17 December 2004

Private & Confidential

Mr George Connelly

Dear Mr Connelly

I refer to your complaint about a health service your late wife, Mrs Doreen Connelly, received from Bundaberg Base Hospital on 2 December 2003. At the outset, I wish to convey my sincere condolences to you for the loss of your wife.

As you are aware, the Commission has been assessing the complaint to determine whether the health service provided to Mrs Connelly was reasonable and whether any further action may be required.

I understand that Mrs Connelly, who had a history of ischaemic heart disease, woke at 0330 hours on 1 December 2003 suffering with chest pain. An ambulance was called and transported her to Bundaberg Base Hospital at 0446 hours. Ambulance records state that on arrival at the scene, Mrs Connelly's pain had ceased in the chest but she still had pain in her back.

At the hospital, the duty medical practitioner noted Mrs Connelly's past history of acute myocardial infarction and hypothyroidism. She was examined, her vital signs monitored and no abnormality was detected. Various tests were performed which included serial electrocardiographs (ECGs) and while the chest x-ray was normal, blood tests showed raised levels of troponin[1]. Mrs Connelly was admitted to a general ward and later that day was reviewed by the specialist medical team who diagnosed her as having unstable angina. Aspirin, Lipitor and Lasix were added to her medication regime and she was discharged home at 1430 hours on 2 December 2003.

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I understand that before Mrs Connelly was discharged, you explained to staff that she had been referred by her general practitioner the previous week for a stress (sestamibi) test to be performed by North Coast Nuclear Medicine at Mater Hospital that day at 10.20 a.m. The hospital's clinical plan for Mrs Connelly had been to take further blood tests and, if normal, the stress test would go ahead as planned. Following the appropriate blood tests and review of those tests, she was discharged with arrangements to transfer Mrs Connelly for her stress test. Before Mrs Connelly left the hospital the nursing staff member contacted North Coast Nuclear Medicine, and was told that the appointment had been reallocated and a new appointment was made for six days time. Tragically, your wife died in the early hours of the morning of 3 December 2003, at home. Her death certificate indicated that she died from a cardiac arrest following a myocardial infarction.

[1] An independent biochemist explained that troponins are muscle proteins found in the blood, which can be tested and analysed, following suspected heart muscle damage. High readings of troponin occur following cardiac damage.

Complaint Issues

I understand that your complaint issues are:

- Mrs Connelly was misdiagnosed and had she been correctly diagnosed and given appropriate treatment she would not have died; and
- Mrs Connelly should have attended the stress test, and if she had, she would have been correctly diagnosed and treated.

Misdiagnosis Issue

You stated that you were later informed that Mrs Connelly's past cardiac history and her elevated troponin levels were not taken into account when the decision was made to discharge her. You stated that you believed that had Mrs Connelly been correctly diagnosed and treated, she would not have died.

Mr Peter Leck, District Manager, Bundaberg District Health Service advised the Commission that the hospital had conducted a review of Mrs Connelly's care. The review confirmed that the combination of Mrs Connelly's past history, prolonged chest pain, ECG changes and raised troponin values indicated that she should have been diagnosed with Acute Coronary Syndrome and remained in hospital for ongoing observation. Mr Leck offered his sincere apologies to you for this failure.

In a further letter to the Commission, Dr Darren Keating, Director of Medical Services, explained that the significance of the raised troponin level was not appreciated. Dr Martin Strahan, general physician who attended to Mrs Connelly, was a visiting consultant who also worked in the private sector. It was explained that Dr Strahan did not appreciate the significance of your wife's troponin measurement because of the different measurement systems being used in the public and private health sectors leading to potential discrepancy between troponin values for the same patient. This discrepancy contributed to Dr Strahan placing limited significance on the test results at Bundaberg Base Hospital.

Dr Keating advised that Dr Strahan's reliance on the private sector method was based on his belief that the public sector method was inaccurate and possibly inferior. Dr Keating said that Dr Strahan reported that he attempted to clarify the matter with Queensland Health Pathology Services in Rockhampton some time prior to Mrs Connelly's admission but did not receive clarification. Subsequently, he ordered a different blood test (creatinine kinase), and as this was normal, he agreed to the stress test and subsequently agreed to the discharge of Mrs Connelly. Dr Keating advised the Commission that the private pathology provider in Bundaberg had recently installed the same troponin analyser as theirs to offset any future confusion.

Dr Keating also advised that the hospital has begun involvement with the Collaborative for Healthcare Improvement, Acute Coronary Syndrome, which provides evidence based guidelines and systematic evaluation of the treatment for this disorder in their hospital. The results for Bundaberg Base Hospital can be compared on a state-wide basis with all hospitals involved in the project. He explained that since Mrs Connelly's death there had been an education session for all medical staff involved in the care of cardiac patients. There were also continuing education sessions for senior medical staff on the specific topic of Acute Coronary Syndrome and the management of patients with raised troponin measurements. Dr Keating also advised that Dr Strahan had since undertaken further study, attended a cardiology conference and sought ongoing advice from cardiology peers.

The Commission approached three independent advisers who agreed that Mrs Connelly should not have been sent home. A Cardiology Registrar (is it appropriate that a junior medical practitioner (in a training program) to make comment on a senior specialist with many years of experience ? Should it not be peers ?) stated that although both methods of troponin measuring give a "*normal/abnormal*" reading, it was possible that the specialist was used to looking at "*one set of numbers*". When asked to comment on the actions of the specialist he stated that the hospital had acknowledged that they had deviated from the state-wide guidelines and indicated they were making changes. An independent Deputy Medical Director

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of a cardiology programme stated that whether or not troponin was positive or negative "*may not be the issue*" and explained it was necessary to look at the systems in place. He explained that at the hospital where he worked, which specialised in heart conditions, if a person with a history of heart condition presented with chest pain, they would be "*kept in automatically*" regardless of troponin readings and this was an example of a systems approach. (Is this comparing apples with apples ? No, a specialist tertiary centre should not be compared with a regional/secondary centre.) The Deputy Medical Director stated that the hospital had admitted to systemic errors and said he felt that the reason why the woman was discharged would not come down to a "*single decision*" but due to the lack of a systemic approach. He said that while it would be "*expedient*" to discipline an individual doctor, this would not solve the greater problems, and would be inappropriate. He agreed with the previous adviser that he felt not much more be "*gained*" by looking at an individual registrant as he felt all pertinent issues had been covered. A Director of Cardiology in a large public hospital was also approached for advice and he and stated, like previous advisers, that the stress test was contraindicated and it would only have confirmed what the hospital should have already known. In relation to the hospital's diagnosis of unstable angina, the Director of Cardiology explained that the term "*acute coronary syndrome*" was a very broad umbrella term to cover lots of coronary conditions and as the hospital stated that the woman was stable throughout her admission, the diagnosis of unstable angina was "*not incorrect*". He stated that the error was to discharge her too soon. He said that the blood tests certainly flagged that she was at a higher risk of suffering a heart related problem, which she did, but the error was "*not so much in the diagnosis as in failing to recognise that her Troponin levels mandated that she receive more intensive therapy rather than be discharged*". He noted that the hospital had undertaken procedural changes and that a sincere apology had been given. Further independent advice said that had Mrs Connelly been kept in hospital, even in the Coronary Care Unit, there were no guarantees that she would have survived her cardiac arrest.

[I think disregard]
* ie. the Collab for Health Care A.C.S. is trying to address/incorporate such issues & P. is now part of this Collaborative.

The Commission has also consulted the Medical Board of Queensland in relation to Dr Strahan's care of Mrs Connelly and whether he warranted investigation by the Board. The Commission is required to consult with the relevant registration Board in matters where there may be possible breaches of professional standards. In this case, the Commission drew to the Board's attention all the information and advice we had obtained. Having taken that information into account, the Board advised the Commission that, in its view, the matter does not warrant further action. This is a decision for the Board to make and the Commission is therefore unable to pursue the matter.

I appreciate that you will feel that the Commission has not done enough in relation to this issue, but, unfortunately, there is insufficient basis for me to be able to take any further action other than to recommend to Bundaberg Base Hospital that it continue to implement the changes in relation to its care of cardiac patients. None of the independent advisers contacted by the Commission have been able to state with sufficient confidence that your wife would have survived, even if she had remained in hospital.

Referral for Stress Test Issue

The other issue you raised in your letter was that at 8.30 a.m. on 2 December 2003, you informed Dr Strahan that Mrs Connelly had a pre-booked stress test appointment at 10.20 a.m. that day at a private hospital. You advised the Commission that you explained to Dr Strahan that Mrs Connelly's general practitioner had made this referral and that Dr Strahan then instructed the nurse to have this done straight away. You said you were informed by Dr Strahan that he suspected a blockage in her heart and that this test would identify where the blockage was. She could then be given something for it and be transferred to Brisbane for an operation.

You further advised that at 10.30 a.m. Dr Strahan (?Dr Khan) informed you that the appointment had been reallocated and a new appointment made for 8 December 2003. You said you were subsequently informed ~~that~~ o.k. Mrs Connelly could go home and the results of the stress test would be sent to Bundaberg Base Hospital. When you made enquiries of the private hospital shortly after speaking to Dr Strahan (? Dr Khan), you were informed that they had not been contacted by Bundaberg Base Hospital and that the appointment had been reallocated at 9.30 a.m. You stated that when you asked the nurse why she had not called in

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This is what C said in his letter/ conversation to Leone.

relation to the stress test, she answered in an off-handed manner that it was the doctor's responsibility to do so. I understand you are of the view that even if Mrs Connelly had been diagnosed with a heart attack she could have still have had a stress test without having to undergo a physical exercise. Also, that had she had the stress test, she would have been correctly diagnosed and treated.

I appreciate that you remain critical of the time taken by the staff to contact the nuclear medicine clinic. The key point I need to consider is whether it would have been appropriate for Mrs Connelly to have the test at that time. The Commission sought clarification of this point from the nuclear physician, Dr Muttatamby Vannitamby, who performs the stress tests at the service that Mrs Connelly was due to attend. Dr Vannitamby stated that the referral from the referring doctor is only part of the information he would take into account. He said he would need to do his own assessment of a patient. He also advised that in most cases following a recent infarct, he would prefer to wait 4 to 6 weeks for the heart to recover before performing the stress test because of the high risk involved in the procedure. On this basis, I am unable to say that the test would definitely have been performed had Mrs Connelly's appointment gone ahead on 2 December 2003. This view is reinforced by advice obtained from independent cardiologists who advised the Commission that the referral to the nuclear medicine unit for the stress test was not particularly relevant, as it would only have confirmed what they should have already known i.e. that Mrs Connelly was a high-risk patient. Further, the stress test was contraindicated and could have made the situation worse.

While the actions of the nurse remain in dispute between yourself and the hospital, I have considered Dr Vannitamby's comments and those of the independent cardiologists, and, as noted above, it is not possible to say whether a stress test would or should have been performed on Mrs Connelly had she presented on 2 December 2003.

I have considered your concerns about the manner in which the hospital cared for Mrs Connelly and the hospital's response to those concerns, as well as the independent and third party comments. It is my view that there was a serious breakdown in procedures and that Mrs Connelly should have remained in hospital. As acknowledged by Bundaberg Base Hospital, they failed to take into account the combination of Mrs Connelly's prolonged chest pain, ECG changes, past medical history and most importantly, the significance of the raised troponin levels. The Commission will request the hospital to review the systems in place for care of cardiac patients to ensure they are contemporaneous and remain so plus strongly recommend continued involvement in the Collaborative for Healthcare, Acute Coronary Syndrome project. (There appears to be a transposing of an individual's error in judgement (i.e. Dr Strahan) to suggest the whole Medical Department practiced in a similar manner. In discussions with Dr Miach, Director of Medicine and resident medical staff in this department, it was very clear that they were aware of the significance of the troponin in management of patients with ACS and practiced accordingly.)

refer to 1 adviser who said her raised T levels mandated she be kept in.

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HRC can only be guided by indep. & 3rd party expert advice (lack of systemic approach)

I understand that you will remain unhappy with the Commission's findings and that you believe the matter should have been taken further. I realise that you may not agree with some of the advice the Commission has obtained, but I trust you will understand why the Commission needs to rely upon this. The Commission itself does not have the clinical expertise to reach findings on complex clinical matters and must rely on independent expert medical opinion or third party medical advice. Please be assured that the Commission will follow up to ensure that the procedural changes are occurring at the hospital in relation to the matters raised.

I am sorry that we have been unable to meet all of your expectations. I nevertheless thank you for bringing your complaint to the notice of the Commission.

Yours sincerely

John Cake

040036

File Note

Consumer: Mrs Doreen CONNELLY (DEC'D) Provider: Bundaberg Base Hospital - Mr Peter LECK — Assessment Extension

Encryption Key:
Date Composed: 15/12/2004 04:28 PM Composed By: Karen Harbus/HRC

Body Text:

"Peter Leck" <Peter_Leck@health.qld.gov.au> on 14/12/2004 03:27:53 PM

To: <Karen.Harbus@hrc.qld.gov.au>
cc:

Subject: Re: Mrs Doreen Connelly (Dec'd)

Hi Karen,

Thankyou for this advice.

The sentence you have sought advice about - concerning the sequence of events relating to Pathology - is correct.

I must admit that I remain a little confused as to what will now be included in the letter and what has been deleted or altered. — I have sent him a complete redraft as attached.

There are a couple of other comments I would like to make in relation to the attachment (proposed) letter from 9 December. I hope these comments can be of assistance and am sorry if they are simply repeating some of the changes you are already making:

1) Misdiagnosis Issues - Page 2

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Paragraph 6 refers to pathology updates for Dr Strahan. I believe that this issue is no longer relevant given that it refers to the Pathology booklet — put in by AA

I have re-included all indep. advice which is quite clear. → refer to new para 6 under missing issue.

Paragraph 7 refers to "systems" issues in relation to the discharge of cardiac patients with histories like Mrs Connelly. I'm not sure that it is possible to attribute the situation to "systems" issues vs an apparent misdiagnosis. Perhaps it would be useful to clarify with the cardiologist(s) concerned what precisely they mean by "systems" in this case. Hospital protocols and policy invariably still rely on the individual judgement of a clinician and I'm not familiar with any that would be so prescriptive as to cover all circumstances including this one. The timing of the discharge of a patient is determined by the doctor under which they are admitted, taking into account all known facts. Protocols will usually request that more junior medical staff clarify any issues/concerns with the relevant specialist. However in this instance, the treating medical officer and decision to discharge was made by the consultant.

2) Misdiagnosis Issue - page 3

The second paragraph refers to the Pathology Booklet. I assume this paragraph is to be deleted. Yes - deleted (put in by AA)

Best Wishes

Peter

>>> <Karen.Harbus@hrc.qld.gov.au> 9/12/2004 16:05:47 >>>

Dear Peter

Thanks for your emails and letter. I have noted the comments you made in relation to the Commission's draft letter. I now attach a copy of the revised letter and wish to point out the following alterations:

Page 2 - Misdiagnosis Issue

As per your suggestion, I have altered this paragraph to read that the significance of the raised troponin level was not appreciated.

In relation to the fourth paragraph, second sentence, I have included here: "Dr Keating said that Dr Strahan reported that he attempted to clarify the matter with Queensland Health Pathology Services in Rockhampton prior to Mrs Connelly's discharge but as he did not find the explanation adequate, he subsequently ordered a different blood test (creatine kinase), which was normal, and he discharged Mrs Connelly. Dr Keating advised the Commission that the private pathology provider in Bundaberg had recently installed the same troponin analyser as theirs to offset any future confusion." As the records and correspondence to not make it clear in what sequence the above occurred, would you please advise me if this sentence is correct.

I have deleted the sixth paragraph which makes reference to the Qld Health Pathology booklet. As it does not specify the normal range of troponin levels, I do not believe this paragraph to be particularly relevant. I note your comments that the pathology report of each test does indicate if the results are outside the normal range and noted that "H" for high was recorded next to Mrs Connelly's troponin T readings.

The seventh paragraph on the draft letter has also been deleted as there is no record of this telephone conversation on the electronic file and the officer who wrote this is presently on secondment.

Page 3 - Misdiagnosis Issue

Where you have indicated that the paragraph stating "Dr Keating said that Dr Strahan tried to clarify the measurement scale for troponin testing with another Queensland Health Hospital, but did not receive clarification prior to Mrs Connelly's discharge" would be more accurately expressed as "... Dr Strahan reported that he attempted to clarify the matter with Queensland Health pathology Services in Rockhampton but did not find the explanation adequate.", you will note from my above comments that I have incorporated these comments into the fourth paragraph on page 2.

(See attached file: Connelly draft.doc)

Kind regards

Karen Harbus
Senior Intake Officer

File Note

040036

Consumer: Mrs Doreen CONNELLY (DEC'D) Provider: Bundaberg Base Hospital - Mr Peter LECK

Assessment Extension

Encryption Key:
Date Composed: 09/12/2004 04:06 PM Composed By: Karen Harbus/HRC

Body Text:

Karen Harbus
09/12/2004 04:05 PM

To: Peter_Leck@health.qld.gov.au
cc:

Subject: Mrs Doreen Connelly (Dec'd)

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Connelly draft.doc

Kind regards

Karen Harbus
Senior Intake Officer

Telephone Conversation

040036

Consumer: Mrs Doreen CONNELLY (DEC'D) Provider: Bundaberg Base Hospital - Mr Peter LECK

Assessment Extension

Encryption Key:
Date Composed: 08/12/2004 02:26 PM Composed By: Karen Harbus/HRC

Caller: Mr Leck, P, to HRC

Body Text:
Mr Leck returned my call and I advised him that I had noted the changes he made to the draft letter, had discussed them with my supervisor and HRC agreed to these. I asked him if he had made further enquiries of Dr Sisolo in relation to the passing of the Troponin readings from the lab. He said that yes he had and Dr Sisolo has no clear recollection of the events but he (Dr S) went to the progress notes where he had written at 0555 on 01/12/03 that Troponin was 0.52 with an arrow to indicate that it was high. Underneath this Dr Sisolo had written that he had discussed this with Dr Strahan. Mr Leck said that Dr Sisolo had no clear memory of the discussions and he was relying on the notes. Thanked him.

Telephone Conversation

Consumer: Mrs Doreen CONNELLY (DEC'D) Provider: Bundaberg Base Hospital - Mr Peter LECK

Assessment Extension

Encryption Key:
Date Composed: 07/12/2004 02:47 PM Composed By: Karen Harbus/HRC

Caller: HRC to Mr Peter Leck, District Manager, P

Body Text:
4152 1222. Mr Leck not available today. Left a message for him to please return my call. [I rang Mr Leck in order to acknowledge receipt of his letter and memorandum from Qld Health Pathology Services Director, Mr Michael Whiley, which enclosed copies of pathology results and "screen dumps" which clearly showed that Mrs Connelly's troponin T levels had been high ("H"). In the memo, the Director explained that results were phoned through to Dr Sisolo on the ward at 5.00 a.m. on 01/12/03 and supporting documentation was enclosed. In his memo, Mr Whiley stated that it appeared from their (pathology) records that all the relevant information (result, abnormality of this and telephone contact) was given to Dr Sisolo. He said that given Dr Strahan's concerns (i.e. did not realise the significance of raised troponin levels) and given the information the laboratory gave to Dr Sisolo, one possible explanation is that all of this information may not have been passed on to Dr Strahan in its entirety.] I was going to ask Mr Leck about whether he had explored this issue any further i.e. what does Dr Sisolo recall being told by the lab and what does Dr Sisolo recall relaying to Dr Strahan? Did Dr Sisolo jot down the results or go by memory?

In relation to the other suggested changes Mr Leck made, I was going to advise him that I agreed with him but would discuss with my supervisor.

Email from Qld. Pathology to Peter Leck

Path. Report re [illegible]
- i.e. Tpn = High
HRC has always known this!

Queensland Health
Pathology Service
Queensland Government

MEMORANDUM

To: Peter Leck - District Manager Bundaberg

Copies To: Peter Lewis-Hughes - Executive Director QHPSS

From: Michael Whiley
Director - QHPS

Tel No: 07 3636 8300 (RBH)

Fax No: 07 3636 1392

File Ref:

Date: 5 Dec 2004

Subject: Troponin T results and Mrs Connelly

In response to your email request I provide the following information for your use:

1] It was in fact Dr Lewis-Hughes and Dr Francis who discussed this case with the Health Rights Commission. I was away on Leave, hence the delay in replying to you whilst I gathered information.

2] The Reports issued by QHPS clearly show the troponin used was Tropoinin T (TnT) and gives the reference range relevant for this assay (<0.03 ug/L). Whenever these results are looked at in Auslab both results on this patient also had the letter H next to the result to indicate this result was high and supportive of a myocardial necrotic event (eg Acute Myocardial Infarction). They display in **BOLD** printing on cumulative reports.

3] The result was phoned by the lab staff to ward medical staff member Dr Sisolo (at 5am) and included the information that this was a high result.
Copies of the Cumulative Result and Screen Dumps of the individual results and phone log are attached

4] The handbook covers the tests available and refers Doctors to reports and their local labs for interpretation and ranges.

I hope this assists you and from this it appears from our records that all the relevant information {result, abnormality of the result and telephone contact} was given to the Dr Sisolo. Given your VMO's concerns "The VMO who treated her says he did not realise the significance of her raised troponin levels" and given the information the Lab gave Dr Sisolo one possible explanation (which would need exploring further) is that all of this information may not have been passed on him in it's entirety.

QUEENSLAND HEALTH PATHOLOGY AND SCIENTIFIC SERVICES

QHPS-Bundaberg Hospital
P.O.Box 34
Bundaberg, QLD, QLD 4670
ph 07-41502530
fax 07-41512539

Patient Location	10 - Medical (BNH)	**UR No**	BN059241	IS	4
Consultant	Strahan, T. Martin(BNH)	**Name**	CONNELLY		
This Report To	**Dr Lipson Sisiolo**	**Given Name**	Doreen	**Sex**	F
	Bundaberg Hosp	**DOB**	22-Apr-1934	**Age**	69 years
	Bourbong St	**Patient Address**			
	Bundaberg Qld 4670				

GENERAL

Time Collected	03:45	05:00	08:45	12:30	07:40		
Date Collected	19 Sep	01 Dec	01 Dec	01 Dec	02 Dec		
Time Registered	04:26	05:21	09:08	14:05	08:56		
Date Registered	19 Sep	01 Dec	01 Dec	01 Dec	02 Dec		
Year	2002	2003	2003	2003	2003		
Lab No	107679131	149221954	149221255	149221062	149286065		
Specimen Type	Blood	Blood	Blood	Blood	Blood		
Sodium	**133**	139				mmol/L	(135 - 145)
Potassium	4.5	4.2				mmol/L	(3.2 - 4.5)
Chloride	106	107				mmol/L	(100 - 110)
Bicarbonate	**21**	25				mmol/L	(22 - 33)
Anion Gap	6	7				mmol/L	(4 - 13)
Osmolality (Calculated)	**269**	277				mmol/kg	(270 - 290)
Glucose	6.8	6.1				mmol/L	(3.0 - 7.8)
						(Fasting	3.0 - 6.0)
Urea	5.5	3.6				mmol/L	(3.0 - 8.0)
Creatinine	0.086	0.091				mmol/L	(0.050 - 0.100)
Urea/Creat	64	**39**					(40 - 100)
Urate	0.29					mmol/L	(0.15 - 0.45)
Protein (Total)	74	65				g/L	(62 - 83)
Albumin	41	36				g/L	(33 - 47)
Globulin	33	29				g/L	(25 - 45)
Bilirubin (Total)	< 4	12				umol/L	(< 20)
Alkaline Phosphatase	109	90				U/L	(30 - 120)
Gamma-GT	18	19				U/L	(< 50)
Alanine Transaminase	20	28				U/L	(< 40)
Aspartate Transaminase	20	23				U/L	(< 35)
Lactate Dehydrogenase	**289**					U/L	(110 - 250)
Creatine Kinase	**210**	121	122	128	123	U/L	(< 160)
cTroponin T	ND	**0.52**		**0.69**		ug/L	(< 0.03)
Calcium	2.31					mmol/L	(2.15 - 2.60)
Calcium (Alb. Corr.)	2.29					mmol/L	(2.15 - 2.60)
Phosphate	**0.57**					mmol/L	(0.70 - 1.40)

Note: ND = Not detected.

CHEMICAL PATHOLOGY

Comments

Lab No **149221954**
05:00 01-Dec-03 Results phoned to ward

Dr H Krause
Director of Pathology
Tel.(07)4920 7301

Please discard any previous **CHEMICAL PATHOLOGY GENERAL** report of the same page number printed before : 09:48 06 Dec 2004

Copy sent to: 10 - Medical (BNH)

[redacted] MR 23

0461

Verified

Lab 14922-1954	Ur BN059241	Name CONNELLY Doreen	Sex F
Dob 22-Apr-1934 Wd DEM~BNH		Dr Sisiolo, Lipso*	c05:00 01-Dec-03

Specimen type	Blood			Protein	66	g/L	(62 - 80)	Chest pain
Sodium	139	mmol/L	(135 - 145)	Albumin	36	g/L	(33 - 47)	
Potassium	4.2	mmol/L	(3.6 - 4.5)	Globulin	29	g/L	(25 - 45)	
Chloride	107	mmol/L	(100 - 110)	Bilirubin	12	umol/L	(< 20)	
Bicarb.	25	mmol/L	(22 - 30)	ALP	90	U/L	(30 - 120)	
Anion Gap	7	mmol/L	(4 - 16)	Gamma GT	19	U/L	(< 50)	
CSM(Calc)	277	mmol/kg	(270 - 295)	ALT	28	U/L	(< 40)	
Glucose	6.1	mmol/L	(3.6 - 7.8)	AST	22	U/L	(< 35)	
Fasting FR			(3.6 - 6.0)	CK	121	U/L	(< 160)	
Urea	3.6	mmol/L	(3.6 - 8.0)	cTnT	0.52	Hug/L	(< 0.03)	
Creatinine	0.09	mmol/L	(0.060 - 0.100)					
eGFR Creat.	59	L	(40 - 100)					

Comment: Age:65 years Comp.Val: Yes

Results phoned to ward

Notes Alert

Phoned 5.49pm

Screen Dump

1st Result

Notes

Lab 14922-1954	Ur BN059241	Name CONNELLY Doreen	Sex F
Dob 22-Apr-1934 Wd DEM~BNH		Dr Sisiolo, Lipso*	c05:00 01-Dec-03

Time/Date	User	D	Specimen Notes
05:49 01-Dec-03	ckl1	B	tnt phoned to DR Sisiolo
05:48 01-Dec-03	ckl1	A	coag added per phone request by Dr Sisiolo. Form to follow

Clinical Notes
Chest pain

F5 Clinical Notes F6 Specimen Notes F7 UR Notes
F8 Set Dept Spec Notes SF7 Set Dept UR Notes

Screen Dump
Phone Log

Validated

Lab 14922-1062	Ur BN059241	Name CONNELLY Doreen	Sex F
Dob 22-Apr-1934	Wd 10-BNH	Dr Sisiolo, Lipso*	c12:30 01-Dec-03

Specimen type Blood

Chest Pain
Current medication - lasix

CK	128	U/L	(< 160)
cTnT	0.69 H	ug/L	(< 0.03)

Comment: Age:69 years

Comp.Val: Yes

■ Notes ■ Audit

Screen Dump Second Result

This is the letter to Peter Leck.

040036/kh

4 November 2004

Private & Confidential

Mr George Connelly

Dear Mr Connelly

I refer to your complaint about a health service your late wife, Mrs Doreen Connelly, received from Bundaberg Base Hospital on 2 December 2003. At the outset, I wish to convey my sincere condolences to you for the loss of your wife.

As you are aware, the Commission has been assessing the complaint to determine whether the health service provided to Mrs Connelly was reasonable and whether any further action may be required.

I understand that Mrs Connelly, who had a history of ischaemic heart disease, woke at 0330 hours on 1 December 2003 suffering with chest pain. An ambulance was called and transported her to Bundaberg Base Hospital at 0446 hours. Ambulance records state that on arrival at the scene, Mrs Connelly's pain had ceased in the chest but she still had pain in her back.

At the hospital, the duty medical practitioner noted Mrs Connelly's past history of acute myocardial infarction and hypothyroidism. She was examined, her vital signs monitored and no abnormality was detected. Various tests were performed which included serial electrocardiographs (ECGs) and while the chest x-ray was normal, blood tests showed raised levels of troponin[1]. Mrs Connelly was admitted to a general ward and later that day was reviewed by the specialist medical team who diagnosed her as having unstable angina. Aspirin, lipitor and lasix were added to her medication regime and she was discharged home at 1430 hours on 2 December 2003.

I understand that before Mrs Connelly was discharged, you explained to staff that she had been referred by her general practitioner the previous week for a stress (sestamibi) test to be performed by North Coast Nuclear Medicine at Mater Hospital that day at 10.20 a.m. The hospital's clinical plan for Mrs Connelly had been to take further blood tests and, if normal, the stress test would go ahead as planned. Following the appropriate blood tests and review of those tests, she was discharged with arrangements to transfer Mrs Connelly for her stress test. Before Mrs Connelly left the hospital the nursing staff member contacted North Coast Nuclear Medicine, and was told that the appointment had been reallocated and a new appointment was made for six days time. Tragically, your wife died in the early hours of the morning of 3 December 2003, at home. Her death certificate indicated that she died from a cardiac arrest following a myocardial infarction.

[1] An independent biochemist explained that troponins are muscle proteins found in the blood, which can be tested and analysed, following suspected heart muscle damage. High readings of troponin occur following cardiac damage.

Complaint Issues

I understand that your complaint issues are:

- Mrs Connelly was misdiagnosed and had she been correctly diagnosed and given appropriate treatment she would not have died; and
- Mrs Connelly should have attended the stress test, and if she had, she would have been correctly diagnosed and treated.

Misdiagnosis Issue

You stated that you were later informed that Mrs Connelly's past cardiac history and her elevated troponin levels were not taken into account when the decision was made to discharge her. You stated that you believed that had Mrs Connelly been correctly diagnosed and treated, she would not have died.

Mr Peter Leck, District Manager, Bundaberg District Health Service advised the Commission that the hospital had conducted a review of Mrs Connelly's care. The review confirmed that the combination of Mrs Connelly's past history, prolonged chest pain, ECG changes and raised troponin values indicated that she should have been diagnosed with Acute Coronary Syndrome and remained in hospital for ongoing observation. Mr Leck offered his sincere apologies to you for this failure.

In a further letter to the Commission, Dr Darren Keating, Director of Medical Services, explained that the failure to detect the raised troponin level was because the hospital used a different measurement from that used in one of the town's private laboratories. Dr Martin Strahan, general physician who attended to Mrs Connelly, was a visiting consultant who also worked in the private sector. It was explained that Dr Strahan did not appreciate the significance of your wife's troponin measurement because of the different measurement systems being used in the public and private health sectors leading to potential discrepancy between troponin values for the same patient. This discrepancy contributed to Dr Strahan placing limited significance on the test results at Bundaberg Base Hospital.

Dr Keating advised that Dr Strahan's reliance on the private sector method was based on his belief that the public sector method was inaccurate and possibly inferior. Subsequently, he ordered a different blood test, which was normal, and he discharged Mrs Connelly. Dr Keating recently advised the Commission that the private pathology provider in Bundaberg had recently installed the same troponin analyser as theirs to offset any future confusion.

Dr Keating also advised that the hospital has begun involvement with the Collaborative for Healthcare Improvement, Acute Coronary Syndrome, which provides evidence based guidelines and systematic evaluation of the treatment for this disorder in their hospital. The results will be compared on a state-wide basis. He explained that since Mrs Connelly's death there had been an education session for all medical staff involved in the care of cardiac patients. There were also continuing education sessions for senior medical staff on the specific topic of Acute Coronary Syndrome and the management of patients with raised troponin measurements. Dr Keating also advised that Dr Strahan had since undertaken further study, attended a cardiology conference and sought ongoing advice from cardiology peers.

The Commission sought comment from Dr Peter Hughes, State Manager of Queensland Health's Pathology services regarding the uniformity of blood testing in Queensland Health hospitals. Dr Hughes advised that each Queensland Health hospital provides medical staff (visiting or salaried) with a booklet informing them of the normal ranges of all pathology tests undertaken at the hospital where they work. Dr Hughes said he was concerned that a doctor working in a Queensland Health hospital was not familiar with the specific pathology measurement ranges for that hospital. Dr Hughes also advised that when a result is abnormal the pathology department usually alerts the doctor to the abnormality.

The Commission then asked Dr Keating if Dr Strahan had been provided with specific information about pathology tests at their hospital. Dr Keating advised that Dr Strahan had worked as a salaried doctor at

the hospital before he became a visiting medical officer and that perhaps Dr Strahan may have missed out on receiving the updates on pathology information. Dr Keating said that Dr Strahan tried to clarify the measurement scale for troponin testing with another Queensland Health hospital, but did not receive any clarification prior to Mrs Connelly's discharge.

Independent advice obtained by the Commission from well-qualified cardiologists confirmed that Mrs Connelly should not have been sent home. An independent Deputy Medical Director of a cardiology program at a public hospital stated that whether troponin was positive or negative may not be the issue and explained that it was necessary to look at the systems in place. He stated that people with cardiac histories should be admitted regardless of the troponin level. Further independent advice said that had Mrs Connelly been kept in hospital, even in the Coronary Care Unit, there were no guarantees that she would have survived her cardiac arrest.

There is no doubt that Mrs Connelly should not been discharged. The Commission has requested that the District ensure that all medical personnel be provided with the current pathology information booklet and ensure that doctors new to the hospital are provided with the appropriate information as part of their orientation, regardless of whether they have previously worked at the hospital or not.

The Commission has also consulted the Medical Board of Queensland in relation to Dr Strahan's care of Mrs Connelly and whether he warranted investigation by the Board. The Commission is required to consult with the relevant registration Board in matters where there may be possible breaches of professional standards. In this case, the Commission drew to the Board's attention all the information and advice we had obtained. Having taken that information into account, the Board advised the Commission that, in its view, the matter does not warrant further action. This is a decision for the Board to make and the Commission is therefore unable to pursue the matter.

I appreciate that you will feel that the Commission has not done enough in relation to this issue, but, unfortunately, there is insufficient basis for me to be able to take any further action other than to recommend to Bundaberg Base Hospital that it continue to implement the changes in relation to its care of cardiac patients. None of the independent advisers contacted by the Commission have been able to state with sufficient confidence that your wife would have survived, even if she had remained in hospital.

Referral for Stress Test Issue

The other issue you raised in your letter was that at 8.30 a.m. on 2 December 2003, you informed Dr Strahan that Mrs Connelly had a pre-booked stress test appointment at 10.20 a.m. that day at a private hospital. You advised the Commission that you explained to Dr Strahan that Mrs Connelly's general practitioner had made this referral and that Dr Strahan then instructed the nurse to have this done straight away. You said you were informed by Dr Strahan that he suspected a blockage in her heart and that this test would identify where the blockage was. She could then be given something for it and be transferred to Brisbane for an operation.

You further advised that at 10.30 a.m. Dr Strahan informed you that the appointment had been reallocated and a new appointment made for 8 December 2003. You said you were subsequently informed that Mrs Connelly could go home and the results of the stress test would be sent to Bundaberg Base Hospital. When you made enquiries of the private hospital shortly after speaking to Dr Strahan, you were informed that they had not been contacted by Bundaberg Base Hospital and that the appointment had been reallocated at 9.30 a.m. You stated that when you asked the nurse why she had not called in relation to the stress test, she answered in an off-handed manner that it was the doctor's responsibility to do so. I understand you are of the view that even if Mrs Connelly had been diagnosed with a heart attack she could have still had a stress test without having to undergo a physical exercise. Also, that had she had the stress test, she would have been correctly diagnosed and treated.

I appreciate that you remain critical of the time taken by the staff to contact the nuclear medicine clinic. The key point I need to consider is whether it would have been appropriate for Mrs Connelly to have the

test at that time. The Commission sought clarification of this point from the nuclear physician, Dr Muttatamby Vannitamby, who performs the stress tests at the service that Mrs Connelly was due to attend. Dr Vannitamby stated that the referral from the referring doctor is only part of the information he would take into account. He said he would need to do his own assessment of a patient. He also advised that in most cases following a recent infarct, he would prefer to wait 4 to 6 weeks for the heart to recover before performing the stress test because of the high risk involved in the procedure. On this basis, I am unable to say that the test would definitely have been performed had Mrs Connelly's appointment gone ahead on 2 December 2003. This view is reinforced by advice obtained from independent cardiologists who advised the Commission that the referral to the nuclear medicine unit for the stress test was not particularly relevant, as it would only have confirmed what they should have already known i.e. that Mrs Connelly was a high-risk patient. Further, the stress test was contraindicated and could have made the situation worse.

While the actions of the nurse remain in dispute between yourself and the hospital, I have considered Dr Vannitamby's comments and those of the independent cardiologists, and, as noted above, it is not possible to say whether a stress test would or should have been performed on Mrs Connelly had she presented on 2 December 2003.

I have considered your concerns about the manner in which the hospital cared for Mrs Connelly and the hospital's response to those concerns, as well as the independent and third party comments. It is my view that there was a serious breakdown in procedures and that Mrs Connelly should have remained in hospital. As acknowledged by Bundaberg Base Hospital, they failed to take into account Mrs Connelly's prolonged chest pain, ECG changes, history and raised troponin levels. The Commission will advise the hospital of the importance of taking a systemic approach to the care of cardiac patients and of its continued involvement in the Collaborative for Healthcare, Acute Coronary Syndrome project.

I understand that you will remain unhappy with the Commission's findings and that you believe the matter should have been taken further. I realise that you may not agree with some of the advice the Commission has obtained, but I trust you will understand why the Commission needs to rely upon this. The Commission itself does not have the clinical expertise to reach findings on complex clinical matters and must rely on independent expert medical opinion or third party medical advice. Please be assured that the Commission will follow up to ensure that the procedural changes are occurring at the hospital in relation to the matters raised.

I am sorry that we have been unable to meet all of your expectations. I nevertheless thank you for bringing your complaint to the notice of the Commission.

Yours sincerely

Annette Anning
Acting Manager Complaints

Queensland Government

Queensland **Health**

Executive Services
Bundaberg Health Service District

Enquiries to: Peter Leck, District Manager
Telephone: 07 41502020
Facsimile: 07 41502029
File Ref: PUBRE/1404/017

Ms Karen Harbus
Senior Intake Officer
Health Rights Commission
GPO Box 3089
Brisbane Qld 4001

Dear Ms Harbus

Re: Mrs Doreen Connelly

Thank you for the opportunity to comment on the draft report.

The following comments will I hope be of assistance:

Page 2 – Misdiagnosis Issue

The third paragraph states that there was a failure to detect a raised troponin level. Technically, perhaps this should read that the significance of the raised troponin level was not appreciated. It was detected but the significance was misunderstood.

The sixth paragraph refers to the Queensland Health Pathology booklet. This booklet is available to all staff – not only in hard copy but also on the intranet website. The booklet does not specify the normal range of troponin levels. However, the pathology report on each test does indicate if the results are outside normal range. A copy of advice from Queensland Health Pathology and a copy of Mrs Connelly's pathology test results are attached for information.

Page 3 – Misdiagnosis Issue

The first paragraph states "Dr Keating said that Dr Strahan tried to clarify the measurement scale for troponin testing with another Queensland Health Hospital, but did not receive clarification prior to Mrs Connelly's discharge."

A more accurate statement would be that Dr Strahan reported that he attempted to clarify the matter with Queensland Health Pathology Services in Rockhampton but did not find the explanation adequate.

The third paragraph concerning access to the Pathology booklet might be reconsidered, given that the Pathology booklet does not describe the normal values/ranges of pathology tests. It describes the tests that are available and their application. Resident Medical staff are provided access to this information as part of their orientation. Pathology tests outside normal range are highlighted in each pathology report.

I trust that this information is able to be of assistance.

Yours sincerely

Peter Leck
District Manager
06/12/2004

Queensland Health
Pathology Service
Queensland Government

MEMORANDUM

To: Peter Leck - District Manager Bundaberg

Copies To: Peter Lewis-Hughes - Executive Director QHPSS

From: Michael Whiley
Director - QHPS

Tel No: 07 3636 8300 (RBH)

Fax No: 07 3636 1392

File Ref:

Date: 5 Dec 2004

Subject: Troponin T results and Mrs Connelly

In response to your email request I provide the following information for your use:

1] It was in fact Dr Lewis-Hughes and Dr Francis who discussed this case with the Health Rights Commission. I was away on Leave, hence the delay in replying to you whilst I gathered information.

2] The Reports issued by QHPS clearly show the troponin used was Tropoinin T (TnT) and gives the reference range relevant for this assay (<0.03 ug/L). Whenever these results are looked at in Auslab both results on this patient also had the letter H next to the result to indicate this result was high and supportive of a myocardial necrotic event (eg Acute Myocardial Infarction). They display in **BOLD** printing on cumulative reports.

3] The result was phoned by the lab staff to ward medical staff member Dr Sisolo (at 5am) and included the information that this was a high result.
Copies of the Cumulative Result and Screen Dumps of the individual results and phone log are attached

4] The handbook covers the tests available and refers Doctors to reports and their local labs for interpretation and ranges.

I hope this assists you and from this it appears from our records that all the relevant information {result, abnormality of the result and telephone contact} was given to the Dr Sisolo. Given your VMO's concerns "The VMO who treated her says he did not realise the significance of her raised troponin levels" and given the information the Lab gave Dr Sisolo one possible explanation (which would need exploring further) is that all of this information may not have been passed on him in it's entirety.

QUEENSLAND HEALTH PATHOLOGY AND SCIENTIFIC SERVICES

QHPS, Bundaberg Hospital
P.O.Box 34
Bundaberg, QLD, QLD 4670
ph 07-41502530
fax 07-41512539

Patient Location	10 - Medical (BNH)	UR No	BN059241	IS 4	GENERAL
Consultant	Strahan, T. Martin(BNH)	Name	CONNELLY		
This Report To	Dr Lipson Sisiolo Bundaberg Hosp Bourbong St Bundaberg Qld 4670	Given Name	Doreen	Sex F	
		DOB	22-Apr-1934	Age 69 years	
		Patient Address			

						Units	Ref Range
Time Collected	03:45	05:00	08:45	12:30	07:40		
Date Collected	19 Sep	01 Dec	01 Dec	01 Dec	02 Dec		
Time Registered	04:26	05:21	09:08	14:05	08:56		
Date Registered	19 Sep	01 Dec	01 Dec	01 Dec	02 Dec		
Year	2002	2003	2003	2003	2003		
Lab No	107679131	149221954	149221255	149221062	149286065		
Specimen Type	Blood	Blood	Blood	Blood	Blood		
Sodium	**133**	139				mmol/L	(135 - 145)
Potassium	4.5	4.2				mmol/L	(3.2 - 4.5)
Chloride	106	107				mmol/L	(100 - 110)
Bicarbonate	**21**	25				mmol/L	(22 - 33)
Anion Gap	6	7				mmol/L	(4 - 13)
Osmolality (Calculated)	**269**	277				mmol/kg	(270 - 290)
Glucose	6.8	6.1				mmol/L	(3.0 - 7.8)
						(Fasting	3.0 - 6.0)
Urea	5.5	3.6				mmol/L	(3.0 - 8.0)
Creatinine	0.086	0.091				mmol/L	(0.050 - 0.100)
Urea/Creat	64	**39**					(40 - 100)
Urate	0.29					mmol/L	(0.15 - 0.45)
Protein (Total)	74	65				g/L	(62 - 83)
Albumin	41	36				g/L	(33 - 47)
Globulin	33	29				g/L	(25 - 45)
Bilirubin (Total)	< 4	12				umol/L	(< 20)
Alkaline Phosphatase	109	90				U/L	(30 - 120)
Gamma-GT	18	19				U/L	(< 50)
Alanine Transaminase	20	28				U/L	(< 40)
Aspartate Transaminase	20	23				U/L	(< 35)
Lactate Dehydrogenase	**289**					U/L	(110 - 250)
Creatine Kinase	**210**	121	122	128	123	U/L	(< 160)
cTroponin T	ND	**0.52**		**0.69**		ug/L	(< 0.03)
Calcium	2.31					mmol/L	(2.15 - 2.60)
Calcium (Alb. Corr.)	2.29					mmol/L	(2.15 - 2.60)
Phosphate	**0.57**					mmol/L	(0.70 - 1.40)

Note: ND = Not detected.

CHEMICAL PATHOLOGY

Comments

Lab No 149221954
05:00 01-Dec-03 Results phoned to ward

Dr H Krause
Director of Pathology
Tel.(07)4920 7301

Please discard any previous CHEMICAL PATHOLOGY GENERAL report of the same page number printed before : 09:48 06 Dec 2004

Copy sent to: 10 - Medical (BNH)

MR 23

0450

\aTicetec

Lab 14922-1954	Ur BN059241	Name CONNELLY Doreen	Sex F
Dob 22-Apr-1934 Wd DEM-BNH		Dr Sisiolo, Lipso*	c05:00 01-Dec-03

Specimen type	Blood		Protein	69	g/L	(62 - 83)	Chest pain
Sodium	135	mmol/L (135 - 145)	Albumin	36	g/L	(33 - 47)	
Potassium	4.2	mmol/L (3.2 - 4.5)	Globulin	29	g/L	(25 - 45)	
Chloride	107	mmol/L (100 - 110)	Bilirubin	12	umol/L	(< 20)	
Bicarb.	25	mmol/L (22 - 30)	ALP	90	U/L	(30 - 120)	
Anion Gap	7	mmol/L (4 - 10)	Gamma GT	15	U/L	(< 50)	
CSN(Calc)	277	mmol/kg (270 - 295)	ALT	28	U/L	(< 40)	
Glucose	6.1	mmol/L (3.0 - 7.8)	AST	22	U/L	(< 35)	
Fasting FR		(3.0 - 6.0)	CK	121	U/L	(< 160)	
Urea	3.6	mmol/L (3.0 - 8.0)	cTnT	0.52	ug/L	(< 0.03)	
Creatinine	0.09	mmol/L (0.060 - 0.100)					
eGFR Creat.	59	L (40 - 100)					

Comment: Age:69 years Comp.Val: Yes

Results phoned to ward

Notes Alert

Phoned 5.49pm

Screen Dump

1st Result

Notes

Lab 14922-1954	Ur BN059241	Name CONNELLY Doreen	Sex F
Dob 22-Apr-1934 Wd DEM~BNH		Dr Sisiolo, Lipso*	c05:00 01-Dec-03

Time/Date	User	D	Specimen Notes
05:49 01-Dec-03	ckll	B	tnt phoned to DR Sisiolo
05:48 01-Dec-03	ckll	A	coag added per phone request by Dr Sisiolo. Form to follow

Clinical Notes
Chest pain

F5 Clinical Notes F6 Specimen Notes F7 UR Notes
F8 Set Dept Spec Notes SF7 Set Dept UR Notes

Screen Dump
Phone Log

Validated

Lab 14922-1062	Ur BN059241	Name CONNELLY Doreen	Sex F
Dob 22-Apr-1934	Wd 10-BNH	Dr Sisiolo, Lipso*	c12:30 01-Dec-03

Specimen type Blood

Chest Pain
Current medication - lasix

CK	128	U/L	(< 160)
cTnT	0.69 H	ug/L	(< 0.03)

Comp.Val: Yes

Comment: Age:69 years

■ Notes ■ Audit

Screen Dump Second Result

Emailed to Karen Harbus 2:45pm 6/12/04
Original Posted 6/12/04

FILE COPY

Health Rights Commission
KH
07 DEC 2004
040036
RECEIVED

Executive Services
Bundaberg Health Service District

Enquiries to: Peter Leck, District Manager
Telephone: 07 41502020
Facsimile: 07 41502029
File Ref: FUBRE/1404/017

Ms Karen Harbus
Senior Intake Officer
Health Rights Commission
GPO Box 3089
Brisbane Qld 4001

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Bundaberg Base Hospital

Postal
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06/12/2004

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Queensland Health
Pathology Service
Queensland Government

MEMORANDUM

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From: Michael Whiley
Director - QHPS

Tel No: 07 3636 8300 (RBH)

Fax No: 07 3636 1392

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CHEMICAL PATHOLOGY

Comments

Lab No **149221954**
05:00 01-Dec-03 Results phoned to ward

Dr H Krause
Director of Pathology
Tel.(07)4920 7301

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Copy sent to: 10 - Medical (BNH)

MR 23

0443

Validated

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Bicarb.	25	mmol/L	(22 - 33)	Bilirubin	12	umol/L	(< 20)
Anion Gap	7	mmol/L	(4 - 16)	ALP	90	U/L	(30 - 120)
CSM(Calc)	277	mmol/kg	(270 - 290)	Gamma GT	19	U/L	(< 50)
Glucose	6.1	mmol/L	(3.0 - 7.8)	ALT	28	U/L	(< 40)
Fasting FR			(3.0 - 6.0)	AST	22	U/L	(< 35)
Urea	3.6	mmol/L	(3.0 - 8.0)	CK	121	U/L	(< 160)
Creatinine	0.59	mmol/L	(0.060 - 0.100)	cTnT	0.02	ug/L	(< 0.03)
Urea/Creat.	39 L		(40 - 100)				

Comment: Age:69 years

Comp.Val: Yes

Results phoned to ward

Notes Alert

Phoned S.49a

Screen Dump

1st Result

Lab 14922-1954	Ur BN059241	Name CONNELLY Doreen	Sex F
Dob 22-Apr-1934 Wd DEM~BNH		Dr Sisiolo, Lipso*	c05:00 01-Dec-03

Time/Date	User	D	Specimen Notes
05:49 01-Dec-03	ckl1	B	tnt phoned to DR Sisiolo
05:48 01-Dec-03	ckl1	A	coag added per phone request by Dr Sisiolo. Form to follow

Clinical Notes
Chest pain

F5 Clinical Notes F6 Specimen Notes F7 UR Notes
F8 Set Dept Spec Notes SF7 Set Dept UR Notes

Screen Dump
Phone Log

Validated

Lab 14922-1062	Ur BN059241	Name CONNELLY Doreen	Sex F
Dob 22-Apr-1934	Wd 10-BNH	Dr Sisiolo, Lipso*	c12:30 01-Dec-03

Specimen type Blood

Chest Pain
Current medication - lasix

CK	128	U/L	(< 160)
cTnT	0.69 H	ug/L	(< 0.03)

Comment: Age:69 years

Comp.Val: Yes

■ Notes ■ Audit

Screen Dump Second Result

Queensland Health
Pathology Service
Queensland Government

MEMORANDUM

To: Peter Leck - District Manager Bundaberg

COPY

Copies To: Peter Lewis-Hughes - Executive Director QHPSS

From: Michael Whiley
Director - QHPS

Tel No: 07 3636 8300 (RBH)

Fax No: 07 3636 1392

File Ref:

Date: 5 Dec 2004

Subject: Troponin T results and Mrs Connelly

In response to your email request I provide the following information for your use:

1] It was in fact Dr Lewis-Hughes and Dr Francis who discussed this case with the Health Rights Commission. I was away on Leave, hence the delay in replying to you whilst I gathered information.

2] The Reports issued by QHPS clearly show the troponin used was Tropoinin T (TnT) and gives the reference range relevant for this assay (<0.03 ug/L). Whenever these results are looked at in Auslab both results on this patient also had the letter H next to the result to indicate this result was high and supportive of a myocardial necrotic event (eg Acute Myocardial Infarction). They display in **BOLD** printing on cumulative reports.

3] The result was phoned by the lab staff to ward medical staff member Dr Sisolo (at 5am) and included the information that this was a high result.
Copies of the Cumulative Result and Screen Dumps of the individual results and phone log are attached

4] The handbook covers the tests available and refers Doctors to reports and their local labs for interpretation and ranges.

I hope this assists you and from this it appears from our records that all the relevant information {result, abnormality of the result and telephone contact} was given to the Dr Sisolo. Given your VMO's concerns "The VMO who treated her says he did not realise the significance of her raised troponin levels" and given the information the Lab gave Dr Sisolo one possible explanation (which would need exploring further) is that all of this information may not have been passed on him in it's entirety.

QUEENSLAND HEALTH PATHOLOGY AND SCIENTIFIC SERVICES

DHPS - Bundaberg Hospital
P.O Box 34, QLD, QLD 4670
Bundaberg, QLD, QLD 4670
ph 07-41512539
fax 07-41512539

Patient Location	10 - Medical (BNH)	UR No	BN059241 IS 4
Consultant	Strahan, T. Martin(BNH)	Name	CONNELLY
This Report To	Dr Lipson Sisiolo Bundaberg Hosp Bourbong St Bundaberg Qld 4670	Given Name	Doreen Sex F
		DOB	22-Apr-1934 Age 69 years
		Patient Address	

GENERAL

Time Collected	03:45	08:00	08:46	12:30	07:40		
Date Collected	19 Sep	01 Dec	01 Dec	01 Dec	02 Dec		
Time Registered	04:26	05:21	09:08	14:05	08:56		
Date Registered	19 Sep	01 Dec	01 Dec	01 Dec	02 Dec		
Year	2002	2003	2003	2003	2003		
Lab No	107679131	149221864	149221258	149221062	149288065		
Specimen Type	Blood	Blood	Blood	Blood	Blood	Units	Ref Range
Sodium	133	139				mmol/L	(135 - 145)
Potassium	4.5	4.2				mmol/L	(3.2 - 4.5)
Chloride	106	107				mmol/L	(100 - 110)
Bicarbonate	21	25				mmol/L	(22 - 33)
Anion Gap	6	7				mmol/L	(4 - 13)
Osmolality (Calculated)	269	277				mmol/kg	(270 - 290)
Glucose	6.8	6.1				mmol/L	(3.0 - 7.8)
						(Fasting	3.0 - 6.0)
Urea	5.5	3.6				mmol/L	(3.0 - 8.0)
Creatinine	0.086	0.091				mmol/L	(0.050 - 0.100)
Urea/Creat	64	39					(40 - 100)
Urate	0.29					mmol/L	(0.15 - 0.45)
Protein (Total)	74	65				g/L	(62 - 83)
Albumin	41	36				g/L	(33 - 47)
Globulin	33	29				g/L	(25 - 45)
Bilirubin (Total)	< 4	12				umol/L	(< 20)
Alkaline Phosphatase	109	90				U/L	(30 - 120)
Gamma-GT	18	19				U/L	(< 50)
Alanine Transaminase	20	28				U/L	(< 40)
Aspartate Transaminase	20	23				U/L	(< 35)
Lactate Dehydrogenase	289					U/L	(110 - 250)
Creatine Kinase	210	121	122	128	123	U/L	(< 180)
cTroponin T	ND	0.52		0.69		ug/L	(< 0.03)
Calcium	2.31					mmol/L	(2.15 - 2.60)
Calcium (Alb. Corr.)	2.29					mmol/L	(2.15 - 2.60)
Phosphate	0.57					mmol/L	(0.70 - 1.40)

Note: ND = Not detected.

CHEMICAL PATHOLOGY

Comments

Lab No 149221864
05:00 01-Dec-03 Results phoned to ward

Dr H Krause
Director of Pathology
Tel (07)4320 7301

Please discard any previous CHEMICAL PATHOLOGY GENERAL report of the same page number printed before : 09:48 06 Dec 2004

Page 1

Copy sent to: 10 - Medical (BNH)

MR 23

4 November 2004

Private & Confidential

Mr George Connelly

Dear Mr Connelly

I refer to your complaint about a health service your late wife, Mrs Doreen Connelly, received from Bundaberg Base Hospital on 2 December 2003. At the outset, I wish to convey my sincere condolences to you for the loss of your wife.

As you are aware, the Commission has been assessing the complaint to determine whether the health service provided to Mrs Connelly was reasonable and whether any further action may be required.

I understand that Mrs Connelly, who had a history of ischaemic heart disease, woke at 0330 hours on 1 December 2003 suffering with chest pain. An ambulance was called and transported her to Bundaberg Base Hospital at 0446 hours. Ambulance records state that on arrival at the scene, Mrs Connelly's pain had ceased in the chest but she still had pain in her back.

At the hospital, the duty medical practitioner noted Mrs Connelly's past history of acute myocardial infarction and hypothyroidism. She was examined, her vital signs monitored and no abnormality was detected. Various tests were performed which included serial electrocardiographs (ECGs) and while the chest x-ray was normal, blood tests showed raised levels of troponin[1]. Mrs Connelly was admitted to a general ward and later that day was reviewed by the specialist medical team who diagnosed her as having unstable angina. Aspirin, lipitor and lasix were added to her medication regime and she was discharged home at 1430 hours on 2 December 2003.

I understand that before Mrs Connelly was discharged, you explained to staff that she had been referred by her general practitioner the previous week for a stress (sestamibi) test to be performed by North Coast Nuclear Medicine at Mater Hospital that day at 10.20 a.m. The hospital's clinical plan for Mrs Connelly had been to take further blood tests and, if normal, the stress test would go ahead as planned. Following the appropriate blood tests and review of those tests, she was discharged with arrangements to transfer Mrs Connelly for her stress test. Before Mrs Connelly left the hospital the nursing staff member contacted North Coast Nuclear Medicine, and was told that the appointment had been reallocated and a new appointment was made for six days time. Tragically, your wife died in the early hours of the morning of 3 December 2003, at home. Her death certificate indicated that she died from a cardiac arrest following a myocardial infarction.

[1] An independent biochemist explained that troponins are muscle proteins found in the blood, which can be tested and analysed, following suspected heart muscle damage. High readings of troponin occur following cardiac damage.

Complaint Issues

I understand that your complaint issues are:

- Mrs Connelly was misdiagnosed and had she been correctly diagnosed and given appropriate treatment she would not have died; and
- Mrs Connelly should have attended the stress test, and if she had, she would have been correctly diagnosed and treated.

Misdiagnosis Issue

You stated that you were later informed that Mrs Connelly's past cardiac history and her elevated troponin levels were not taken into account when the decision was made to discharge her. You stated that you believed that had Mrs Connelly been correctly diagnosed and treated, she would not have died.

Mr Peter Leck, District Manager, Bundaberg District Health Service advised the Commission that the hospital had conducted a review of Mrs Connelly's care. The review confirmed that the combination of Mrs Connelly's past history, prolonged chest pain, ECG changes and raised troponin values indicated that she should have been diagnosed with Acute Coronary Syndrome and remained in hospital for ongoing observation. Mr Leck offered his sincere apologies to you for this failure.

In a further letter to the Commission, Dr Darren Keating, Director of Medical Services, explained that the failure to detect the raised troponin level was because the hospital used a different measurement from that used in one of the town's private laboratories. Dr Martin Strahan, general physician who attended to Mrs Connelly, was a visiting consultant who also worked in the private sector. It was explained that Dr Strahan did not appreciate the significance of your wife's troponin measurement because of the different measurement systems being used in the public and private health sectors leading to potential discrepancy between troponin values for the same patient. This discrepancy contributed to Dr Strahan placing limited significance on the test results at Bundaberg Base Hospital.

Dr Keating advised that Dr Strahan's reliance on the private sector method was based on his belief that the public sector method was inaccurate and possibly inferior. Subsequently, he ordered a different blood test, which was normal, and he discharged Mrs Connelly. Dr Keating recently advised the Commission that the private pathology provider in Bundaberg had recently installed the same troponin analyser as theirs to offset any future confusion.

Dr Keating also advised that the hospital has begun involvement with the Collaborative for Healthcare Improvement, Acute Coronary Syndrome, which provides evidence based guidelines and systematic evaluation of the treatment for this disorder in their hospital. The results will be compared on a state-wide basis. He explained that since Mrs Connelly's death there had been an education session for all medical staff involved in the care of cardiac patients. There were also continuing education sessions for senior medical staff on the specific topic of Acute Coronary Syndrome and the management of patients with raised troponin measurements. Dr Keating also advised that Dr Strahan had since undertaken further study, attended a cardiology conference and sought ongoing advice from cardiology peers.

The Commission sought comment from Dr Peter Hughes, State Manager of Queensland Health's Pathology Services regarding the uniformity of blood testing in Queensland Health hospitals. Dr Hughes advised that each Queensland Health hospital provides medical staff (visiting or salaried) with a booklet informing them of the normal ranges of all pathology tests undertaken at the hospital where they work. Dr Hughes said he was concerned that a doctor working in a Queensland Health hospital was not familiar with the specific pathology measurement ranges for that hospital. Dr Hughes also advised that when a result is abnormal the pathology department usually alerts the doctor to the abnormality.

The Commission then asked Dr Keating if Dr Strahan had been provided with specific information about pathology tests at their hospital. Dr Keating advised that Dr Strahan had worked as a salaried doctor at

the hospital before he became a visiting medical officer and that perhaps Dr Strahan may have missed out on receiving the updates on pathology information. Dr Keating said that Dr Strahan tried to clarify the measurement scale for troponin testing with another Queensland Health hospital, but did not receive any clarification prior to Mrs Connelly's discharge.

Independent advice obtained by the Commission from well-qualified cardiologists confirmed that Mrs Connelly should not have been sent home. An independent Deputy Medical Director of a cardiology program at a public hospital stated that whether troponin was positive or negative may not be the issue and explained that it was necessary to look at the systems in place. He stated that people with cardiac histories should be admitted regardless of the troponin level. Further independent advice said that had Mrs Connelly been kept in hospital, even in the Coronary Care Unit, there were no guarantees that she would have survived her cardiac arrest.

There is no doubt that Mrs Connelly should not been discharged. The Commission has requested that the District ensure that all medical personnel be provided with the current pathology information booklet and ensure that doctors new to the hospital are provided with the appropriate information as part of their orientation, regardless of whether they have previously worked at the hospital or not.

The Commission has also consulted the Medical Board of Queensland in relation to Dr Strahan's care of Mrs Connelly and whether he warranted investigation by the Board. The Commission is required to consult with the relevant registration Board in matters where there may be possible breaches of professional standards. In this case, the Commission drew to the Board's attention all the information and advice we had obtained. Having taken that information into account, the Board advised the Commission that, in its view, the matter does not warrant further action. This is a decision for the Board to make and the Commission is therefore unable to pursue the matter.

I appreciate that you will feel that the Commission has not done enough in relation to this issue, but, unfortunately, there is insufficient basis for me to be able to take any further action other than to recommend to Bundaberg Base Hospital that it continue to implement the changes in relation to its care of cardiac patients. None of the independent advisers contacted by the Commission have been able to state with sufficient confidence that your wife would have survived, even if she had remained in hospital.

Referral for Stress Test Issue

The other issue you raised in your letter was that at 8.30 a.m. on 2 December 2003, you informed Dr Strahan that Mrs Connelly had a pre-booked stress test appointment at 10.20 a.m. that day at a private hospital. You advised the Commission that you explained to Dr Strahan that Mrs Connelly's general practitioner had made this referral and that Dr Strahan then instructed the nurse to have this done straight away. You said you were informed by Dr Strahan that he suspected a blockage in her heart and that this test would identify where the blockage was. She could then be given something for it and be transferred to Brisbane for an operation.

You further advised that at 10.30 a.m. Dr Strahan informed you that the appointment had been reallocated and a new appointment made for 8 December 2003. You said you were subsequently informed that Mrs Connelly could go home and the results of the stress test would be sent to Bundaberg Base Hospital. When you made enquiries of the private hospital shortly after speaking to Dr Strahan, you were informed that they had not been contacted by Bundaberg Base Hospital and that the appointment had been reallocated at 9.30 a.m. You stated that when you asked the nurse why she had not called in relation to the stress test, she answered in an off-handed manner that it was the doctor's responsibility to do so. I understand you are of the view that even if Mrs Connelly had been diagnosed with a heart attack she could have still had a stress test without having to undergo a physical exercise. Also, that had she had the stress test, she would have been correctly diagnosed and treated.

I appreciate that you remain critical of the time taken by the staff to contact the nuclear medicine clinic. The key point I need to consider is whether it would have been appropriate for Mrs Connelly to have the

test at that time. The Commission sought clarification of this point from the nuclear physician, Dr Muttatamby Vannitamby, who performs the stress tests at the service that Mrs Connelly was due to attend. Dr Vannitamby stated that the referral from the referring doctor is only part of the information he would take into account. He said he would need to do his own assessment of a patient. He also advised that in most cases following a recent infarct, he would prefer to wait 4 to 6 weeks for the heart to recover before performing the stress test because of the high risk involved in the procedure. On this basis, I am unable to say that the test would definitely have been performed had Mrs Connelly's appointment gone ahead on 2 December 2003. This view is reinforced by advice obtained from independent cardiologists who advised the Commission that the referral to the nuclear medicine unit for the stress test was not particularly relevant, as it would only have confirmed what they should have already known i.e. that Mrs Connelly was a high-risk patient. Further, the stress test was contraindicated and could have made the situation worse.

While the actions of the nurse remain in dispute between yourself and the hospital, I have considered Dr Vannitamby's comments and those of the independent cardiologists, and, as noted above, it is not possible to say whether a stress test would or should have been performed on Mrs Connelly had she presented on 2 December 2003.

I have considered your concerns about the manner in which the hospital cared for Mrs Connelly and the hospital's response to those concerns, as well as the independent and third party comments. It is my view that there was a serious breakdown in procedures and that Mrs Connelly should have remained in hospital. As acknowledged by Bundaberg Base Hospital, they failed to take into account Mrs Connelly's prolonged chest pain, ECG changes, history and raised troponin levels. The Commission will advise the hospital of the importance of taking a systemic approach to the care of cardiac patients and of its continued involvement in the Collaborative for Healthcare, Acute Coronary Syndrome project.

I understand that you will remain unhappy with the Commission's findings and that you believe the matter should have been taken further. I realise that you may not agree with some of the advice the Commission has obtained, but I trust you will understand why the Commission needs to rely upon this. The Commission itself does not have the clinical expertise to reach findings on complex clinical matters and must rely on independent expert medical opinion or third party medical advice. Please be assured that the Commission will follow up to ensure that the procedural changes are occurring at the hospital in relation to the matters raised.

I am sorry that we have been unable to meet all of your expectations. I nevertheless thank you for bringing your complaint to the notice of the Commission.

Yours sincerely

Annette Anning
Acting Manager Complaints

[redacted] Assessment Extension

Consumer: Mrs [redacted] (DEC'D) Provider: [redacted]

Encryption Key:
Date Composed: 29/09/2004 02:48 PM Composed By: Karen Harbus/HRC

Short Description: HRC to Sullivan & Niccolaides, Dr D. Kanowski (Biochemist)

Body Text:

Generic advice: given in confidence: 3377 8666. I telephoned Dr Kanowski to enquire about some general issues.

- What is the difference between Troponin T and Troponin I?
- What is the relevance of Creatine Kinase (CK) in cardiac damage?

Dr Kanowski explained in general terms that troponins are muscle proteins which can be tested and analysed following heart muscle damage - high readings will be given following cardiac damage. He stated that there is a much larger reference range in relation to TI than to TT i.e. a high reading following cardiac damage for Troponin T could be in the range of 15 to 20 whereas a high reading for Troponin I '*following a massive event*" could be 1.5. He further explained that when figures alone were looked at, a very big heart event would read as "*normal*" under the Troponin I figures. He stated that a reference range had to be taken into account when tracking for both types of Troponin but explained that if a report was obtained over the telephone, or individual figures were written down without a reference range, then errors could occur. He stated that their laboratory tracked for Troponin T and any abnormal figures were printed out in red in order to help eliminate errors. He stated that different techniques are employed when measuring both types of Troponin.

TI attached on 4/10/04 KH

In relation to Creatine Kinase, the adviser stated that phosphocratine kinase (CK or CPK) is a type of protein called an enzyme and is a useful tool to use following muscle damage. He stated that the measuring of CK was a good indicator for quite a few general things e.g. general muscle damage and not just the heart muscle damage. He explained that it can be a very useful measuring tool following a cardiac event because, while Troponins can take up to 6 or 7 days to drop, CK will go down quite quickly following cardiac damage. He said that because of this factor, "*CK is sometimes more useful than troponins for diagnostic purposes*". He pointed out that CK is very specific to individuals i.e. where a normal range could be considered to be around 140, an older person's normal reading may be 60 or 70 due to loss of muscle tone.

Valicatec

Lab 14922-1954	Ur BN059241	Name CONNELLY Doreen	Sex F
Dob 22-Apr-1934	Wd DEM-BNH	Dr Sisiolo, Lipso*	c05:00 01-Dec-03

Specimen type Blood

Sodium	139	mmol/L	(135 - 145)	Protein	65	g/L	(62 - 83)
Potassium	4.2	mmol/L	(3.2 - 4.5)	Albumin	36	g/L	(33 - 47)
Chloride	107	mmol/L	(100 - 110)	Globulin	29	g/L	(25 - 45)
Bicarb.	25	mmol/L	(22 - 33)	Bilirubin	12	umol/L	(< 20)
Anion Gap	7	mmol/L	(4 - 16)	ALP	90	U/L	(30 - 120)
CSM(Calc)	277	mmol/kg	(270 - 295)	Gamma GT	19	U/L	(< 50)
Glucose	6.1	mmol/L	(3.0 - 7.8)	ALT	28	U/L	(< 40)
Fasting FR			(3.0 - 6.0)	AST	22	U/L	(< 35)
Urea	3.6	mmol/L	(2.0 - 8.0)	CK	121	U/L	(< 160)
Creatinine	0.09	mmol/L	(0.050 - 0.110)	cTnT	0.52	ug/L	(< 0.03)
Urea/Creat.	39 L		(40 - 100)				

Chest pain

Comment: Age:69 years

Comp.Act: Yes

Results phoned to ward

Health Rights Commission
040036.
06 DEC 2004
RECEIVED

Notes Alert

Phoned 5.49pm

Screen Dump
1st Result

Notes

Lab 14922-1954	Ur BN059241	Name CONNELLY Doreen	Sex F
Dob 22-Apr-1934 Wd DEM-BNH		Dr Sisiolo, Lipso*	c05:00 01-Dec-03

Time/Date	User	D	Specimen Notes
05:49 01-Dec-03	ckll	B	tnt phoned to DR Sisiolo
05:48 01-Dec-03	ckll	A	coag added per phone request by Dr Sisiolo. Form to follow

Clinical Notes
Chest pain

F5 Clinical Notes F6 Specimen Notes F7 UR Notes
F8 Set Dept Spec Notes SF7 Set Dept UR Notes

Screen Dump
Phone Log

0432

Validated

Lab 14922-1062	Ur BN059241	Name CONNELLY Doreen	Sex F
Dob 22-Apr-1934	Wd 10-BNH	Dr Sisiolo, Lipso*	c12:30 01-Dec-03

Specimen type Blood

Chest Pain
Current medication - lasix

CK	128	U/L	(< 160)
cTnT	0.69 H	ug/L	(< 0.03)

Comment: Age:69 years

Comp.Val: Yes

■ Notes ■ Audit

Screen Dump Second Result

0431

Health Rights Commission

Level 19
288 Edward Street
BRISBANE QLD 4000

Postal : GPO Box 3089
BRISBANE QLD 4001

FAXED

FACSIMILE COVER SHEET

TO: Dr David Kanowski, Biochemist

ADDRESS: Sullivan & Niccolaides

PHONE: 3377 8666

FAX: **3371 9277**

FROM: Karen Harbus

PHONE: 3234 0258

TOTAL NUMBER OF PAGES (including this sheet): 2

DATE: 29/09/04 **TIME:** 5.30 p.m.

COMMENTS:

Private & Confidential

Dear Dr Kanowski

Thank you very much for your thoughtful and considered comments today. I have attempted to write up a File Note of our discussion and I would be grateful if you could please check it to ensure it accurately reflects our discussion. I will be out of the office tomorrow, Thursday 30/09/04, but will be back again on Friday 01/10/04. I look forward to speaking to you at your convenience.

Kind regards

K.H.

Karen Harbus

IF YOU DO NOT RECEIVE ALL PAGES, PLEASE ADVISE IMMEDIATELY! 0430

CONSULTATION COVER SHEET – 040036

Attention: Medical Board of Qld

Date of Consultation: 22 September 2004

Board already notified of complaint? Yes ☐ No ☑

Consultation prior to Commissioner's decision to accept complaint for action, pursuant to section 71(3). Any comments must be received by 20 October 2004, pursuant to section 71(5)(b).

Date complaint received: 15 March 2004

Statutory Date: 2 July 2004

RECEIVED
20 SEP 2004
BY:

User: Mrs Doreen Connelly (Dec'd)

Complainant: Mr George Connelly

Provider: Dr Thomas Strahan

Speciality: Internal Medicine

Case Officer: Karen Harbus

Officer's Signature: Karen Harbus.

A man said that his wife was taken to a public hospital as she was suffering chest pain. He stated that she was admitted for one night, various tests were performed and she was discharged the following day with a diagnosis of unstable angina. The man said that his wife died of a cardiac arrest less than 24 hours after discharge by her treating doctor. The man said that his wife missed a pre-arranged stress (sestamibi) test scheduled for the morning of discharge and he blamed the nurse at the hospital for not ensuring another appointment was made for later that day. He was seeking the deregistration of the doctor and the nurse. The hospital responded to the Commission during assessment and provided a copy of the medical records. The hospital stated that given the woman's past history of acute myocardial infarction, prolonged chest pain, raised troponin values and echocardiograph changes, she should have been diagnosed with acute coronary syndrome and remained in hospital. The hospital also stated that they had begun involvement with the Collaborative for Healthcare Improvement, Acute Coronary Syndrome (this was verified by an officer of the Commission) and an education session had been conducted for all medical staff involved in the care of coronary patients. Independent advice was sought from both a Deputy Director of Emergency Medicine at an interstate public hospital as well as a Cardiology Registrar of a public hospital in Queensland. Both advisers agreed with the hospital that the woman should not have been discharged but should have remained in hospital for ongoing observation. The former adviser stated that the referral for the stress test was not particularly relevant as it would "*only have confirmed what they should have already known i.e. that the woman was a high risk patient*". The latter adviser said that the stress test was "*contraindicated*" and could have made the situation worse. The Commission requested further information from the hospital in relation to how the misdiagnosis had occurred. The hospital responded and stated that the specialist who treated the woman worked in both the private and public health sectors and he did not appreciate the significance of the woman's raised troponin result due to "*different measurement systems being used in the public and private health sectors... leading to potential discrepancy between troponin value for the same patient*". It was explained that this discrepancy contributed to the specialist attaching "*limited significance to such values*" at the public hospital. The hospital stated that the specialist used a private sector pathology laboratory which measured cardiac troponins using a portable card reader method where negative values are recorded as less than 0.05 mg/L and positive values are recorded as greater than 0.1 mg/L. It was explained that there is "*a grey or uncertain zone between these two readings*" and the value is also "*operator dependent*". The hospital stated that their troponin-measuring machine was more sensitive as negative values are recorded as less than 0.03 mg/L while positive values are equal to or greater than 0.03 mg/L. The Commission was informed that due to confusion between the two systems, the specialist favoured the private sector

felt the error was due to systemic issues, closure of this complaint is recommended pending further advice from the Board.

Board/QNC Representative (signature): _____

Board/QNC Referral ☐ Conciliation ☐ Closure ☑ Other ☐

Comments:

_____

_____

_____

_____

Commission Representative (signature): _____ 22.9.04.

File Note

040036

Consumer: Mrs [redacted] (DEC'D) Provider: [redacted] Assessment Extension

Encryption Key:
Date Composed: 07/09/2004 02:16 PM Composed By: Karen Harbus/HRC

Body Text:

Director of Cardiology (public hospital)

Given in Confidence. Do not release name of adviser. Dr [redacted] returned my call. I advised him that I was seeking his informal independent advice about a complaint where a 69 y.o. woman who had a history of heart problems, began to suffer ongoing chest pain and was taken by ambulance to a public hospital in a rural area in Queensland in the early hours of 01/12/03. I informed the adviser that the woman was diagnosed with "*unstable angina*" and given aspirin, lipitor and lasix in addition to her regular medication regime. I explained that a blood test was taken, results "*checked*" and the hospital began to make arrangements to transfer her to a pre-arranged stress (sestamibi) test booked for 9.30 a.m. on 02/12/03 at a private nuclear medicine unit. I informed the adviser that in its response to the HRC, the provider acknowledged that the woman had raised Troponin levels which indicated she had suffered a recent heart event, was misdiagnosed with "*unstable angina*" and discharged whereas they said they should have diagnosed her with "*acute coronary syndrome*" and kept her in for observation. I informed the adviser that the woman died in the early hours of 03/12/03 from 1.(a) cardiac arrest; (b) myocardial infarction; and (c) ischaemic heart disease. I explained that the widower believed that if his wife had been diagnosed and treated appropriately, she would have been given appropriate medication and still be alive. I also informed the adviser that the man was concerned that a nurse at the public hospital had not acted in a timely manner in transferring her to the stress test appointment at the private facility as he believed that as the provider had misdiagnosed the woman, the stress test would have correctly diagnosed her and he "*could have flown her down to Brisbane in a private plane for an urgent operation*". I explained to the adviser that the man wanted the doctor and the nurse sacked/de-registered.

The adviser agreed with previous independent advice obtained that (a) the stress test was contraindicated and (b) it would only have confirmed what the hospital should have already known and he further said that, in any event, given the time frame of the woman's death, by the time the stress test had been performed, diagnosis made and arrangements made to transport the woman to a larger public hospital for surgery, it would have been "*too late*", as she died in the early hours of 03/12/03. I explained to the adviser that after its initial response, the HRC wrote back to the provider to ask who had made the misdiagnosis and how. I read him excerpts from the provider's subsequent letter as follows:

[The doctor, FRACP, general physician, internal medicine] was the attending specialist medical practitioner to [the woman] during her last admission to hospital. The doctor is a Visiting Medical Officer in General Medicine, who also practices in the private sector. He acknowledges that he didn't appreciate the significance of [the woman's] raised Troponin result due to different measurement systems being used in the public and private health sectors in [the area] leading to potential discrepancy between Troponin values for the same patient. This discrepancy contributed to the doctor attaching limited significance to such values at the public hospital.

The doctor was using a private sector pathology laboratory which measured cardiac Troponins using a portable card reader method. Negative values are recorded as less than 0.05 mg/L and positive values are recorded as greater than 0.1 mg/L. There is a grey or uncertain zone between these two readings. The value is also operator dependent. At the public hospital, Troponins are measured using a Roche Elecsys 1010 analyser, which is internally validated daily and externally checked under the Royal College of Pathologists Australasia Quality Program every two weeks. Negative values are recorded as less than 0.03 mg/L while positive values are equal to or greater than 0.03 mg/L. This level is standard across all Queensland Health pathology laboratories with the result electronically recorded and distributed. The public sector method is more sensitive than the private sector method.

The doctor's confusion led to a strong support of the private sector method and belief that the public sector method was inaccurate and possibly inferior. Consequently the doctor also utilised measurement of creatinine kinase (CK) in patients with acute coronary syndrome as happened in [the woman's] case. The doctor asserts that he attempted to reduce his concern through inquiries with the [larger area] based management of the public hospital's pathology laboratory, but didn't obtain a satisfactory response. After [the woman's] death, he again made inquiries and was provided with the above information. At this time he realised his mistaken belief in the private sector's methodology for troponin measurement. Since this time, he has undertaken further study, attended a cardiology conference and sought ongoing advice from his cardiology peers. He states that he understands the significance of troponin values, particularly in risk stratification of patients with coronary artery disease. It should be noted that the private pathology provider in [the area] has recently installed the same troponin analyser as installed in the public hospital in order to improve sensitivity of testing, reduce unnecessary duplication and avoid discrepancy in values.

The adviser stated that in his opinion a good question would be: "*Was it an error to have diagnosed the woman with unstable angina?*". He said no and explained that the term "*acute coronary syndrome*" was a very broad umbrella term to cover lots of coronary conditions and as the hospital stated "*the woman was stable throughout her admission*", the hospital was not incorrect to have diagnosed her with unstable angina. He explained that the health care system had been dealing with subtle changes of differing nomenclature for a few years now. He stated that the error made by the hospital was to discharge her too soon. He stated that the blood tests certainly flagged that she was at a higher risk of suffering from a heart related problem and this had indeed occurred. The adviser stated, "*So the error was not so much in the diagnosis as in failing to recognise that her Troponin levels mandated that she receive more intensive therapy rather than be discharged*". The adviser noted that the hospital had undertaken procedural changes and that the man was given a sincere apology.

In relation to the outcome that the man was seeking, the adviser stated that he "*absolutely disagreed*" with this as, firstly, the hospital was not in a major metropolitan area, and by sacking a doctor who had made an error, one would be depriving that locality of a specialist doctor. He said he noted from the provider's response that the doctor had undertaken further study, attended a cardiology conference and liaised with cardiology peers and was sorry for what had happened. He explained that secondly, another important issue that had to be taken into account was that some laboratories track Troponin 'T' and some laboratories track Troponin 'I' and the testing methodology for testing the normal range for each of these is different. He said that by way of example, a doctor may look at a reading of 0.04 mg/L in

reference to what he recognised as normal but this would depend on which type of Troponin the laboratory was testing for (i.e. T or I types). He said some laboratories tested for the I type of Troponin whereas some track for the T type. He said that by of example, Sullivan & Niccolaides in Brisbane track Troponin T and the [redacted] Hospital tracks for Troponin I. He said he was informed by his hospital's laboratory that [redacted] Hospital tracks Troponin T. He said the point to make here is that the tracking of different types of Troponin was not uniform across Queensland, and stressed that there was no uniformity between the public and private sectors nor, it appeared, between the public to public system.

In summing up, the independent adviser stated that it appeared an "*honest mistake*" had been made and as it appeared to be a "*one off*" on the part of the specialist, he did not see the error as being an issue of a breach of professional standards by the doctor involved but rather one of "*a simple error*". He stated that he could understand the man's grief and anger but "*to deprive a community of a specialist who was willing to work in both the private and public arenas*" was not the answer. He said that about 85% of doctors preferred to work in the private sector.

Thanked him and agreed to fax him a copy of the File Note for clarification.

File Note

040036

Consumer: Mrs [redacted] (DEC'D) Provider: [redacted] Assessment Extension

Encryption Key:
Date Composed: 31/08/2004 11:47 AM Composed By: Karen Harbus/HRC

Body Text:

Given in confidence: 5519 8211. I called Dr [redacted] (Cardiology Registrar) as he had given earlier indep. advice in relation to this matter. He said he could recall the case quite well. I advised him that I had written to P again to request who made the misdiagnosis and how, and had received a response which named the doctor and explained that he worked in both the public and private sectors and that the private sector used a "portable card reader method" to measure cardiac troponins whereas the public hospital used a Roche Elecsys 1010 analyser. The adviser commented that both methods give a "normal/abnormal" reading so this should not really have been an issue for the doctor and said that the results have "a reference range beside them". He stated that it was possible that the doctor was used to looking at "one set of numbers" and this may be how the error occurred. I outlined the contents of the further letter from P and he said he did not think it would be "productive" to look further into the matter. He stated that P had admitted to systems errors and said they were making changes as a result of this i.e. the private sector had purchased a tropinin machine which matched theirs. He said that P acknowledged they had deviated from the state-wide guidelines. I advised him that C wanted to see the doctor de-registered and he commented that P could not "defend the issue" and the man may be able to seek compensation through the legal system. I advised him that the man did not want compensation and he commented that he could understand that C "wanted justice done". The adviser said that maybe C could go back to the District and say he wasn't happy with their explanations and changes. Thanked him for his assistance.

File Note

040036

Consumer: Mrs [redacted] (DEC'D) Provider: [redacted] Assessment Extension

[redacted]

Encryption Key:
Date Composed: 31/08/2004 10:09 AM Composed By: Karen Harbus/HRC

Body Text:
3350 8111. I rang to speak to Dr[redacted], cardiologist who is on HRC's indep. database but I was told he was out of the country until next week. I was referred to Dr[redacted] (Deputy Medical Director, Cardiology Program). **GIVEN IN CONFIDENCE:** I outlined the complaint that a public hospital acknowledged that they had discharged a 69 y.o. woman with a diagnosis of unstable angina when in fact she should have been kept in the hospital and diagnosed with acute coronary syndrome. I explained that she had a past history of heart problems, had presented to the hospital with prolonged chest pain, ECG changes had occurred and she had raised troponin reading and had suffered a "minor event". The adviser explained that whether or not troponin was positive or negative may not be the issue here and he explained that it was necessary to look at the bigger picture and look at the systems in place. He explained that at his hospital, which specialises in heart conditions, if a person had a history of heart condition and presented with chest pain, they would be "kept in automatically" regardless of troponin readings and this was an example of a systems approach. He said that other systems' problems would be:

- the public hospital had one type of measurement system for troponin but the private sector had another type, yet they were both in same locality;
- quality assurance - results should be checked in a systematic manner, not individually;
- patients with certain coronary histories should be "admitted as part of normal procedure" regardless of troponin readings etc.
- due to lack of funding/resources, there is great pressure to get patients out of public hospitals as the pressure for beds is very high.

He stated that it was helpful to look at the "big picture" as concentric circles - there are several levels: clinical level, systems level. The adviser said that the provider had admitted to systemic errors. He said that the question "What was the driving force behind discharging the woman?" needed to be asked and it would not come down to a "single decision" as the matter was not black and white and involved several levels. He said that the answer would be a mixture of the above systems issues. He explained that while it was "easy to blame one doctor", the big picture needed to be looked at. He commented that the lack of a systemic approach should be brought to the hospital's attention and while it would be "expedient" to discipline an individual doctor, this would not solve the greater problems and would not be appropriate. He stated that he could understand that C wanted to "blame" someone as he was very angry. I asked him if he would be willing to look at a de-identified copy of the letter from P but he declined, stating that he felt he had covered all the pertinent issues and could not see what more could be gained by looking at an individual registrant. Thanked him for his general advice.

FAX MESSAGE

Bundaberg Health Service District
P O Box 34
BUNDABERG QLD 4670

Queensland Government
Queensland Health

TO:		FROM:	
Fax:	07 32340333	**Fax:**	07 41502029
Name:	Karen Harbus	**Phone:**	07 41502020
Organisation:	Health Rights Commission	**Name:**	Dr Darren Keating
Date:	21/09/2004	**Position:**	Director of Medical Serviced

CONFIDENTIAL COMMUNICATION

SUBJECT: Mr George Connelly

Pages 2 **(Inclusive)**

As per our discussion, attached please find article which appeared in yesterday's edition of the Bundaberg News Mail.

Darren

Health Rights Commission
21 SEP 2004
RECEIVED

News Mail 20/8/04 - Page 3

HEALTH

Complaint referred to board

By GREG CHAPMAN

INQUIRY CALL

GEORGE Connelly's push for answers finally received some results recently when the Health Rights Commission (HRC) referred his medical complaint to the next level.

The 66-year-old pensioner's nine-month quest for answers about the death of his wife came to a crossroads on Friday when the HRC told him his case would be referred to the Queensland Medical Board.

The HRC also told Mr Connelly it would recommend action be taken against the medical specialist who treated his wife.

The News-Mail put its weight behind Mr Connelly's cause by publishing several articles and personally writing to the HRC appealing for quick action on the matter.

The story was later followed by Channel 7's current affairs program *Today Tonight*.

Doreen, 69 died following a suspected heart attack just hours after being released from Bundaberg Base Hospital in December last year.

"I'm not happy — it has taken nine months for them (HRC) to come up with this," Mr Connelly said.

"The HRC said they will be holding a meeting with the board this week, they won't let me attend and whether they'll give me any results, I don't know."

Mr Connelly, who has emphysema, disputed the HRC's recommendation against the specialist who works for Bundaberg Hospital and has a private practice.

Mr Connelly obtained documents under the Freedom of Information Act of the HRC's investigation.

A woman interviewed by the HRC said Mrs Connelly was "treated unreasonably" and should have been given an urgent angiogram to assess her heart problem.

The woman and another person that was interviewed both said Mrs Connelly should have been given the blood-thinning medication heparin.

"Reading those letters it shows complete negligence by the hospital as a whole," Mr Connolly said.

"The stress test was ordered by the specialist, but it was never done."

A spokesman for Health Minister Gordon Nuttall, whose department handles the Queensland Medical Board, could not say when Mr Connelly's case would be heard.

MAKING PROGRESS: George Connelly's quest for answers about his wife's death has gained momentum. *Photo: GREG CHAPMAN ge01709*

MEETINGS

Businesses look to European opportunities

BUNDABERG'S innovative industries have attracted the attention the businesses that Mr Dawson will hold meetings with.

Peter Beasley said Mr Dawson's visit was only out of interest.

Ainsworth to meet with him because the United Kingdom and

Mr Smith said the commissioner would then spend the rest of the

Queensland Government

Queensland Health

THE PRINCE CHARLES HOSPITAL HEALTH SERVICE DISTRICT

FACSIMILE COVER SHEET

TO: KAREN HARRIS

ADDRESS: Health Rights Commission

PHONE NUMBER: **FAX NUMBER:** 3234 0333

FROM: CHEST PAIN ASSESSMENT SERVICE

ADDRESS: CORONARY CARE UNIT THE PRINCE CHARLES HOSPITAL

PHONE NUMBER: 3350 ~~8039~~ 8567 **FAX NUMBER:** 3350 8208

TOTAL NUMBER OF PAGES (INCLUDING THIS SHEET): 3

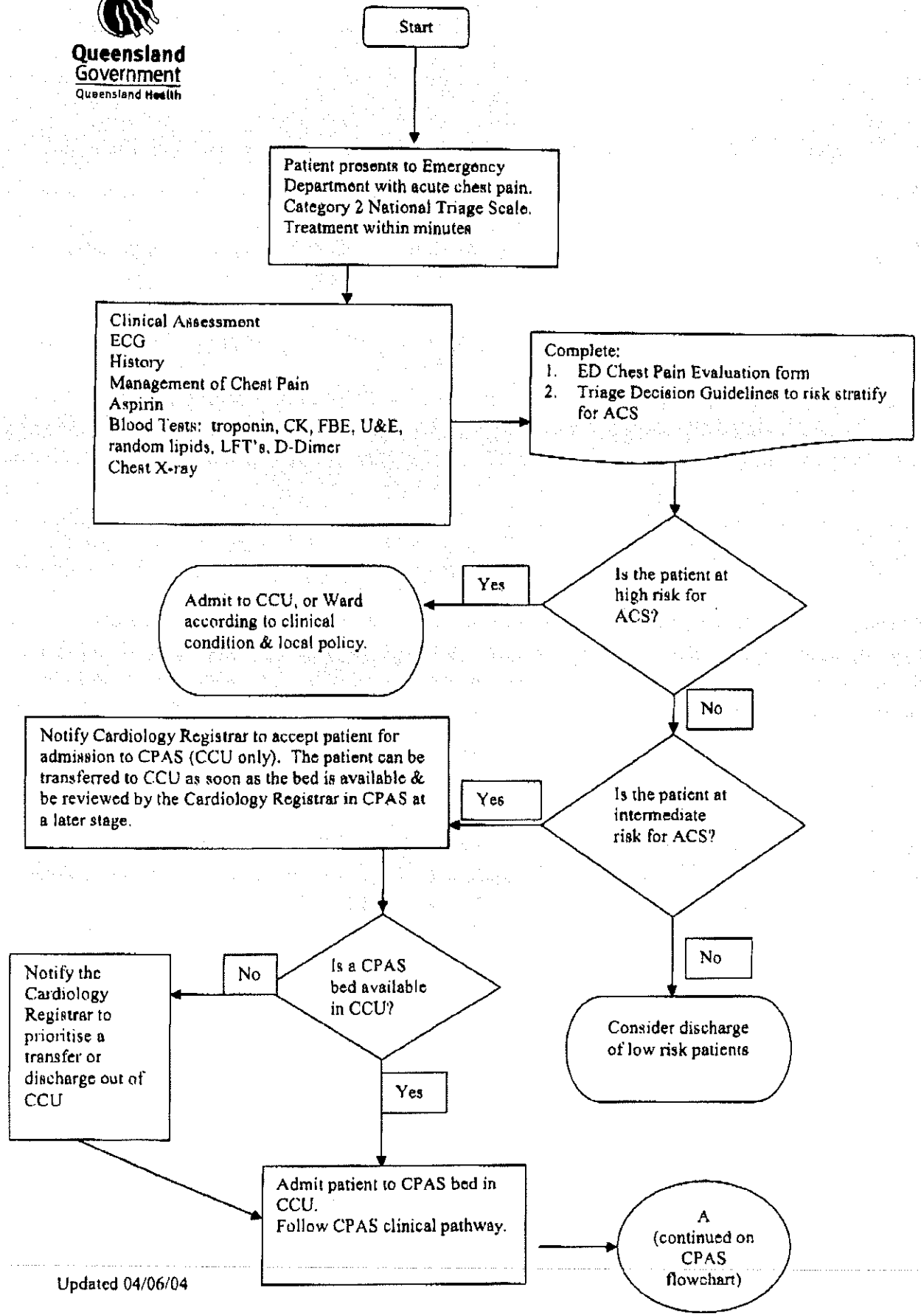
DATE: 15/9/4

COMMENTS: Please contact us if more detailed information is required (Heather Dunlevie contact person)

IF YOU DO NOT RECEIVE ALL PAGES PLEASE ADVISE IMMEDIATELY.

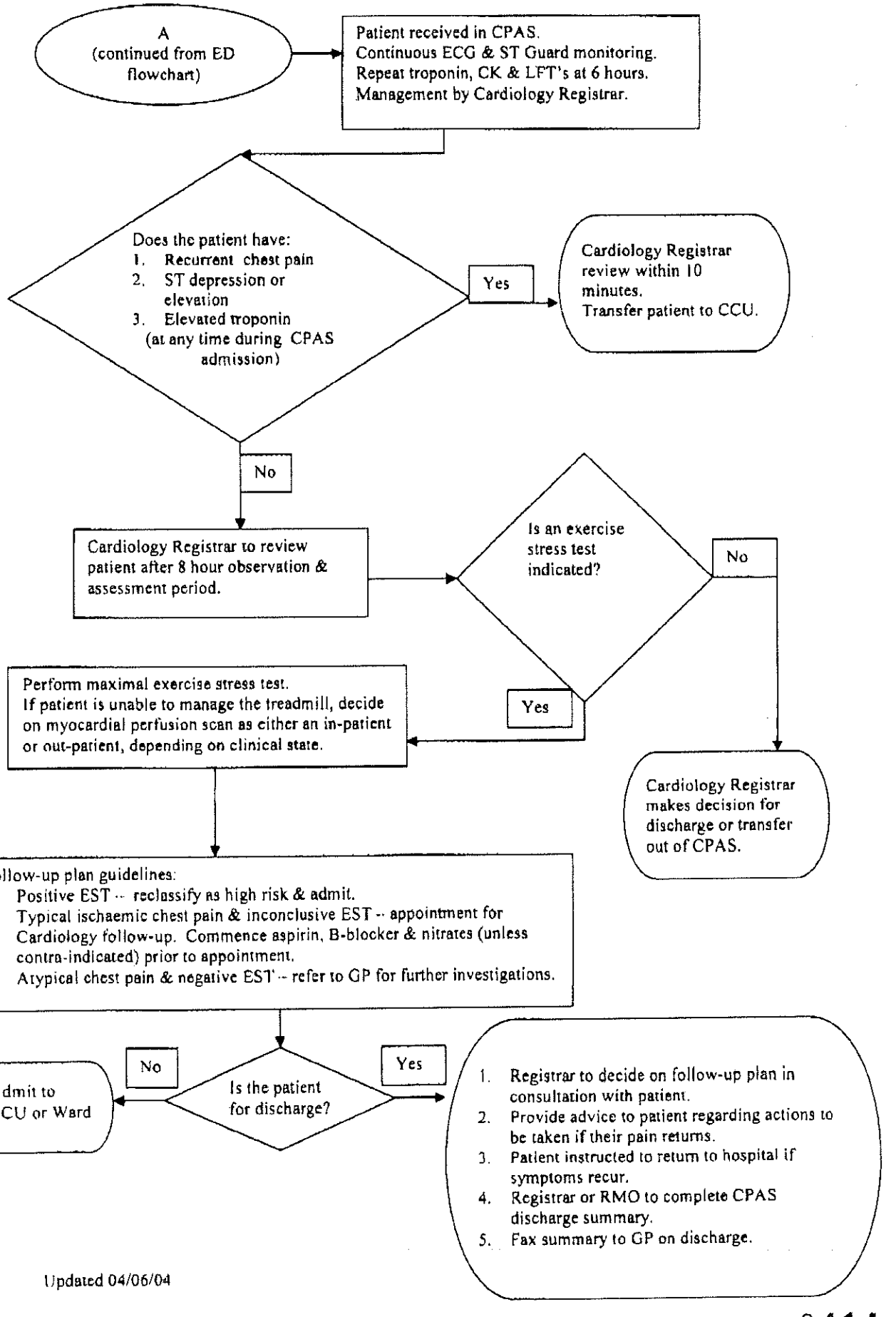
The Prince Charles Hospital Health Service District:
ED Flowchart for risk stratification for ACS and Admission Process to CPAS

Queensland Government
Queensland Health



Updated 04/06/04

The Prince Charles Hospital Health Service District: CPAS Flowchart



Updated 04/06/04

News Mail 20/8/04 - Page 3

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Photo: GREG CHAPMAN gsp1709

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De-identified for Consult - Cover Sheet 20.9.04

File Note

040036

Consumer: Mrs [redacted] (DEC'D) Provider: [redacted] Assessment Extension

Encryption Key:
Date Composed: 07/09/2004 02:16 PM Composed By: Karen Harbus/HRC

Body Text:

Director of Cardiology (public hospital)

Given in Confidence. Do not release name of adviser. Dr [redacted] returned my call. I advised him that I was seeking his informal independent advice about a complaint where a 69 y.o. woman who had a history of heart problems, began to suffer ongoing chest pain and was taken by ambulance to a public hospital in a rural area in Queensland in the early hours of 01/12/03. I informed the adviser that the woman was diagnosed with "*unstable angina*" and given aspirin, lipitor and lasix in addition to her regular medication regime. I explained that a blood test was taken, results "*checked*" and the hospital began to make arrangements to transfer her to a pre-arranged stress (sestamibi) test booked for 9.30 a.m. on 02/12/03 at a private nuclear medicine unit. I informed the adviser that in its response to the HRC, the provider acknowledged that the woman had raised Troponin levels which indicated she had suffered a recent heart event, was misdiagnosed with "*unstable angina*" and discharged whereas they said they should have diagnosed her with "*acute coronary syndrome*" and kept her in for observation. I informed the adviser that the woman died in the early hours of 03/12/03 from 1.(a) cardiac arrest; (b) myocardial infarction; and (c) ischaemic heart disease. I explained that the widower believed that if his wife had been diagnosed and treated appropriately, she would have been given appropriate medication and still be alive. I also informed the adviser that the man was concerned that a nurse at the public hospital had not acted in a timely manner in transferring her to the stress test appointment at the private facility as he believed that as the provider had misdiagnosed the woman, the stress test would have correctly diagnosed her and he "*could have flown her down to Brisbane in a private plane for an urgent operation*". I explained to the adviser that the man wanted the doctor and the nurse sacked/de-registered.

The adviser agreed with previous independent advice obtained that (a) the stress test was contraindicated and (b) it would only have confirmed what the hospital should have already known and he further said that, in any event, given the time frame of the woman's death, by the time the stress test had been performed, diagnosis made and arrangements made to transport the woman to a larger public hospital for surgery, it would have been "*too late*", as she died in the early hours of 03/12/03. I explained to the adviser that after its initial response, the HRC wrote back to the provider to ask who had made the misdiagnosis and how. I read him excerpts from the provider's subsequent letter as follows:

[The doctor, FRACP, general physician, internal medicine] was the attending specialist medical practitioner to [the woman] during her last admission to hospital. The doctor is a Visiting Medical Officer in General Medicine, who also practices in the private sector. He acknowledges that he didn't appreciate the significance of [the woman's] raised Troponin result due to different measurement systems being used in the public and private health sectors in [the area] leading to potential discrepancy between Troponin values for the same patient. This discrepancy contributed to the doctor attaching limited significance to such values at the public hospital.

The doctor was using a private sector pathology laboratory which measured cardiac Troponins using a portable card reader method. Negative values are recorded as less than 0.05 mg/L and positive values are recorded as greater than 0.1 mg/L. There is a grey or uncertain zone between these two readings. The value is also operator dependent. At the public hospital, Troponins are measured using a Roche Elecsys 1010 analyser, which is internally validated daily and externally checked under the Royal College of Pathologists Australasia Quality Program every two weeks. Negative values are recorded as less than 0.03 mg/L while positive values are equal to or greater than 0.03 mg/L. This level is standard across all Queensland Health pathology laboratories with the result electronically recorded and distributed. The public sector method is more sensitive than the private sector method.

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In relation to the outcome that the man was seeking, the adviser stated that he "*absolutely disagreed*" with this as, firstly, the hospital was not in a major metropolitan area, and by sacking a doctor who had made an error, one would be depriving that locality of a specialist doctor. He said he noted from the provider's response that the doctor had undertaken further study, attended a cardiology conference and liaised with cardiology peers and was sorry for what had happened. He explained that secondly, another important issue that had to be taken into account was that some laboratories track Troponin 'T' and some laboratories track Troponin 'I' and the testing methodology for testing the normal range for each of these is different. He said that by way of example, a doctor may look at a reading of 0.04 mg/L in

reference to what he recognised as normal but this would depend on which type of Troponin the laboratory was testing for (i.e. T or I types). He said some laboratories tested for the I type of Troponin whereas some track for the T type. He said that by of example, Sullivan & Niccolaides in Brisbane track Troponin T and the [redacted] Hospital tracks for Troponin I. He said he was informed by his hospital's laboratory that [redacted] Hospital tracks Troponin T. He said the point to make here is that the tracking of different types of Troponin was not uniform across Queensland, and stressed that there was no uniformity between the public and private sectors nor, it appeared, between the public to public system.

In summing up, the independent adviser stated that it appeared an "*honest mistake*" had been made and as it appeared to be a "*one off*" on the part of the specialist, he did not see the error as being an issue of a breach of professional standards by the doctor involved but rather one of "*a simple error*". He stated that he could understand the man's grief and anger but "*to deprive a community of a specialist who was willing to work in both the private and public arenas*" was not the answer. He said that about 85% of doctors preferred to work in the private sector.

Thanked him and agreed to fax him a copy of the File Note for clarification.

Consultation
Cover Sheet
20.9.04

File Note

040036

Consumer: Mrs [redacted] (DEC'D) Provider: [redacted] Assessment Extension

Encryption Key:
Date Composed: 31/08/2004 10:09 AM Composed By: Karen Harbus/HRC

Body Text:
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122. Inhibition of platelet glycoprotein IIb/IIIa with eptifibatide in patients with acute coronary syndromes. The PURSUIT Trial Investigators. Platelet Glycoprotein IIb/IIIa in Unstable Angina: Receptor Suppression Using Integrilin Therapy. *N Engl J Med* 1998; 339: 436-443.
123. International, randomized, controlled trial of lamifiban (a platelet glycoprotein IIb/IIIa inhibitor), heparin, or both in unstable angina. The PARAGON Investigators. Platelet IIb/IIIa Antagonism for the Reduction of Acute coronary syndrome events in a Global Organization Network. *Circulation* 1998; 97: 2386-2395.
124. A comparison of aspirin plus tirofiban with aspirin plus heparin for unstable angina. Platelet Receptor Inhibition in Ischemic Syndrome Management (PRISM) Study Investigators. *N Engl J Med* 1998; 338: 1498-1505.
125. Cohen M, Theroux P, Weber S, et al. Combination therapy with tirofiban and enoxaparin in acute coronary syndromes. *Int J Cardiol* 1999; 71: 273-281.
126. Neumann FJ, Zohlnhofer D, Fakhoury L, et al. Effect of glycoprotein IIb/IIIa receptor blockade on platelet-leukocyte interaction and surface expression of the leukocyte integrin Mac-1 in acute myocardial infarction. *J Am Coll Cardiol* 1999; 34: 1420-1426.
127. Mickelson JK, Ali MN, Kleiman NS, et al. Chimeric 7E3 Fab (ReoPro) decreases detectable CD11b on neutrophils from patients undergoing coronary angioplasty. *J Am Coll Cardiol* 1999; 33: 97-106.
128. Le Breton H, Plow EF, Topol EJ. Role of platelets in restenosis after percutaneous coronary revascularization. *J Am Coll Cardiol* 1996; 28: 1643-1651.
129. Tam SH, Sassoli PM, Jordan RE, Nakada MT. Abciximab (ReoPro, chimeric 7E3 Fab) demonstrates equivalent affinity and functional blockade of glycoprotein IIb/IIIa and alpha(v)beta3 integrins. *Circulation* 1998; 98: 1085-1091.
130. Gupta S, Leatham EW, Carrington D, et al. Elevated Chlamydia pneumoniae antibodies, cardiovascular events, and azithromycin in male survivors of myocardial infarction. *Circulation* 1997; 96: 404-407.
131. Gurfinkel E, Bozovich G, Daroca A, et al. Randomised trial of roxithromycin in non-Q-wave coronary syndromes: ROXIS Pilot Study. ROXIS Study Group. *Lancet* 1997; 350: 404-407.
132. Gurfinkel E, Bozovich G, Beck E, et al. Treatment with the antibiotic roxithromycin in patients with acute non-Q-wave coronary syndromes. The final report of the ROXIS Study. *Eur Heart J* 1999; 20: 121-127.
133. Boden WE, O'Rourke RA, Crawford MH, et al. Outcomes in patients with acute non-Q-wave myocardial infarction randomly assigned to an invasive as compared with a conservative management strategy. Veterans Affairs Non-Q-Wave Infarction Strategies in Hospital (VANQWISH) Trial Investigators. *N Engl J Med* 1998; 338: 1785-1792.
134. Zhao XQ, Theroux P, Snapinn SM, Sax FL. Intracoronary thrombus and platelet glycoprotein IIb/IIIa receptor blockade with tirofiban in unstable angina or non-Q-wave myocardial infarction: angiographic results from the PRISM-PLUS trial (Platelet receptor inhibition for ischemic syndrome management in patients limited by unstable signs and symptoms). *Circulation* 1999; 100: 1609-1615.
135. Use of a monoclonal antibody directed against the platelet glycoprotein IIb/IIIa receptor in high-risk coronary angioplasty. The EPIC Investigation. *N Engl J Med* 1994; 330: 956-961.
136. Topol EJ, Califf RM, Weisman HF, et al. Randomised trial of coronary intervention with antibody against platelet IIb/IIIa integrin for reduction of clinical restenosis: results at six months. The EPIC Investigators. *Lancet* 1994; 343: 881-886.
137. Platelet glycoprotein IIb/IIIa receptor blockade and low-dose heparin during percutaneous coronary revascularization. The EPILOG Investigators. *N Engl J Med* 1997; 336: 1689-1696.
138. Randomised placebo-controlled trial of abciximab before and during coronary intervention in refractory unstable angina: the CAPTURE Study. *Lancet* 1997; 349: 429-435.
139. Randomised placebo-controlled and balloon-angioplasty-controlled trial to assess safety of coronary stenting with use of platelet glycoprotein-IIb/IIIa blockade. The EPISTENT Investigators. Evaluation of Platelet IIb/IIIa Inhibitor for Stenting. *Lancet* 1998; 352: 87-92.
140. Topol EJ, Ferguson JJ, Weisman HF, et al. Long-term protection from myocardial ischemic events in a randomized trial of brief integrin beta3 blockade with percutaneous coronary intervention. EPIC Investigator Group. Evaluation of Platelet IIb/IIIa Inhibition for Prevention of Ischemic Complication. *JAMA* 1997; 278: 479-484.
141. Moussa I, Oetgen M, Roubin G, et al. Effectiveness of clopidogrel and aspirin versus ticlopidine and aspirin in preventing stent thrombosis after coronary stent implantation. *Circulation* 1999; 99: 2364-2366.
142. Berger PB, Bell MR, Hasdai D, et al. Safety and efficacy of ticlopidine for only 2 weeks after successful intracoronary stent placement. *Circulation* 1999; 99: 248-253.
143. Madsen JK, Grande P, Saunamaki K, et al. Danish multicenter randomized study of invasive versus conservative treatment in patients with inducible ischemia after thrombolysis in acute myocardial infarction (DANAMI). DANish trial in Acute Myocardial Infarction. *Circulation* 1997; 96: 748-755.
144. Diver DJ, Bier JD, Ferriera PE, et al. Clinical and arteriographic characterization of patients with unstable angina without critical coronary arterial narrowing. *Am J Cardiol* 1994; 74: 531.
145. Brown G, Albers JJ, Fisher LD, et al. Regression of coronary artery disease as a result of intensive lipid-lowering therapy in men with high levels of apolipoprotein B. *N Engl J Med* 1990; 323: 1289-1298.
146. Voskuil J, Cramer MJ, Breumelhof R, et al. Prevalence of esophageal disorders in patients with chest pain newly referred to the cardiologist. *Chest* 1996; 109: 1210.
147. Bassan R, Araujo M, Volschan A, et al. Does the diagnosis of unstable angina require the presence of ischemic changes? *J Am Coll Cardiol* 1999; 33: 393A.
148. Goldman L, Cook EF, Brand DA, et al. A computer protocol to predict myocardial infarction in emergency department patients with chest pain. *N Engl J Med* 1988; 318: 797-803.
149. Zalenski RJ, McCarren M, Roberts R, et al. An evaluation of a chest pain diagnostic protocol to exclude acute cardiac ischemia in the emergency department. *Arch Intern Med* 1997; 157: 1085-1091.
150. DeWood MA, Notske RN, Hensley GR, et al. Intraaortic balloon counterpulsation with and without reperfusion for myocardial infarction shock. *Circulation* 1980; 61: 1105-1112.
151. Anderson RD, Ohman EM, Holmes DR Jr, et al. Use of intraaortic balloon counterpulsation in patients presenting with cardiogenic shock: observations from the GUSTO-I Study. Global Utilization of Streptokinase and TPA for Occluded Coronary Arteries. *J Am Coll Cardiol* 1997; 30: 708-715.
152. Early treatment of unstable angina in the coronary care unit: a randomised, double blind, placebo controlled comparison of recurrent ischaemia in patients treated with nifedipine or metoprolol or both. Report of The Holland Interuniversity Nifedipine/Metoprolol Trial (HINT) Research Group. *Br Heart J* 1986; 56: 400-413.
153. Randomised trial of cholesterol lowering in 4444 patients with coronary heart disease: the Scandinavian Simvastatin Survival Study (4S). *Lancet* 1994; 344: 1383-1389.
154. Sacks FM, Pfeffer MA, Moye LA, et al. The effect of pravastatin on coronary events after myocardial infarction in patients with average cholesterol levels. Cholesterol and Recurrent Events Trial investigators. *N Engl J Med* 1996; 335: 1001-1009.
155. Prevention of cardiovascular events and death with pravastatin in patients with coronary heart disease and a broad range of initial cholesterol levels. The Long-Term Intervention with Pravastatin in Ischaemic Disease (LIPID) Study Group. *N Engl J Med* 1998; 339: 1349-1357.
156. Yusuf S, Sleight P, Pogue J, et al. Effects of an angiotensin-converting-enzyme inhibitor, ramipril, on cardiovascular events in high-risk patients. The Heart Outcomes Prevention Evaluation Study Investigators. *N Engl J Med* 2000; 342: 145-153.
157. Recommendations for Cardiac Rehabilitation. Canberra: National Heart Foundation of Australia, 1998.
158. Oldridge NB, Guyatt GH, Fischer ME, Rimm AA. Cardiac rehabilitation after myocardial infarction: combined experience of randomized clinical trials. *JAMA* 1988; 260: 945-950
159. O'Connor GT, Buring JE, Yusuf S, et al. An overview of randomized trials of rehabilitation with exercise after myocardial infarction. *Circulation* 1989; 80: 234-244. ❑

74. Persantine and aspirin in coronary heart disease. The Persantine–Aspirin Reinfarction Study Research Group. *Circulation* 1980; 62: 449-461.
75. Collaborative overview of randomised trials of antiplatelet therapy — I: Prevention of death, myocardial infarction, and stroke by prolonged antiplatelet therapy in various categories of patients. Antiplatelet Trialists' Collaboration. *BMJ* 1994; 308: 81-106.
76. Balsano F, Rizzon P, Violi F, et al. Antiplatelet treatment with ticlopidine in unstable angina. A controlled multicenter clinical trial. The Studio della Ticlopidina nell'Angina Instabile Group. *Circulation* 1990; 82: 17-26.
77. A randomised, blinded, trial of clopidogrel versus aspirin in patients at risk of ischaemic events (CAPRIE). CAPRIE Steering Committee. *Lancet* 1996; 348: 1329-1339.
78. dePace NL, Herling IH, Kotler ML, et al. Intravenous nitroglycerin for rest angina. Potential pathophysiologic mechanisms of action. *Arch Intern Med* 1982; 142: 1806.
79. Roubin GS, Harris PJ, Eckhardt I, et al. Intravenous nitroglycerin in refractory unstable angina pectoris. *Aust N Z J Med* 1982; 12: 598.
80. Kaplan K, Davison R, Parker M, et al. Intravenous nitroglycerin for the treatment of angina at rest unresponsive to standard nitrate therapy. *Am J Cardiol* 1983; 51: 694-698.
81. Horowitz JD, Henry CA, Syrjanen ML, et al. Combined use of nitroglycerin and N-acetylcysteine in the management of unstable angina pectoris. *Circulation* 1988; 77: 787.
82. Ardissino D, Merlini PA, Savonitto S, et al. Effect of transdermal nitroglycerin or N-acetylcysteine, or both, in the long-term treatment of unstable angina pectoris. *J Am Coll Cardiol* 1997; 29: 941.
83. Krumenacker M, Roland E. Clinical profile of nicorandil: an overview of its hemodynamic properties and therapeutic efficacy. *J Cardiovasc Pharmacol* 1992; 20: S93-S102.
84. Tsutamoto T, Kinoshita M, Nakae I, et al. Absence of hemodynamic tolerance to nicorandil in patients with severe congestive heart failure. *Am Heart J* 1994; 127: 866-873.
85. Patel DJ, Purcell HJ, Fox KM. Cardioprotection by opening of the K(ATP) channel in unstable angina. Is this a clinical manifestation of myocardial preconditioning? Results of a randomized study with nicorandil. CESAR 2 investigation. Clinical European studies in angina and revascularization. *Eur Heart J* 1999; 20: 51-57.
86. Telford AM, Wilson C. Trial of heparin versus atenolol in prevention of myocardial infarction in intermediate coronary syndrome. *Lancet* 1981; 1: 1225-1228.
87. Gottlieb SO, Weisfeldt ML, Ouyang P, et al. Effect of the addition of propranolol to therapy with nifedipine for unstable angina pectoris: a randomized, double-blind, placebo-controlled trial. *Circulation* 1986; 73: 331-337.
88. Lubsen J, Tijssen JG. Efficacy of nifedipine and metoprolol in the early treatment of unstable angina in the coronary care unit: findings from the Holland Interuniversity Nifedipine/metoprolol Trial (HINT). *Am J Cardiol* 1987; 60: 18A-25A.
89. Yusuf S, Wittes J, Friedman L. Overview of results of randomized clinical trials in heart disease. II. Unstable angina, heart failure, primary prevention with aspirin, and risk factor modification. *JAMA* 1988; 260: 2259-2263.
90. Mehta J, Conti CR. Verapamil therapy for unstable angina pectoris: review of double-blind placebo-controlled randomized clinical trials. *Am J Cardiol* 1982; 50: 919-922.
91. Theroux P, Taeymans Y, Morissette D, et al. A randomized study comparing propranolol and diltiazem in the treatment of unstable angina. *J Am Coll Cardiol* 1985; 5: 717-722.
92. Parodi O, Simonetti I, Michelassi C, et al. Comparison of verapamil and propranolol therapy for angina pectoris at rest: a randomized, multiple-crossover, controlled trial in the coronary care unit. *Am J Cardiol* 1986; 57: 899-906.
93. Kennedy JA, Unger SA, Horowitz JD. Inhibition of carnitine palmitoyltransferase-1 in rat heart and liver by perhexiline and amiodarone. *Biochem Pharmacol* 1996; 52: 273-280.
94. Cole PL, Beamer AD, McGowan N, et al. Efficacy and safety of perhexiline maleate in refractory angina. A double-blind placebo-controlled clinical trial of a novel antianginal agent. *Circulation* 1990; 81: 1260-1270.
95. Stewart S, Voss DW, Northey DL, Horowitz JD. Relationship between plasma perhexiline concentration and symptomatic status during short-term perhexiline therapy. *Ther Drug Monit* 1996; 18: 635-639.
96. Oler A, Whooley MA, Oler J, Grady D. Adding heparin to aspirin reduces the incidence of myocardial infarction and death in patients with unstable angina. A meta-analysis. *JAMA* 1996; 276: 811-815.
97. Becker RC, Cannon CP, Tracy RP, et al. Relation between systemic anticoagulation as determined by activated partial thromboplastin time and heparin measurements and in-hospital clinical events in unstable angina and non-Q wave myocardial infarction. Thrombolysis in Myocardial Ischemia III B Investigators. *Am Heart J* 1996; 131: 421-433.
98. Chong BH. Heparin induced thrombocytopenia. *Br J Haematol* 1995; 89: 431-439.
99. Massonnet-Castel S, Pelissier E, Bara L, et al. Partial reversal of low molecular weight heparin (PK 10169) anti-Xa activity by protamine sulfate: in vitro and in vivo study during cardiac surgery with extracorporeal circulation. *Haemostasis* 1986; 16: 139-146.
100. Van Ryn-McKenna J, Cai L, Ofosu FA, et al. Neutralization of enoxaparine-induced bleeding by protamine sulfate. *Thromb Haemost* 1990; 63: 271-274.
101. FRISC Investigators. Low-molecular-weight heparin during instability in coronary artery disease, Fragmin during Instability in Coronary Artery Disease. *Lancet* 1996; 347: 561-568.
102. Klein W, Buchwald A, Hillis SE, et al. Comparison of low-molecular-weight heparin with unfractionated heparin acutely and with placebo for 6 weeks in the management of unstable coronary artery disease. Fragmin in unstable coronary artery disease study (FRIC). *Circulation* 1997; 96: 61-68.
103. Comparison of two treatment durations (6 days and 14 days) of a low molecular weight heparin with a 6-day treatment of unfractionated heparin in the initial management of unstable angina or non-Q wave myocardial infarction: FRAX.I.S. (FRAxiparine in Ischaemic Syndrome). *Eur Heart J* 1999; 20: 1553-1562.
104. Cohen M, Demers C, Gurfinkel EP, et al. A comparison of low-molecular-weight heparin with unfractionated heparin for unstable coronary artery disease. Efficacy and Safety of Subcutaneous Enoxaparin in Non-Q-Wave Coronary Events Study Group. *N Engl J Med* 1997; 337: 447-452.
105. Antman EM, McCabe CH, Gurfinkel EP, et al. Enoxaparin prevents death and cardiac ischemic events in unstable Angina/Non-Q-wave myocardial infarction: results of the thrombolysis In myocardial infarction (TIMI) 11B trial. *Circulation* 1999; 100: 1593-1601.
106. Antman EM, Cohen M, Radley D, et al. Assessment of the treatment effect of enoxaparin for unstable angina/non-Q-wave myocardial infarction. TIMI 11B-ESSENCE meta-analysis. *Circulation* 1999; 100: 1602-1608.
107. Goodman S, Langer A, Demers C, et al. One year follow-up of the ESSENCE trial: sustained clinical benefit. *Can J Cardiol* 1998; 14: 122F.
108. Mark DB, Cowper PA, Berkowitz SD, et al. Economic assessment of low-molecular-weight heparin (enoxaparin) versus unfractionated heparin in acute coronary syndrome patients: results from the ESSENCE randomized trial. Efficacy and Safety of Subcutaneous Enoxaparin in Non-Q wave Coronary Events [unstable angina or non-Q-wave myocardial infarction]. *Circulation* 1998; 97: 1702-1707.
109. O'Brien BJ, Willan A, Blackhouse G, et al. Will the use of low-molecular-weight heparin (enoxaparin) in patients with acute coronary syndrome save costs in Canada? *Am Heart J* 2000; 139: 423-429.
110. Hirsh J. Low-molecular-weight heparin: a review of the results of recent studies of the treatment of venous thromboembolism and unstable angina. *Circulation* 1998; 98: 1575-1582.
111. Antman EA, Handin R. Low-molecular-weight heparins. An intriguing new twist with profound implications. *Circulation* 1998; 98: 294-299.
112. Montelescot G, Phillippe F, Ankri A, et al. Early increase of von Willebrand factor predicts adverse outcome in unstable coronary artery disease. Beneficial effects of enoxaparin. *Circulation* 1998; 98: 294-299.
113. Long-term low-molecular-mass heparin in unstable coronary-artery disease: FRISC II prospective randomised multicentre study. FRISC II Investigators. *Lancet* 1999; 354: 701-707.
114. Effects of recombinant hirudin (lepirudin) compared with heparin on death, myocardial infarction, refractory angina, and revascularisation procedures in patients with acute myocardial ischaemia without ST elevation: a randomised trial. Organisation to Assess Strategies for Ischemic Syndromes (OASIS-2) Investigators. *Lancet* 1999; 353: 429-438.
115. A low molecular weight, selective thrombin inhibitor, inogatran, vs heparin, in unstable coronary artery disease in 1209 patients. A double-blind, randomized, dose-finding study. Thrombin inhibition in Myocardial Ischaemia (TRIM) study group. *Eur Heart J* 1997; 18: 1416-1425.
116. A comparison of recombinant hirudin with heparin for the treatment of acute coronary syndromes. The Global Use of Strategies to Open Occluded Coronary Arteries (GUSTO) IIb investigators. *N Engl J Med* 1996; 335: 775-782.
117. Rao AK, Sun L, Chesebro JH, et al. Distinct effects of recombinant desulfatohirudin (Revasc) and heparin on plasma levels of fibrinopeptide A and prothrombin fragment F1.2 in unstable angina. A multicenter trial. *Circulation* 1996; 94: 2389-2395.
118. Effects of tissue plasminogen activator and a comparison of early invasive and conservative strategies in unstable angina and non-Q-wave myocardial infarction. Results of the TIMI IIIB Trial. Thrombolysis in Myocardial Ischemia. *Circulation* 1994; 89: 1545-1556.
119. Cohen M, Adams PC, Parry G, et al. Combination antithrombotic therapy in unstable rest angina and non-Q- wave infarction in nonprior aspirin users. Primary end points analysis from the ATACS trial. Antithrombotic Therapy in Acute Coronary Syndromes Research Group. *Circulation* 1994; 89: 81-88.
120. Anand SS, Yusuf S, Pogue J, et al. Long-term oral anticoagulant therapy in patients with unstable angina or suspected non-Q-wave myocardial infarction: organization to assess strategies for ischemic syndromes (OASIS) pilot study results. *Circulation* 1998; 98: 1064-1070.
121. Inhibition of the platelet glycoprotein IIb/IIIa receptor with tirofiban in unstable angina and non-Q-wave myocardial infarction. Platelet Receptor Inhibition in Ischemic Syndrome Management in Patients Limited by Unstable Signs and Symptoms (PRISM-PLUS) Study Investigators. *N Engl J Med* 1998; 338: 1488-1497.

Summary of recommendations with levels of evidence

Prehospital care

- Patients with a history of rest or prolonged chest discomfort (longer than 10 minutes, not relieved by sublingual nitrates), recurrent chest discomfort, or discomfort associated with syncope or heart failure are considered a medical emergency and should be transferred by ambulance to a hospital for urgent assessment (*E4*).

Acute hospital assessment and triage

- The following investigations should be carried out for patients presenting with rest or prolonged chest or arm discomfort:
  - Baseline tests: electrocardiography (ECG), serum cardiac troponin (I or T) level (or serum CK-MB level if troponin testing is not available), full blood count, serum creatinine and electrolyte levels, serum CK levels, serum lipids levels (within 24 hours of onset of acute coronary syndrome), blood glucose level and chest x-ray.
  - The ECG is the sole test required to select patients for emergency reperfusion (thrombolytic therapy or direct PCI).
  - Continuous ECG monitoring of heart rhythm (ST segment monitoring is desirable, if available) and serial ECGs should be performed in patients with high and intermediate risk features.
  - Ongoing chest discomfort requires frequent follow-up 12-lead ECGs (15 minutes apart if continuous ST segment monitoring is unavailable) to rapidly diagnose eligibility for a reperfusion strategy.
  - A repeat serum troponin assay (unless already positive) should be performed at least six hours after presentation.
  - In patients with myocardial infarction, serial measurements of total CK should be performed for 48 hours, so that if reinfarction is suspected later total CK can be remeasured to confirm a second event. (A specific marker, such as CK-MB, is not required for the diagnosis of reinfarction, and troponin is not useful for diagnosing early reinfarction, as it remains elevated for 7–10 days.)
- All patients presenting with chest discomfort should have an ECG completed within five minutes of arrival at a medical facility and be assessed by the most appropriately qualified available person (*E4*).
- Patients with chest discomfort should be evaluated with a careful history and physical examination completed within 10 minutes of arrival at a medical facility (*E4*).
- High and intermediate risk patients should be admitted for continuous ECG monitoring, intensive medical therapy and observation by appropriately qualified nursing and medical staff (*E1*).
- Recurrent chest discomfort (including in patients observed in a general ward environment) requires urgent reassessment, including immediate ECG, by the most appropriately qualified person.
- Patients whose ECG shows ST segment elevation or left bundle branch block should be immediately considered for reperfusion therapy (*E1*).

Management

Low risk patients

- Low risk patients with a history of a moderate increase in angina frequency or severity, or angina onset more than two weeks before presentation, can be referred for cardiac assessment within two weeks (*E4*).

Intermediate risk patients

- Intermediate risk patients who develop further ischaemia (pain or dynamic ST deviation) or positive serum markers are reclassified as high risk (*E1*).
- Intermediate risk patients need not be treated with low molecular weight heparin unless reclassified into a high-risk group (*E2*).
- Intermediate risk patients who have no further pain, have a normal ECG for the period of observation, have normal serum markers at baseline and six hours after presentation and a normal stress test may be reclassified as being at low risk (*E1*).
- Patients with documented coronary artery disease without high-risk features are usually managed by increasing anti-anginal therapy, followed by a period of observation for clinical stability. Investigation will depend on the clinical context (*E4*).
- Patients with symptoms strongly suggestive of myocardial ischaemia, but normal ECGs and serum markers, may be considered for either further provocative testing (stress radionuclide or stress echocardiography) and outpatient cardiac follow-up or coronary angiography in selected patients (*E4*).

continued over

High risk pa tients

Intensive medical management

- In addition to aspirin, either LMW heparin or intravenous tirofiban with unfractionated heparin is recommended in high risk patients (*E1*), and in geographically isolated patients requiring transfer to a tertiary facility, or patients not suitable for an invasive approach (very elderly, severe comorbidities) (*E4*).
- Intravenous tirofiban and unfractionated heparin are particularly recommended where LMW heparin fails (*E4*), and in high-risk patients for whom an invasive strategy is planned (*E2*).

Early invasive approach

- With the exception of patients of advanced age or with severe or multiple comorbidities, an early invasive approach should be considered in patients with the following high-risk features:
  - pain or ischaemia refractory to medical therapy (*E4*);
  - electrocardiographic changes (ST-segment depression or T-wave inversion in multiple leads) (*E2*);
  - positive serum markers (troponin I or T) (*E2*);
  - associated heart failure or haemodynamic instability (*E3*);
  - high-risk features on early exercise testing (*E3*); or
  - recent myocardial infarction or revascularisation (*E3*).

  In rural centres and units that lack invasive facilities, these markers may be used to determine which patients may benefit from transfer for an invasive approach.
- The complementary use of aggressive medical and invasive approaches should be considered in all patients with high-risk features (*E1*).

Long term management

- Statin therapy should be considered in all patients following unstable angina or non-ST elevation myocardial infarction if serum cholesterol level is greater than 4 mmol/L (*E2*).
- Angiotensin-converting enzyme (ACE) inhibitors should be considered, particularly if hypertension or diabetes is present (*E2*).
- All patients with a diagnosis of unstable angina or myocardial infarction should be referred to, and participate in, an appropriate cardiac rehabilitation and prevention program (*E1*).

E1 Level I: Evidence obtained from a systematic review of all relevant randomised controlled trials.

E2 Level II: Evidence obtained from at least one properly designed randomised controlled trial.

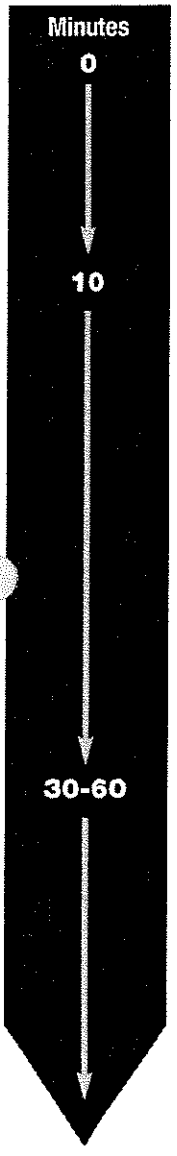
E3 Level III: Evidence obtained from all well-designed controlled trials without randomisation, well-designed cohort or case–control analytic studies, preferably from more than one centre or research group, or from multiple time series with or without the intervention. Dramatic results in uncontrolled experiments (such as the results of the introduction of penicillin treatment in the 1940s) could also be regarded as this type of evidence;

E4 Level IV: Opinions of respected authorities, based on clinical experience, descriptive studies, or reports of expert committees.

National Health and Medical Research Council, 1995

Reperfusion Therapy Algorithm

Emergency department guidelines for patients with chest pain or discomfort



Chest pain / Discomfort

- ☐ Commence oxygen and ECG monitoring
- ☐ Insert a cannula + obtain 12 lead ECG immediately
- ☐ Pain relief
- ☐ Give aspirin 150–300 mg unless already given or allergic

↓

Immediate ECG

↓

Doctor to see patient within 10 minutes of arrival
(National triage category 1 or 2)

↓

Meets indications for reperfusion therapy?* ..... NO .....➤ **If patient doesn't meet indications then continue to monitor:**
Refer to Indications for reperfusion (below)

- Chest pain
- ECG every 15 mins
- Troponin

- **Re-evaluate & risk stratify**

± Cardiology consult

YES

↓

Reperfusion options

Primary angioplasty

For all if:

- Facilities available and door-to-balloon ≤ 60 mins
- Contraindications to fibrinolytic Rx**
- For potentially large anterior AMI if balloon available within 4 hours
- For haemodynamic instability / cardiogenic shock

Fibrinolysis
Aim: Door-to-needle ≤ 30 mins

↓

Contraindications to fibrinolysis?**
Refer to Contraindications for fibrinolysis (below)

YES → **Consider:**
- transfer for angioplasty
or
- follow usual practice

NO → **Give:**
appropriate fibrinolytic agent as soon as possible

↓

Transfer to CCU / HDU as soon as possible

*** Indications for reperfusion (angioplasty / stent; fibrinolysis)**

Ischaemic / infarction symptoms > 20 minutes
(chest pain, discomfort or pressure; shortness of breath; pulmonary oedema; sweating; dizziness & light-headedness)

Onset of symptoms within 12 hours

ECG changes
ST elevation ≥ 1 mm in 2 contiguous limb leads
ST elevation ≥ 2 mm in 2 contiguous chest leads
Left Bundle Branch Block (LBBB)

**** Contraindications for fibrinolysis**

Risk of bleeding
Active bleeding
Recent (< 1 month) major surgery or trauma

Risk of intracranial haemorrhage
Haemorrhagic stroke ever, or ischaemic stroke within a year
Anatomical abnormalities, intracerebral neoplasm, AV malformation

Relative contraindications for fibrinolysis

Risk of bleeding
Prior use of anticoagulants, INR greater than 2.0
Non-compressible vascular punctures
Prolonged cardiopulmonary resuscitation (> 10 minutes)

Risk of intracranial haemorrhage
Severe hypertension that cannot be controlled (BP > 180 mmHg systolic and / or 110 mmHg diastolic),
Previous stroke at anytime
Previous TIA

Other
Pregnancy

Many contraindications are relative and potential benefits versus relative risk should always be considered.

Heart Foundation

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INFORMATION

Guidelines

Reperfusion Therapy For Acute Myocardial Infarction

Prepared by the Medical Issues Committee

The Heart Foundation would like to thank Dr Philip Aylward (Cardiologist, Flinders Medical Centre, Adelaide) and Dr David Hunt (Cardiologist, Royal Melbourne Hospital, Melbourne), the principal authors of these guidelines.

Preamble

The following National Heart Foundation of Australia (NHFA) guidelines are intended to assist physicians and other health care providers in the appropriate management of patients presenting with acute myocardial infarction with ST elevation requiring reperfusion therapy. The guidelines describe the key elements required to obtain optimum reperfusion and outcome for an individual patient with myocardial infarction. They try to encompass most circumstances that apply (e.g. metropolitan and country) and give broad boundaries for practice. It is recognised that the treatment of the individual patient will vary depending on a number of factors, some patient related (e.g. age, blood pressure, type of infarct, previous treatments), and some place related (e.g. availability of hospital, angioplasty and bypass surgery).

These guidelines are deliberately succinct and do not include all the classification of evidence. For a more detailed review of the evidence the reader is referred to the American College of Cardiology AHA Guidelines available on the web site of the American College of Cardiology (www.acc.org) and the American Heart Association (www.americanheart.org).

Acute myocardial infarction (AMI) is usually caused by the complete occlusion of a coronary artery by thrombus secondary to the rupture of an atherosclerotic plaque. Studies have shown that prompt and sustained restoration of blood flow to the myocardium reduces mortality and other complications of AMI.[1, 2, 3, 4]

In order to obtain optimum early treatment: the patient must recognise the symptoms of AMI and seek medical help; the correct diagnosis must be made by medical practitioners and the most appropriate reperfusion therapy must be chosen for a given patient.

Key issues

1. Chest pain with ST segment elevation on ECG must be managed speedily and aggressively to produce the best outcome.
2. Patient should have initial assessment in the Emergency Department within 10 minutes of arrival (ECG within 5 minutes of arrival).
3. Time to reperfusion is the key to successful therapy. Fibrinolytic therapy should be delivered within 30 minutes of hospital arrival ('door to needle time'). Percutaneous coronary angioplasty (PTCA) should be performed within 60 minutes ('door to balloon time'). If it is not possible to reach a hospital with these facilities within 90 minutes, out-of-hospital reperfusion therapy should be considered.
4. The choice between primary angioplasty and fibrinolysis will depend on local circumstances and in particular time required before PTCA can be effected.
5. Choice of fibrinolytic agent will depend on patient characteristics, particularly age, infarct size, convenience and cost. New bolus fibrinolytic agents offer great convenience and widen the potential sites of fibrinolytic delivery (e.g. home, ambulance, small hospital).

6. Contraindications to fibrinolytic therapy are few but include active bleeding and risk of stroke. Hypertension on presentation increases the risk of stroke. Blood pressure should be lowered prior to delivery of fibrinolytic therapy which should not be given if blood pressure remains above 200mm Hg systolic and/or 110mm Hg diastolic.
7. Combination of PTCA and fibrinolytic therapy (rescue) should be considered if ST segment elevation on the ECG has not resolved more than 70% within 60 minutes of commencement of thrombolytic therapy.
8. Cardiogenic shock should be treated aggressively with balloon pump support and early angiography with a view to early revascularisation.

Patient Presentation

Delays in presentation have been documented worldwide and within Australia.[5, 6] The delays in presentation relate to failure to recognise symptoms or denial of symptoms. Ongoing education for the public, particularly cardiac patients, about the need to seek early treatment is vital. Advice to patients will vary between metropolitan and rural centres.

(i) Metropolitan

Patients should be advised that they, their relative or work colleague should call an ambulance to take them to the nearest appropriate hospital. The patient should not drive themselves to the hospital due to the real danger of ventricular fibrillation in the early hours of myocardial infarction.

If the patient contacts their general practitioner (GP), the GP should call an ambulance and advise the patient to take an Aspirin unless contraindicated (see below). The GP should only attend the patient if they can get there prior to the ambulance. They should not advise the patient to attend the surgery.

If a patient attends the GP or physician's rooms, in addition to calling an ambulance, an intravenous line should be inserted if possible.

Most ambulances now contain defibrillators and are able to treat ventricular fibrillation, the fatal early complication of AMI.

At this time the NHF does not recommend pre-hospital treatment in the metropolitan area. This is because the transport times in general are short (15-20 minutes) and the time necessary to make the diagnosis and confirm the ECG as showing ST elevation would be better spent transporting the patient to hospital. It would be anticipated that with improvements in technology ECGs may be transmitted to the receiving hospital. This will improve door to treatment times and lay the potential groundwork for future out-of-hospital treatment.

(ii) Rural and Remote *

Clearly it may not be feasible to get to a major hospital in a short period of time in rural and remote areas. The best arrangement will vary depending on distance, availability of GPs or other medical officers and ambulance services and the accredited skills of the nurse attending the patient. The patient may be driven to hospital, call the ambulance, including the Royal Flying Doctor Service, or a medical practitioner. The course of action will depend on the particular situation and geographic location. Most hospitals in rural and remote areas can now deliver fibrinolytic therapy. Doctors serving rural and remote communities should ideally be able to deliver fibrinolytic therapy at home.[7] It may be appropriate for accredited nurses and ambulance officers to deliver fibrinolytic therapy providing the ECG can be confirmed either at the local hospital or at a larger centre prior to therapy being given.

Patient triage

The diagnosis of AMI and the recognition of the need for reperfusion therapy will depend on the history, examination and ECG. Patients admitted to the emergency department with

* It is recognised that the definition of 'rural' and 'remote' and associated issues relating to professional healthcare responsibilities are complex. These guidelines should be read in the context of the particular situation.

chest pain or discomfort should be treated as emergencies and should be regarded as qualifying for the Australasian College for Emergency Medicine triage category 2. The ECG should be completed within 5 minutes of arrival and reviewed immediately by a medical officer, and a careful and brief history and physical examination completed within 10 minutes of arrival.

A patient with clinical features of AMI in the preceding 12 hours without contraindications and with appropriate ECG changes should be offered reperfusion therapy.[2, 8, 9]

(i) History

Will include current illness, previous cardiac history, contraindications to fibrinolytic therapy, particularly the risk of stroke and bleeding (see Table 1).

(ii) Examination

Will include assessment of haemodynamic stability and exclusion of other causes of symptoms (eg pericarditis, aortic dissection, perforated ulcer or pneumothorax).

(iii) ECG

Reperfusion therapy is appropriate for patients with ST elevation on their ECG ≥ 1 mm in 2 contiguous limb leads, ≥ 2 mm in two contiguous chest leads, and for patients with left bundle branch block.

In general, patients with ST depression should not be treated with acute reperfusion therapy. The exception to this is when it is suspected that anterior ST depression represents a posterior infarct. An ECG with chest leads one intercostal space higher may be helpful in this situation which will often show lateral ST elevation.

A patient with a normal ECG should not be treated with reperfusion therapy.

Patients who are haemodynamically unstable with cardiogenic shock will need aggressive management and early assessment of coronary anatomy with a view to revascularisation by angioplasty or surgery. Where possible a balloon pump should be inserted early.[10]

Table 1: Patient Selection for Reperfusion Therapy

Indications
Ischaemic/Infarction symptoms > 20 minutes. This would include not only chest pain but other symptoms of myocardial infarction, such as chest discomfort or pressure, shortness of breath, pulmonary oedema, sweating, dizziness, and light-headedness.
Patient's symptoms commenced within 12 hours.
ST elevation or left bundle branch block on ECG.
No contraindications to reperfusion therapy.

Choice of reperfusion therapy

The choice of reperfusion therapy in the individual patient will depend on the facilities available, contraindications for an individual patient and cost. The aim should be the earliest possible successful reperfusion with the least risk of complications and in the most cost-effective manner.

Of note, the important time is the time to reperfusion, not necessarily the time to treatment.

Coronary angioplasty

In Australia, coronary angioplasty is available in a limited number of centres in the usual time of catheter laboratory operation and in even fewer out-of-hours. Coronary angioplasty has been shown in relatively small trials to reduce mortality and stroke compared to fibrinolytic therapy.[4, 11] Practice registries from the United States suggest that outside clinical trials, angioplasty is equal to but not necessarily better than fibrinolytic therapy.

The major issue with coronary angioplasty is the delay to re-opening the artery as the cardiac catheter laboratory may not be immediately available, particularly out of hours.

As in all areas of Cardiology, the field is moving rapidly and with the use of intracoronary stents and adjunctive glycoprotein IIb/IIIa inhibitors better results may be obtained. At this time the use of adjunctive glycoprotein IIb/IIIa inhibitors would be recommended. The use of a coronary stent in this situation has not been totally clarified. There is evidence that stents provide better long-term outcomes, but there is some debate about their use in the acute situation.[12] Their use would be up to the individual cardiologist.

Currently coronary angioplasty/coronary stenting is recommended for:

1. All patients – if reperfusion could be obtained within 1 hour of the decision to treat.
2. Patients with contraindications to fibrinolytic therapy. This may require transfer to an angioplasty centre.
3. Patients with major haemodynamic instability, particularly cardiogenic shock.
4. Patients with potentially large anterior infarcts if reperfusion could be obtained within 4 hours.

Rescue angioplasty

Rescue angioplasty is increasingly utilised when patients with large infarcts have not clinically reperfused at 60-90 minutes after thrombolytic therapy as indicated by failure of >70% ST segment elevation resolution. There is little hard data to support this treatment. The RESCUE trials showed some minor benefits in left ventricular function but angioplasty was performed some hours after the onset of chest pain and thrombolytic therapy.[13] Based on data that TIMI III flow at 90 minutes is a major predictor of outcome[14] and that >70% ST segment resolution on the ECG by 90 minutes also predicts outcome, it would appear reasonable to try to obtain reperfusion by angioplasty in this situation. However, no firm recommendations can be made at this stage.

Fibrinolytic therapy

Fibrinolytic therapy remains the most common form of reperfusion therapy in Australia when there is no contraindication.

Contraindications to fibrinolytic therapy (See Table 2)

Fibrinolytic therapy is contraindicated for people who are at high risk of significant bleeding, particularly intracerebral haemorrhage.

Contraindications include active bleeding and recent surgery or trauma within a month. Other conditions which put the patient at risk of bleeding, such as recent arterial puncture, prolonged cardiopulmonary resuscitation, prior use of oral anticoagulants with an elevated INR, are relative contraindications.

The devastating complication of fibrinolytic therapy is intracerebral haemorrhage and patients with high risk of this should be treated with caution. Previous haemorrhagic stroke or recent ischaemic stroke within a year, known intracerebral tumours or intracranial anatomical abnormalities are contraindications. Hypertension at presentation is a major risk factor of stroke and should be lowered if possible by pain relief and intravenous nitroglycerine. If it is not possible to lower the blood pressure below 180 mm Hg systolic and 110 mm Hg diastolic then fibrinolytic therapy may be inadvisable.

Age per se is not a contraindication to fibrinolytic therapy as, although there is increased risk of bleeding and stroke, the risk of death from the myocardial infarct is also much greater. It may influence the choice of reperfusion therapy.

The decision to use fibrinolytic therapy in the face of contraindications will depend on the potential infarct size and other available treatments.

Table 2: Contraindications to Fibrinolytic Therapy

ABSOLUTE CONTRAINDICATIONS
(a) Risk of bleeding • Active bleeding • Recent (less than 1 month) major surgery or trauma (b) Risk of intracranial haemorrhage • History of haemorrhagic stroke ever, or ischaemic stroke within a year • Anatomical abnormalities, intracerebral neoplasm, AV malformation
RELATIVE CONTRAINDICATIONS
(a) Risk of bleeding • Prior use of anticoagulants, INR greater than 2.0 • Non-compressible vascular punctures • Prolonged cardiopulmonary resuscitation (>10 minutes) (b) Risk of intracranial haemorrhage • Previous stroke at anytime • Previous TIA • Severe hypertension that cannot be controlled, blood pressure >180 mm Hg systolic and/or >110 mm Hg diastolic (c) Other • Pregnancy

Fibrinolytic agents (See Table 3)

There are four fibrinolytic agents currently available in Australia – Streptokinase, tissue plasminogen activator (Alteplase), r-PA (Reteplase) and TNK t-PA (Tenecteplase)(Table 3).

Streptokinase

Streptokinase was the first fibrinolytic agent available and has been shown to reduce mortality in acute myocardial infarction. It saves approximately 25 lives per thousand patients treated[2] and is associated with an average risk of non-fatal intracerebral bleeding of around 3/1000 – this risk is higher in older patients. Streptokinase reduces infarct mortality by 25% and so will provide more absolute benefit in patients with a higher predicted infarct mortality rate. The benefit is higher the earlier the treatment is given.

It is given as an infusion of 1.5 million units intravenously, usually over 60 minutes although many centres give it more rapidly, as quickly as over 30 minutes.

Streptokinase is a naturally occurring product of streptococcus and will induce antibodies and thus has the potential for allergic reactions.

Clinical problems related to Streptokinase include:

1. Hypotension due to vasodilation – this will occur in up to 1/3 of patients and is in part dependent upon the speed of the infusion. Slowing down of the infusion, elevation of the foot of the bed or treatment with intravenous fluid will usually resolve the problem.
2. Allergy – this will often result in a rash or hives but can occasionally be associated with true anaphylaxis. Late allergic reactions have been described.

3. Antibodies will develop within about five days of administration and persist for many years. These antibodies will not only potentially give rise to allergy if a second dose is given but may also render it ineffective. Current policy would suggest that Streptokinase should only be given once and the patient clearly told of potential problems with future reuse.

   High levels of antibodies may also be present in groups with a high rate of exposure to Streptococcal infections such as some groups of Aboriginal peoples and Torres Strait Islanders.

The major advantage of Streptokinase is its low cost of approximately $200 per dose. It remains the most commonly used fibrinolytic in Australia.

Tissue Plasminogen Activator Alteplase

Tissue plasminogen activator is a recombinant product of a naturally occurring protein. It produces improved reperfusion compared to Streptokinase (TIMI III flow 55% versus 30% at 90 minutes) resulting in an extra 10 lives per thousand patients treated being saved. It is also associated with a slightly higher intracranial haemorrhage rate producing one extra disabling stroke per thousand patients treated.

The major disadvantage of t-PA is cost of approximately $2,000 per dose.

r-PA *(Reteplase)*

Reteplase is a variant of t-PA produced by genetic engineering. Its major advantage is that it is delivered as a double bolus of 10 units 30 minutes apart. In a large comparative study[15] it was shown to be clinically equivalent to t-PA. The cost is similar to t-PA.

TNK t-PA *(Tenecteplase)*

Tenecteplase is also variant of t-PA produced by genetic engineering. Its advantage is that it is delivered as a single bolus. A large comparitive study has shown it to be equivalent to Alteplase in its effect on 30 day mortality.[16] The cost is similar to t-PA.

Table 3: Fibrinolytic Agents

	Streptokinase	**Alteplase**	**Reteplase**	**Tenecteplase**
Dose (also see product information)	1.5 million international units in 30-60 min	100mg in 90 min on basis of bodyweight	10 units x 2 30 min apart	Up to 10, 000 international units on basis
Bolus administration	No	No	Yes	Yes
Antigenic	Yes	No	No	No
Systemic fibrinogen depletion	Marked	Mild	Moderate	Minimal
Lives saved per 1000 treated	25 approx.	35 approx.	35 approx.	35 approx.
Cost per dose	$200 approx.	$2000 approx.	$2000 approx.	$2000 approx.

Choice of fibrinolytic agent

In general in Australia, Streptokinase remains the drug of first choice. However, the plasminogen activators are increasingly utilised. The more expensive agents are recommended for high risk patients who have been shown to benefit in trials. These include:

- Patients under the age of 75 having a large myocardial infarction either anterior or inferior with associated anterior ST segment depression, especially if they present early.
- Patients who have previously received Streptokinase.

In addition, the advantage of bolus administration with r-PA or TNK t-PA must be recognised. It is of particular benefit in busy Emergency Departments and coronary care units and its ease of administration makes it suitable for country and smaller hospitals.

Bolus fibrinolytic drugs increase the potential for out of hospital treatment or treatment in places from which the patient may be readily transferred. It may be appropriate to give the first dose at an emergency centre which does not have a coronary care unit and transfer the patients to the coronary care unit for the second bolus. (r-PA)

Further improvements in fibrinolytic therapy include the currently experimental combination of fibrinolytic agents with glycoprotein IIb/IIIa inhibitors and low molecular weight heparin.

Adjunctive therapy for fibrinolytic therapy

Aspirin

Aspirin is indicated for all patients with acute myocardial infarction unless contraindications exist. The patient should be advised to take this while still at home or it should be given in the ambulance while in transit to hospital. The initial dose should be 150-300 mg. Aspirin should be continued daily thereafter unless there are definite contraindications.

Heparin

Unfractionated Heparin is currently advised for use with both t-PA, r-PA and TNK t-PA and is optional and of unproven value with Streptokinase. Recent data suggests lower initial doses of Heparin may be more appropriate. The ACC/AHA guidelines recommend an initial bolus of 60 units/kg maximum 4,000 units and an initial infusion of 12 units/kg per hour. An APTT of 50-70 seconds should be obtained by regular monitoring and appropriate adjustment of Heparin infusion and re-bolusing if necessary. Heparin should be continued for at least 24 hours. The use of a Heparin nomogram and bedside monitoring may be helpful.

Low molecular weight heparin

At this time there is little data for the use of low molecular weight heparin with fibrinolytic therapy. The recent HART II Study[17] showed that the combination of enoxaparin with t-PA produced equal patency rates, and lower reocclusion rates than standard unfractionated Heparin with no adverse safety profile. Further data will be required concerning use of this agent with other thrombolytics before it could be routinely recommended.

Other management

All other aspects of the patient with an infarct have to be treated. Due care and attention should be given to rest and reassurance, monitoring, oxygen therapy, pain relief with whatever means are necessary and transfer to an appropriate environment for ongoing care.

Statin therapy should be considered in all patients following reperfusion if serum cholesterol is > 4 mmol/L.

All patients with a diagnosis of coronary heart disease should be referred to and participate in an appropriate cardiac rehabilitation and prevention program, and in consultation with their general practitioner have ongoing attention to a healthy lifestyle including appropriate physical activity, healthy eating, weight management and no smoking.

References

1 Gruppo Italiano per lo Studio della Streptochinasi nell'Infarto Miocardico (GISSI)). Effectiveness of intravenous thrombolytic treatment in acute myocardial infarction. Lancet 1986; 1: 397-402.

2 ISIS-2 (Second International Study of Infarct Survival) Collaborative Group. Randomised trial of intravenous streptokinase, oral aspirin, both or neither among 17,187 cases of suspected acute myocardial infarction:ISIS-2. Lancet 1988; 2: 349-360.

3 The GUSTO investigators. An international randomized trial comparing four thrombolytic strategies for acute myocardial infarction. N Engl J Med 1993; 329 :673-682.

4 Grines CL, Browne KF, Marco J, et al. A comparison of immediate angioplasty with thrombolytic therapy for acute myocardial infarction: the Primary Angioplasty in Myocardial Infarction Study Group. N Engl J Med 1993; 328: 673-679.

5 Bett N, Aroney G, Thompson P. Delays preceding admission to hospital and treatment with thrombolytic agents of patients with possible heart attack. Aust NZ J Med 1993; 23: 312–313.

6 Dracup K, McKinley SM, Moser DK. Australian patients delay in response to heart attack symptoms. MJA 1997; 166: 233–236.

7 GREAT Group. Feasibility, safety, and efficacy of domiciliary thrombolysis by general practitioners. Grampian Regional Early Anistreplase Trial. BMJ 1992; 305: 548–553.

8 Fibrinolytic Therapy Trialists' (FFT) Collaborative Group. Indications for fibrinolytic therapy in suspected acute myocardial infarction: collaborative overview of early mortality and major morbidity results from all randomised trials of more than 1000 patients. Lancet 1994; 343: 311–322.

9 Late Assessment of Thrombolytic Efficacy (LATE) study with alteplase 6–24 hours after onset of acute myocardial infarction. Lancet 1993; 342: 759–766.

10 Hochman JS, Sleeper LA, Webb JG, et al. Early revascularization in acute myocardial infarction complicated by cardiogenic shock. New Engl J Med 1999; 341: 625–634.

11 GUSTO IIb Angioplasty Substudy Investigators. A clinical trial comparing primary coronary angioplasty with tissue plasminogen activator for acute myocardial infarction: the Global Use of Strategies to Open Occluded Coronary Arteries in Acute Coronary Syndromes. N Engl J Med 1997; 336: 1621–1628.

12 Grines CL, Morice MD, Mattos L, et al. A prospective multicenter trial using the JJIS heparin-coated stent for reperfusion of acute myocardial infarction. J Am Coll Cardiol 1997; 29: Suppl 389A.

13 Ellis SG, Da Silva ER, Spaulding CM, et al. Review of immediate angioplasty after fibrinolytic therapy for acute myocardial infarction: Insights from the RESCUE I, RESCUE II, and other contemporary clinical experiences. Am Heart J 2000; 139: 1046-1053.

14 Simes RJ, Topol EJ, Holmes DR Jr, et al. Link between the angiographic substudy and mortality outcomes in a large randomized trial of myocardial reperfusion: Importance of early and complete infarct artery reperfusion. GUSTO-I Investigators. Circulation 1995; 91: 1923–1928.

15 Miller JM, Smalling R, Ohman EM, et al. Effectiveness of early coronary angioplasty and abciximab for failed thrombolysis (reteplase or alteplase) during acute myocardial infarction (results from the GUSTO-III trial). Global Use of Strategies to Open Occluded Coronary Arteries. Am J Cardiol 1999; 84: 779-784.

16 Assessment of the Safety and Efficacy of a New Thrombolytic (ASSENT-2) Investigators. Single bolus tenecteplase compared with front loaded alteplase in acute myocardial infarction. *Lancet* 1999; 354: 716-722.

17 Abstract. HART II. Late breaking clinical trials. ACC Annual Scientific Session, March 2000.