detected inside Mrs A's mouth was due to vomiting up these tablets. This explained the "tingling" which went "from her tongue to her tummy" and further supported his assessment that the pain came from her alimentary tract rather than her heart.

In response to my provisional opinion, Dr B recalled that he was not surprised ranitidine did not relieve Mrs A's pain. He explained that Mrs A did not have indigestion but gastritis caused by her ferrous sulphate tablets. Dr B prescribed ranitidine, which is not a pain killer, to reduce the acid in Mrs A's stomach caused by the ferrous sulphate tablets (and thus relieve her pain indirectly) and help her stomach to heal. He expected that this process would take longer than the relatively brief consultation. Dr B did not consider that the lack of response of Mrs A's pain to ranitidine indicated the presence of cardiac pathology. Dr B also explained that he prescribed ranitidine intravenously because Mrs A may not have properly absorbed the omeprazole taken orally (also prescribed to reduce stomach acid) because of her vomiting.

Dr B said that he knew that the causes of chest pain can be explored with cardiac enzymes blood tests, including a troponin-T level. The troponin-T level indicates whether there is any damage to the heart muscle. Dr B said that he did not know what blood tests had been requested by Mrs A's GP and had not been told during his orientation that he could obtain results requested by GPs on the hospital computer through a facility in the ED. The medical laboratory, which is based at the Public Hospital, advised that the troponin-T result was available at 9.57pm.

Dr B explained that he might have checked the GP's blood results if he had known they were available. He acknowledged that if the blood results or an ECG were abnormal, his diagnosis of gastritis would have been less likely or that gastritis could have been masking the symptoms of an acute coronary syndrome occurring at the same time.

Dr B said that, despite this, he would have expected the laboratory to inform Mrs A's GP immediately or the on-call GP to arrange admission if a cardiac enzyme test (which takes less than an hour) had been abnormal. This was the practice in every hospital he had worked in. Furthermore, Mrs A's GP could not have suspected that she had angina because blood tests cannot confirm this diagnosis. Dr B submitted that if Mrs A's GP suspected that she had unstable angina or a MI he would have referred her to hospital immediately without blood tests because this would have been a medical emergency.

Dr B recalled that he did not consider requesting an ECG or troponin-T blood test because he was confident in his diagnosis of gastritis, and Mrs A's medical history did not indicate that she was at risk of MI.

In response to my provisional opinion, Dr B advised that Mrs A had only one risk factor for coronary heart disease (smoking) and did not have, for example, hypertension, diabetes mellitus or hypercholesterolaemia [elevated cholesterol level in the blood]. Her father's angina did not increase her risk of MI. Dr B further advised that Mrs A's smoking did not mean that she would develop MI because a significant number of patients who experience this condition have none of the above risk factors. Dr B also stated that Mrs A had congenitally narrowed arteries (detected by post-mortem) which would have significantly increased her risk of MI and sudden cardiac death.

Dr B also advised that he was not surprised Mrs A presented to the ED at night because the symptoms of patients with gastro-oesophageal reflux are often worse at night or early in the morning. This is because the acid in the stomach (in this case iron sulphate) seeps up into the gullet and causes abdominal and chest pain when such patients lie down.

Dr B is genuinely sorry that, with hindsight, an ECG may have prevented Mrs A's death and saved her family from torment. Her death has caused him a lot of anguish, and he has discussed her case many times with junior and senior colleagues. Dr B has been receptive to their comments and has become significantly more cautious in relation to patients who present to the ED with abdominal and "non-cardiac" chest pain. Dr B advised in response to my provisional opinion that he now requests an ECG and orders blood tests when patients present with these symptoms.

Since the complaint he has also read extensively about atypical chest pain, to prevent a similar tragedy occurring, and is now well versed in the risk stratification of patients presenting with chest pain.

Training, availability of senior ED staff and guidelines

The Public Hospital advised that Dr B was employed at the Public Hospital from 26 November 2001 until 8 November 2002. His orientation was for one day when he began

work in the ED. Formal training in the ED was two hours every week. Dr B had previously worked in the ED.

Dr B recalled that his induction consisted of a tour of the ED and information about the practicalities of working there. He said that it was not always possible to attend the weekly teaching sessions because of shift work or leave.

Dr B advised that before the incident he had approximately eight weeks' experience in emergency medicine. Medical officers special scale (MOSS) provided supervision in the ED from 8am to 6pm during the weekends and for two days during the working week. A consultant was sometimes (Dr D stated "usually") available if urgent matters arose within normal working hours but there was no senior cover in the ED after hours. At the time of the incident the coverage was by the on-call medical registrar.

Dr D advised that the "problem" of after-hours coverage by ED senior staff is shared by other hospitals in New Zealand of a similar size. In response to my provisional opinion, Dr D advised that in November 2002 the Public Hospital attempted to recruit a second ED consultant by advertising the position internationally but there were no applications. An ED consultant from overseas has now been appointed (after the position was readvertised in July 2003) and will commence duties at the Public Hospital at the end of September 2004.

Dr B could not recall whether he had had any formal training in chest pain during the weekly teaching sessions or his orientation.

Dr D explained that patients with chest pain commonly present to the ED and there are several available textbooks that deal with the management of chest pain. At the time of the incident there was in place a clinical guideline for the management of acute MI in adults. This was developed and implemented with the cardiologists at the Public Hospital. The guideline states that an MI should be suspected if a patient presents with cardiac-type retrosternal chest pain lasting at least 15 minutes, which is unresponsive to glyceryl trinitrate. Patients with an MI can present with atypical chest pain, and an ECG can be nearly normal in the early stages. The guidelines require patients presenting with suspected MI to be admitted to the coronary care unit, and those with cardiac-type chest pain to be assessed within ten minutes, treated with a thrombolytic agent if appropriate, and administered nitrolingual spray. If the spray is not effective within 15 minutes, an ECG is to be conducted, and the patient assessed by a house surgeon or registrar.

However, Dr D advised that there were no hospital guidelines concerning the management of chest pain. In response to my provisional opinion, Dr D advised that, in light of comments made by my expert advisor (below), the absence of chest pain guidelines did not excuse Dr B's failure to consult with the on-call registrar about Mrs A's presentation. Dr D explained that it is not possible to have guidelines covering all clinical scenarios, but that the department is well endowed with textbooks.

Troponin-T tests
Dr D provided me with a joint memorandum from himself, a cardiologist and a specialist physician to senior staff dated 21 February 2002, concerning the use of troponin-T. The memorandum stated that:

- initial decisions on admission to hospital, thrombolysis and other management of patients with acute coronary syndromes should be based on clinical and ECG findings, not troponin-T or serum creatinine kinase (CK) results (CK is another cardiac enzyme which rises after MI).

- troponin-T should not be measured until at least six hours after the onset of pain and should not be used to rule out MI until at least ten hours after the pain.

Actions taken by Clinical Director of Emergency Department
Dr D advised that he took the following actions in light of Mrs A's case:

- Informed the customer relations officer

- Discussed the case with Mrs A's GP and presented it anonymously to ED medical staff at a weekly feedback session

- Informed the ED team leader and clinical nurse co-ordinator, who informed staff at the ED service improvement team meeting on 3 July 2002, that all patients who present with atypical chest pain must have an ECG

- Produced a guideline on atypical chest pain for the doctors in ED (Appendix A). In response to my provisional opinion, Dr D advised that this guideline was under discussion at the time of the incident, was partially implemented on 18 July 2002, and was fully in place from October 2002. The atypical chest pain guideline is now part of the induction for all doctors commencing work in the ED, many of whom are familiar with such a guideline because of their previous work experience in other hospitals.

- Continued to develop with cardiologists at the Public Hospital the clinical guideline for the management of acute MI in adults.

Dr D also advised that the Public Hospital and GP reporting systems for laboratory results have been amalgamated.

Apology
In his response to my provisional opinion, Dr B advised that he was willing to apologise to Mr A for incorrectly diagnosing his wife.

Independent advice to Commissioner

The following independent expert advice was obtained from Dr Chip Jaffurs, emergency medicine specialist:

> "Thank you for asking me to review complaint file 03/01502 for patient Mrs [A].
>
> I will summarise the case briefly. I will then answer your specific questions and conclude with some suggestions. I have read and will follow the Guidelines for Independent Advisors dated September 2003.
>
> I am an Emergency Medicine Specialist with fellowships in the Australasian College of Emergency Medicine and the American College of Emergency Physicians. I am currently the Director for Emergency Medicine for Whangarei Hospital.
>
> The patient Mrs [A] is a 43 year old female who presented three times over 6 days to her General Practitioner for fatigue, cough, laboratory and normal ECG testing and chest pain. She was thought to have gastroesophageal reflux and started on appropriate medications. She was not referred to the Emergency Department. Early in the morning of the seventh day she presented to the Emergency Department (ED) with nausea and vomiting with blood in the final vomitus. She presented at 0106 hrs and was seen by the Doctor and Nurse immediately according to the ED record. This is somewhat at odds with Dr [B's] recollection that he was attending to another very ill patient and she had to wait for awhile.
>
> The ED record is legible and complete. The final diagnosis was gastritis, consistent with the features described including chest pain and 'tummy' pain, burning in nature and abdominal tenderness. She had recently begun iron tablets which can irritate the stomach. Questioning reveals smoking history but family history is not mentioned nor are other pertinent negatives for cardiac risk assessment. Mrs [A] was treated with appropriate medicines for gastritis. I note that her pain required morphine. The nursing notes are sequential and confirm stable vital signs but not her response to medication. She is finally discharged from ED 1 and ¼ hrs later, apparently feeling better. The following day despite feeling better she suffers a cardiac arrest at home, is temporarily resuscitated by ambulance personnel, but dies in the ED despite an heroic attempt and duration of resuscitation. The subsequent available laboratory tests done the day before her death show minimally elevated troponin, and elevated blood lipids. The post mortem exam reveals recent posterior wall myocardial infarction, but no evidence of gastritis or similar gastrointestinal illness.
>
> In answer to your questions.
>
> **1. Did Dr [B] adequately assess and treat Mrs [A]? If not, what should he have done?**

Dr [B] displayed good practice in his general assessment and treatment of Mrs [A]. He saw her in a timely manner despite other pressing needs in ED. His record keeping is good. He formulated a working diagnosis and treated Mrs A successfully it seemed. He also fell into one of the most dangerous traps in Emergency Medicine. A more experienced Emergency Physician could have pursued a diagnosis of ischemic heart disease, sought consultation, or admitted Mrs [A] to Hospital.

2. Should Dr [B] have conducted an electrocardiogram and a troponin-T test?

It is reasonable to think that Dr [B] should have ordered an ECG, as a primary feature of Mrs [A's] complaint is chest pain. In my opinion this is a moderate departure from the standard of care for New Zealand Emergency Departments. Even if he accepted the GP's diagnosis of reflux esophogitis, the GP had done a 'normal' ECG that was unavailable to him. The ECG is an inexpensive non-invasive test that helps avoid misdiagnosis.

3. Should Dr [B] have reviewed the result of the troponin-T test or any other tests requested by Mrs [A's] general practitioner on 29 June which were available through a computer facility in the Emergency Department or contacted the laboratory technician about the results of these tests?

The results of the troponin test, if available, should have been checked. However the Hospital staff had been issued with a memorandum on 21 February 2002 from Dr […] contained in the documents from [the Public Hospital] discouraging the use of troponin testing for making admission decisions. I agree with this policy. Troponin test results that are negative can be extremely misleading and lead to inappropriate discharge. A borderline result such as that on record for Mrs [A], if available, should have led at a minimum to consultation with the Medical Registrar. At what time was this test result actually available?

4. Should Dr [B] have consulted with a more Senior Doctor?

Dr [B] was confident of his diagnosis. His treatment was effective. He did not appreciate the risk of an alternative diagnosis for this patient. In the materials you provided me there is no requirement for Medical or Surgical Consultation for un-differentiated chest/ abdominal pain in a policy. Possibly consulting a more Senior Doctor would have led to admission. It was reasonable in this scenario not to consult.

5. Should Dr [B] have diagnosed the cause of Mrs [A's] symptoms?

Dr [B] was not alone in misdiagnosing Mrs [A]. Her GP saw her for similar complaints and did not seriously think she could have ischemic heart disease, or he

would have referred her to the ED immediately. Although in retrospect the diagnosis is clear, Mrs [A] presented with a reasonably clear picture of a different diagnosis. I would not expect a Senior House Officer with less than 2 months' experience in Emergency Medicine to make a leap to the correct diagnosis. Many experienced Emergency Physicians miss this diagnosis making this the most common cause of expensive malpractice settlements in the United States, where Emergency Departments are uniformly staffed with Senior Doctors. Particularly women, even with known ischemic heart disease, are most often misdiagnosed when presenting to ED's with acute myocardial infarction (attachment one). Finally I note that Mrs [A] had a notation in her autopsy report of congenitally narrowed coronary arteries in some locations. This was unknown prior to her death and presumably would have added to her risk of premature myocardial infarction.

6. Please comment on the adequacy of: the availability of Senior Staff to provide advice to Dr [B].

Dr [B's] letter is the best source for a description of Senior staff available. He states 'No Senior cover within the Emergency Department was available out of hours. Matters would be referred to the appropriate specialist team on call'. This is the reality of ED staffing for base Hospitals in New Zealand at this time. Several Hospitals in a recent survey I conducted did not even have dedicated House Officers but relied on House Officers covering the whole Hospital to see patients in ED (attachment two). Senior staff including Moss' and Specialists are extremely difficult to recruit and I believe are in short supply at the Public Hospital. They are scheduled clinically and administratively to have the most impact on the greatest number of patients, that is during the day and evening. Dr [B] would have been able to consult a Medical Registrar for advice but chose not to do so being certain of his diagnosis.

7. All policies and guidelines relevant to the Emergency Department on 30 June 2003.

The only guideline I have been provided with as of 30 June 2003 is the Clinical Guideline for Management of acute myocardial infarction in adults dated 25 August 2000. This is a comprehensive guideline outlining a continuum of care beginning at presentation on page 41. Reference is made to circumstances for obtaining an ECG and atypical chest pain. I suspect the policy has almost no utility for an Emergency Department House Officer who is not seriously considering myocardial infarction as a diagnosis. Policies do not turn House Officers with 8 weeks' experience in Emergency Medicine into experienced Emergency Physicians. Dr [D's] excellent handout on atypical chest pain dated 3 July 2002 would also likely be soon forgotten in the barrage of paperwork and pathology encountered by a House Officer in the course of their routine duties.

8. The steps taken by the [Public Hospital] to address the circumstances giving rise to the complaint.

The documentation provided does not indicate steps taken specifically by the [Public Hospital] to address the circumstances. Dr [D], the ED Director, has undertaken extensive review of the case and urgently instituted several measures to prevent recurrence. I note exceptional concern for the family and health care professionals involved in the case, which is the first step towards repair in a case of this sort. I presume Dr [D] is representing the [Public Hospital's] interest, as well as the Emergency Department.

In conclusion I would suggest the Emergency Department develop a brief policy for evaluation of chest pain for use specifically in the Emergency Department. The policy should include un-differentiated chest and abdominal pain with an action plan for the House Officer. Faced with a similar challenge in our Emergency Department, we now insist all such patients see two doctors, one of whom must be a Registrar, Moss or Specialist (attachment three). It is an ongoing task to make House Officers aware that such a policy exists, despite orientation activities and documents. The increasing presence of locum House Officers who are not available for orientation prior to commencing work adds to the risk in such situations. I have attached our policy adapted from Hutt Hospital for your information. As I stated before, a policy does not make up for inexperience. As a long term goal, the [Public Hospital] must urgently recruit more Senior staff for its Emergency Department and eventually discontinue reliance on House Officers who are not immediately supervised by Emergency Physicians, whether they be Emergency Department Moss's or Specialists.

Attachments:

1. The Incidence of unrecognised MI in Women with CAD Ann Intern Med 2001: 134 Abstract format.
2. Survey of ED Staffing: Jaffurs.
3. Chest Pain Policy for Whangarei Emergency Department: Jaffurs/ Hutt Hospital."

[These attachments are in Appendix B].

Additional advice

I obtained further independent expert advice from Dr Jaffurs as follows:

1. Did Dr [B], taking into account his limited training and experience in Emergency Medicine, take adequate steps to rule out cardiac causes for Mrs [A's] pain in view of the fact that Mrs [A]:

- **Presented to the Emergency Department in the early hours of the morning reporting chest pain following a consultation with a General practitioner the previous day about chest pain;**
- **Informed Dr [B] that her father had angina;**
- **Smoked;**
- **Was given antacid medication then required intravenous morphine to treat her pain.**

No, Dr [B] did not adequately rule out cardiac causes of chest pain. The history does not contain either a complete description of the pain or a complete list of cardiac risk factors. The history as written supports a diagnosis of gastritis, but does not contain pertinent negatives for cardiopulmonary causes of chest pain. Troponin/cardiac enzymes should have been ordered in the Emergency Department, although results can be misleading for clinical decision making.

2. Please advise on the significance of Dr [B's] statement that he "found the inside of (Mrs [A's]) mouth and tongue to be black. This, at the time, I thought to be as a result of having vomited up the ferrous sulphate tablets. This explained to me the 'tingling' that Mrs [A] described going 'from her tongue to her tummy' and further supported the diagnosis of her pain emanating from her alimentary tract rather than from her heart."

A black coating on the tongue is most often produced by medications, most frequently antibiotics or those containing bismuth salts. The description of tingling is not diagnostic of gastrointestinal pain. I do accept that a black coating on the tongue could result from vomiting iron containing stomach contents, but it seems more likely to have resulted from her smoking or self administered bismuth containing antacid. I note she was treated one week earlier with antibiotics as well.[1]

3. In your initial advice you stated that was reasonable that Dr [B] did not consult with a more Senior Doctor about Mrs [A's] presentation. Please clarify your reasons for this. I note that, although the result of the Troponin-T requested by Mrs [A's] GP was available to him, (see information from the laboratory), Dr [B] has contended that he did not know this.

Dr [B] had what he thought was enough information to make a diagnosis. We now know it was the wrong diagnosis. Mrs [A's] General Practitioner appears to have made a similar diagnosis. Senior Doctors are consulted regarding patients for whom a diagnosis is unclear or who require admission. I am not aware of a Base Hospital anywhere in New Zealand that requires discharged patients be discussed with a more

[1] Atlas of Emergency Medicine 2nd Edition McGraw Hill P184 'Black Hairy Tongue'

Senior Doctor in a real time sense. The New Zealand model is frontline staffing by non-specialist Emergency Doctors, who must be at least in their second post graduate year. Policies, guidelines, teaching and orientation are falsely relied upon to provide an expert level of care. In this system, Dr [B] was following the rules.

With regard to troponin, even if the GP's result was in hand, it should have been repeated by a reasonably cautious practitioner.

4. Was Dr [B's] management of Mrs [A] appropriate, in particular discharging her home?

Taken from the point of diagnosis, which was gastritis, Dr [B's] management and discharge decision were appropriate. This may not seem sensible, but the error is in the diagnosis, not the subsequent management plan. Clearly, a patient with suspected acute cardiac ischemia would be managed with monitoring oxygen, drugs and admission to Hospital. As I stated in my previous letter of 30th September 2003, many Specialist Emergency Physicians have made similar errors as witnessed by the situation in the United States where Emergency Departments are routinely staffed by Specialist Emergency Physicians, and missed myocardial infarction continues to result in the largest settlements for malpractice actions.

5. What policies and procedures, if any, should the [Public Hospital] have had in place at the ED at the time of the incident to guide Senior House Surgeons when they were not supervised by Senior Staff present in the ED, in the assessment and treatment of undifferentiated chest/ abdominal pain? In your response please include advice on policies and procedures requiring ECGs and consultation or referrals.

At a minimum, [the Public Hospital] should have had available a policy pertaining to the management of chest pain which presents to the Emergency Department. The policy should specify rapid ECG testing, identification of myocardial infarction and criteria for thrombolysis.

While many hospitals have such policies, experience at our hospital and [the Public Hospital] clearly shows weakness in identification of coronary ischemia in the setting of atypical chest pain, and undifferentiated chest/ abdominal pain.

My September 30th letter contained a suggested policy for risk stratification. The most important innovation is that all patients with chest pain must be seen by a Senior Doctor (Registrar, Moss, or Consultant) prior to discharge. This policy is held in addition to, not instead of, the thrombolysis policy.

6. Please comment on the adequacy of the steps taken on behalf of the [Public Hospital] by Dr [D] since the incident.

Dr [D] has responded in a timely and forthright manner. I suggest the addition of a chest pain stratification tool that is brief, intelligible, and mandates Senior Doctor Consultation as described in answer 5.

The long term goal for the [Public Hospital] should be to staff the Emergency Department with Registrars and Moss's specific to Emergency Medicine after hours, rather than continuing to rely on House Officers.

7. I note your comment that the ED should develop a brief policy for evaluation of chest pain. What training, if any, should be put into place in respect of this policy?

The chest pain policy must be discussed in orientation along with other pertinent core policies. ECG skills and Troponin interpretation are best taught repeatedly and supervised by more Senior Doctors. This can happen in teaching sessions and at the bedside. The Advanced Cardiac Life Support Course also emphasises these skills and should be required of non-specialist Doctors before commencing work in the Emergency Department.

In many ED's including mine, these 'requirements' are not realistic. Inadequate or no time at all, may be allowed for orientation. Cost and availability limits attendance of ACLS. Doctors miss teaching because of fatigue or work requirements."

Code of Health and Disability Services Consumers' Rights

The following Right in the Code of Health and Disability Services Consumers' Rights is 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.

Opinion: Breach – Dr B

In my opinion, Dr B breached Right 4(1) of the Code of Health and Disability Services Consumers' Rights (the Code) by failing to adequately assess Mrs A's condition on 30 June 2002.

Mr A complained that his late wife, Mrs A, was not correctly diagnosed and treated by Dr B at her first presentation to the ED at the Public Hospital on 30 June 2002. Mrs A presented with ongoing severe "chest/stomach" pains and vomiting. Dr B was informed that her GP thought she might have a heart problem and had ordered blood tests to check this, although her ECG was normal. Dr B diagnosed Mrs A with stomach problems and discharged her home. Later that day Mrs A collapsed and was taken by ambulance to the ED where she died. Mr A provided a post-mortem report, which concluded that Mrs A died from an MI occurring approximately 12-24 hours before her death.

Dr B advised that he diagnosed Mrs A with inflammatory gastritis because her lower pain was sharp and burning and located in her upper abdomen, rather than her chest. He attributed the pain to ferrous sulphate tablets prescribed by Mrs A's GP, since they are a gastric irritant. Mrs A's upper pain was in her mouth and pharynx (but not her neck and jaw) and seemed to be caused by vomiting up the tablets. The tingling she described – from her tongue to her tummy – further supported his assessment that the origin of Mrs A's pain was her alimentary tract.

Dr B explained that he did not request a troponin-T blood test or an ECG to investigate possible cardiac causes of Mrs A's pain because he was confident in his diagnosis and her medical history did not indicate that she was at risk of MI.

My expert in emergency medicine, Dr Jaffurs, advised that it was reasonable for Dr B not to diagnose Mrs A with an MI in light of his experience and training and given that Mrs A presented with a reasonably clear picture of gastritis. It was also reasonable for him not to consult a more senior doctor, because there was no such policy in place at the ED concerning atypical chest pain or undifferentiated chest and abdominal pain. However, Dr B "did not adequately rule out cardiac causes of Mrs A's chest pain" by failing to request an ECG or check the results of the troponin-T test requested by her GP. My advisor considered that the failure to order an ECG was a moderate departure from the appropriate standard of care.

Dr Jaffurs stated in his supplementary advice that Dr B should also have requested a troponin-T test himself and further explored the nature of Mrs A's pain, for example, its duration and frequency. This was warranted in light of Mrs A's presentation to the ED in the early hours of the morning with ongoing chest pain, and her cardiac risk factors, for example smoking and the fact that ranitidine did not ease her pain, and she required morphine. I also note Dr Jaffurs' advice that the symptom of tingling is not diagnostic of gastrointestinal pain, and the blackness inside Mrs A's mouth and tongue was more likely caused by smoking, medication containing bismuth salts, or antibiotics.

In response to my provisional opinion, Dr B advised that his diagnosis of gastritis and treatment of Mrs A was reasonable because she had only one risk factor for cardiac pathology (smoking). Her other risk factor, congenitally narrowed coronary arteries, had not been detected at the time of the consultation. Further, Mrs A's lack of response to ranitidine did not indicate the possibility of cardiac pathology, as this medication takes time to reduce acid in the stomach and relieve pain. It was not suspicious that Mrs A presented at 1.06am to the ED because symptoms of gastro-oesophageal reflux are often worse at night when patients are lying down.

Nonetheless, even allowing for the benefit of hindsight and his relative inexperience, in my view Dr B's confidence that Mrs A's description of "chest pain" and other symptoms were adequately explained by his diagnosis of gastritis was not warranted. At the very least Dr B should have recognised that the origin of Mrs A's pain (abdomen or chest) was not clear and warranted further investigation with an ECG or troponin T test, which are both straightforward investigations in a hospital setting, to rule out cardiac causes for her symptoms. In the circumstances a cautious approach to Mrs A's management (including possibly discussing her case with the on-call consultant) was appropriate to minimise potential harm to her. In my opinion, Dr B's failure to rule out a cardiac cause for Mrs A's chest pain showed a lack of reasonable care and skill, in breach of Right 4(1) of the Code.

Opinion: Breach – Public Hospital

While I commend the steps taken by the Public Hospital since this case to ensure that patients who present to the ED with chest pain are safely managed, and acknowledge Dr D's comments that it is not possible for an ED to have guidelines covering all clinical situations, in my opinion the Public Hospital breached Right 4(1) of the Code for the reasons set out below.

The Public Hospital was subject to a legal duty to provide emergency medicine services at the level of care and skill reasonably expected of a Public Hospital. The evidence, and my expert advice, indicates that the Public Hospital fell short of its corporate responsibility as a provider of publicly funded emergency medicine services.

My expert advisor considered that at the time of the incident the Public Hospital should have had in place a written policy, with an action plan, to guide ED medical staff on the management of chest pain. In my opinion, a chest pain policy is especially warranted in public hospitals where junior doctors are not under direct supervision from senior ED or other medical staff. Patients with chest pain commonly present to the ED. Cardiac pathology can be easily overlooked in the presence of atypical chest pain or undifferentiated chest and abdominal pain, even by experienced emergency medicine providers. It is important to minimise this risk, to ensure that patients presenting with possible cardiac pathology are identified and treated at an early stage.

This risk appears to have been recognised by the Public Hospital in 2002. Its guideline on atypical chest pain states that "approximately 2% of patients with MI are discharged inadvertently from emergency departments". The guideline was partially implemented on 18 July 2002 and was fully in place from October 2002.

In my opinion, by failing to have in place at the time of the incident a written ED policy concerning the management of chest pain, the Public Hospital breached Right 4(1) of the Code.

Recommendation

- I recommend that the Public Hospital and Dr B apologise in writing to Mr A.

Follow-up actions

- A copy of this report will be sent to the Medical Council of New Zealand.
- A copy of this report, with details identifying the parties removed, will be sent to the Australasian College for Emergency Medicine (New Zealand Faculty) and placed on the Health and Disability Commissioner website, www.hdc.org.nz, for educational purposes.
- A copy of this report, with personal identifying details removed, will also be sent to the Chief Medical Advisors of all District Health Boards.

APPENDIX A

Atypical Chest Pain

Legal settlements for missed myocardial infarction (MI) are the most expensive group of claims made against emergency doctors in the U.S.A.

Please bear in mind the following points:

- Myocardial infarction can occur in relatively young people. Groups to consider are those with:
  - Any cardiac history
  - Diabetics
  - Certain ethnic groups, e.g. Commonwealth Asians (from India & Pakistan)

- Atypical symptoms may include:
  - Pleuritic or sharp pain
  - Epigastric pain
  - Burning or 'dyspeptic' pain
  - Pain in tongue
  - Dyspnoea without pain
  - Weakness
  - Light-headedness
  - No symptoms – e.g. in diabetics and the elderly

  ECG's should therefore be obtained in such patients.

- Risk factors are important but patients without risk factors do die from MI.

- Do not assume that an apparently good response to antacid or GTN indicates that the pain is attributable to gastritis or stable angina – the pain of MI is frequently episodic and administration of medication may have a placebo effect.

- The physical examination frequently reveals no objective abnormality.

- ECG's of patients presenting to emergency departments with chest pain are normal in 50% of those who are subsequently found to have MI. Repeat your ECG's at appropriate intervals.

- Troponin-T cannot be used alone to rule out MI until at least 9 hours following the onset of chest pain.

- Approximately 2% of patients with MI and 2% of patients with unstable angina are discharged inadvertently from emergency departments. Continuing ischaemia in unstable angina is potentially as problematic as myocardial infarction – hence the term *acute coronary syndrome* which embraces both of these conditions. Mortality from a first prolonged ischaemic episode is 34%.

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- Provocative testing plays an important role in ruling out MI, unstable angina, and coronary disease. A protocol for this is currently in preparation.

- Women of less than 55 years who are suffering an acute coronary syndrome have a sevenfold risk of inappropriate discharge from ED.

- In a study of patients discharged inappropriately, 83% of ECG's were initially normal and 82% of patients died.

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APPENDIX B

Abstracts

TH[illegible]racts and commentaries in this issue were prepared by editorial board members of the *Year Book of Emergency Medicine*. These selections will appear in the 2002 volume. To order a copy of the entire 2002 *Year Book of Emergency Medicine*, [illegible] Mosby's toll-free number 800-453-4351 or 314-453-4351 outside the United States.

Section Editor
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Reprints not available.

The Incidence of Unrecognized Myocardial Infarction in Women With Coronary Heart Disease

Shlipak MG, for the Heart and Estrogen/progestin Replacement Study Research Group
(University of California, San Francisco, CA; Stanford University, Stanford, CA; Wake Forest University, Winston-Salem, NC
Ann Intern Med. *2001;134:1043-1047*

Introduction: The incidence of unrecognized myocardial infarction is not well characterized for populations with known coronary heart disease (CHD). Data on the risk for unrecognized myocardial infarction in such populations could affect surveillance strategies. Serial ECGs were used to determine the incidence of unrecognized myocardial infarction in women with CHD.

Methods: Participants were the 2,763 women in the Heart and Estrogen/progestin Replacement Study (HERS). All were postmenopausal, younger than 80 years, had a uterus, and had received a diagnosis of coronary artery disease. Randomization was to placebo or to a single tablet containing conjugated estrogens, 0.625 mg, plus medroxyprogesterone acetate, 2.5 mg. The mean duration of follow-up was 4.1 years. Follow-up included 12-lead ECGs, performed at baseline and annually thereafter. Characteristics of patients who had unrecognized myocardial infarction were compared with those of patients who had clinically recognized myocardial infarction.

Results: A total of 256 nonfatal myocardial infarctions occurred during follow-up, but only 11 (4.3%) were unrecognized. These events occurred in 4 of 1,380 women in the hormone therapy group and 7 of 1,383 women in the placebo group. The incidence of unrecognized myocardial infarctions did not differ according to participant age, but women with unrecognized myocardial infarction had lower lipoprotein(a) levels, were less likely to have diabetes mellitus or recent angina, and were more likely to have had bypass surgery before the unrecognized event. Forty-five (18%) women with recognized myocardial infarction and 1 (9%) with unrecognized myocardial infarction died during follow-up.

Conclusion: In contrast with the findings of previous studies, the incidence of unrecognized myocardial infarction in women with known previous CHD was low. The lower observed rate of unrecognized myocardial infarction, compared with other studies, may be attributed to the fact that HERS participants were contacted more frequently, knew their risk for ischemic events, and could have recognized atypical symptoms.

Comment: *In a population of women with known coronary artery disease, the incidence of silent, or unrecognized, myocardial infarction was 4.3%. Interestingly, this is less than half the number observed among women in the Framingham study. Participants in the present study may have been more alert to subtle symptoms because they knew they had coronary artery disease.*

Even so, assuming that "silent" myocardial infarction is not a completely separate clinical entity, it must represent the end of a spectrum of severity of symptoms. We will see a finite number of patients whose symptoms are extremely subtle who will turn out to have infarctions. The best ECG reading

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skills and the finest clinical acumen will still result in a few "missed" infarctions.

W. P. Burdick, MD

False-Positive ECG Reports of Anterior Myocardial Infarction in Women

Colaco R, Reay P, Beckett C, et al
(University of Glasgow, Glasgow, Scotland)
J Electrocardiol. *2000;33:239-244*

Background: One known source of inconsistency in the diagnostic interpretation of the 12-lead ECG is variation in precordial electrode placement. It has been suggested in one study that the clinically relevant change in automated ECG interpretations as a result of incorrect placement of electrodes may be as high as 50%. For women in particular, alteration in placements of precordial electrode V2 to V4 and the subsequent effect on the diagnosis of anterior myocardial infarction has never been the subject of a detailed investigation. In an earlier investigation of the prevalence of poor R-wave progression, all ECGs recorded at the Glasgow Royal Infirmary during a 2-week period were reviewed. The results indicated a higher prevalence of poor R-wave progression in women (19%) than in men (11%). The positioning of chest electrodes in women as a possible reason for this discrepancy was investigated.

Methods: Eighty-four women were recruited for this study. Chest electrodes were placed strictly as recommended, with the fourth and fifth intercostal spaces as references; the widely adopted technique of placing electrodes V3 to V6 under the left breast was used. R-wave amplitudes were then compared in V3 to V6 from both sets of recordings.

Results: Measurements on the breast recorded by electrode V3 were found to have a significantly smaller R-wave magnitude than corresponding measurements below the breast, with the mean difference being 34 μV. The opposite was found to be true for electrodes V5 and V6, with measurements taken on the breast being larger (119 and 134 μV, respectively) than those taken below the breast. No significant difference was noted for electrode V4. In 17 women with poor R-wave progression that was suggestive of old anterior myocardial infarction, examination of clinical data indicated that 11 of these women had a history that was suggestive of myocardial infarction, for a positive predictive value of 65%.

Conclusions: The positioning of electrodes beneath rather than on top of the breast did not account for the increased prevalence of poor R-wave progression in women. Also, the criterion of isolated poor R-wave progression was too nonspecific to be of clinical value.

Comment: *Generally accepted ECG criteria for acute myocardial infarction are new or presumably new Q waves, ST-segment elevation or depression, and left bundle-branch block. Approximately one third of patients with prior or old anterior myocardial infarction may show only "poor R-wave progression" on the standard ECG. Several definitions or criteria for poor R-wave progression exist, and it is clear that this ECG finding may also be encountered in patients with left ventricular hypertrophy, right ventricular hypertrophy, left bundle-branch block, some cases of Wolff-Parkinson-White syndrome, and as a normal variant in healthy persons. The prevalence in healthy patients is about the same as that in patients with prior anterior myocardial infarction. The specificity of this ECG finding for anterior myocardial infarction is 50% to 60%.*[1]

The study by Colaco et al attempted to determine why poor R-wave progression is more commonly encountered in women. ECG lead placement is a recognized cause of poor R-wave progression. In this study, the investigators varied the position of precordial leads V3 to V6 (ie, as recommended or under the left breast). Measurement of the resulting R-wave amplitudes for these lead placements in the same patient failed to prove that positioning the electrodes beneath rather than on top of the breast was responsible for the increased prevalence of poor R-wave progression in women. The authors' conclusion is that of others, namely, that isolated poor R-wave progression is too nonspecific to be of clinical value.

Another lesson from this study is that ascribing "abnormal" ECG findings to lead placement is fraught with error.

J. T. Niemann, MD

1. Zema MJ, Klingfield P. ECG poor R-wave progression: review and synthesis. Arch Intern Med. 1982;142:1145-1148.

Age-Related Differences in In-Hospital Mortality and the Use of Thrombolytic Therapy for Acute Myocardial Infarction

Boucher J-M, for the Quebec Acute Coronary Care Working Group
(Université de Montréal, Montréal, Quebec, Canada; et al)
Can Med Assoc J. *2001;164:1285-1290*

Introduction: Acute myocardial infarction (AMI) remains a leading cause of death, especially in elderly patients. Thrombolytic therapy reduces the short- and long-term mortality from AMI, but some trials have not confirmed these benefits in patients older than 75 years. The recent age-related in-hospital AMI mortality rate in a large patient cohort was examined, and the age-related differences in the use of thrombolytic therapy for patients with AMI were determined.

Methods: Included in a prospective registry were 44 acute care hospitals in Quebec. The study population consisted of 8,917 consecutive patients admitted to emergency departments with a suspected acute coronary syndrome. Patients were divided into 5 age groups. Independent variables controlled for included coronary artery disease and relevant interventions, symptoms on admission, and time between onset of symptoms and hospital arrival.

Results: A final diagnosis of AMI was made in 3,741 patients, 3,612 of whom were eligible for analysis. Older patients were less likely to be men and smokers and were more likely to have a history of hypertension or coronary artery disease. Older patients were also less likely to be admitted with typical chest pain. In-hospital mortality rates increased sharply with age, from 2.1% in those younger than 55, to 26.3% in those aged 85 years or older. After adjustment for potential confounders, older patients were significantly less likely to receive thrombolytic therapy. Odds ratios for receiving the therapy, compared with the youngest patient group, were 0.68, 0.48, and 0.13, respectively, for patients aged 65 to 74, 75 to 84, and 85 or older. Other variables associated with a lower rate of thrombolytic therapy included

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Emergency Department Survey

15 October 2002

27 Emergency Department in New Zealand
6 with Annual visits 20,000 – 30,000
(Excluded: Starship Children's Hospital)
(Included: Tauranga Hospital; volume= 36,000)

Location	Annual Visits	Staff	ED Senior Cover		SHO At Night	Night SHO Covers Ward
			Night OC	Weekend On Site		
Whangarei	20,000	SHO x 5 Moss x 2 Reg x 2 Director x 1 *Specialist	Yes	Yes	Yes	No
Hawkes Bay	30,000	SHO x 1 SHO x 5 (Nov 02) **Mossx6 Director x 1	No	Yes	Yes Yes	Yes (at present) No (Nov 02)
Palmerston North	28,000	SHO x 7 *Moss x 2 Director x 1 *Specialist x 1	No	No p.m. shift Yes Sat No Sun	Yes	No
Rotorua	20,000	SHO x 5 MOSS x 2 Locums	No	Yes	Yes	No
Southland	24,000	SHO x 5 MOSS x 3 *Director x 1 *Specialist x1	No	Yes	Yes	Yes – At Present No- Dec 02
Taranaki	21,000	SHO x 2 ***MOSS x 5 Director X 1 Specialist x 1 Locums	No	Yes	No	Not Applicable
Tauranga	36,000 No trauma	SHO x 5 MOSS x 4 Director x 1 Specialist x 1	No	Yes	Yes	No

* Denotes Positions Vacant

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CHEST PAIN MANAGEMENT IN ED

Patients with Chest Pain require a careful and cautious approach in order to minimise risk. History and Physical, blood testing and ECG findings are used in concert to stratify patients into Management Groups in the following scheme.

Particular risks include patients whose Chest Pain resolves in ED with or without analgesia. Resolution of pain does not alter Group assignment, undifferentiated chest and abdominal pain are **Group 4** if any cardiac risk factors are present. **Group 5** Chest Pain patients may not be discharged from ED without full consultation with a more Senior Doctor (Registrar, Moss or Consultant). All other groups require admission.

Troponin testing is only helpful for diagnosis if positive. Negative troponin in the Emergency Department does not rule out Ischemic Coronary Syndrome.

CHEST PAIN GROUPS

GROUP 1: (CCU)

Criteria: MI suitable for thrombolysis if no contraindication

ST elevation

LBBB (not known to be old).

GROUP 2: (CCU UNLESS MULTIPLE CO-MORBIDITIES)

High Risk Unstable Angina and Non ST elevation Infarct

Criteria: History of Ischaemic Chest Pain plus any one of the following:

Troponin I +ve

ST depression

Angioplasty <6 months ago

Pulmonary oedema

Hypotension

N.B. Some patients continuing to be unstable may be suitable for Tirofiban – D/w Consultant.

GROUP 3A: (CCU/ MEDICAL WARD)

Unstable Angina

Criteria: History consistent with Unstable Angina plus any one of the following:

Previous MI or clearly documented coronary disease

Angioplasty >6 months ago

Diabetes

Poor LV function (EF <50%)

GROUP 3B: (MEDICAL WARD)

Unstable Angina

Criteria: History consistent with Unstable Angina

GROUP 4: (MEDICAL WARD)

Possible Unstable Angina

Criteria: Chest Pain of uncertain clinical diagnosis

GROUP 5: (CONSIDER MEDICAL ADMISSION, MEDICAL OR EMERGENCY MEDICINE CONSULTANT, MOSS, REGISTRAR REVIEW IS REQUIRED)

Non Cardiac Chest Pain, Undifferentiated Chest and Abdominal Pain without alternative diagnosis.

Negative ECG

Non Cardiac risk factors

Clinical Director
Emergency Medicine
July 2002

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