Managing chest pain in the emergency department

A resource for emergency nurses

Jeremy Pallas - 2014

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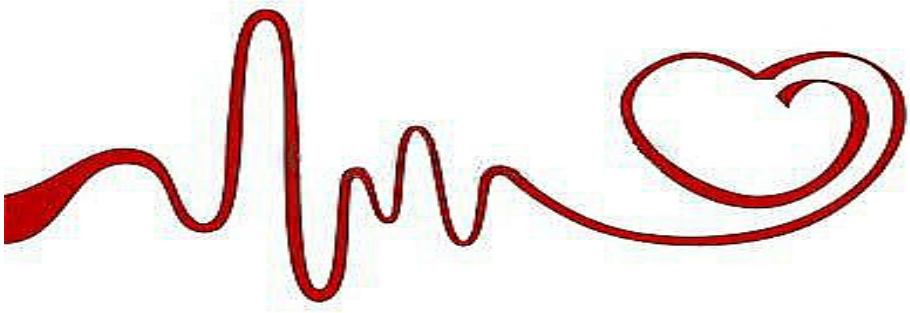
Why should you care?

- Heart disease is the leading cause of death worldwide.
- It accounted for 12.8% of all deaths worldwide in 2008 (WHO, 2010).
- In Australia, heart disease accounted for 16% of all deaths in 2009 and remains our leading cause of death overall (Australian Bureau of statistics, 2011).
- The incidence of heart disease is significantly higher in western society.
- In the emergency setting all patients suffering chest pain of presumed cardiac origin are to recieve an Australiasian triage category 2 and should be treated as a high priority (i.e clinical care initiated within 10 minutes).

Where do we start?

First of all you should have an idea of:

- What your dealing with
- Why your dealing with it
- How you can deal with it
- When you should deal with it

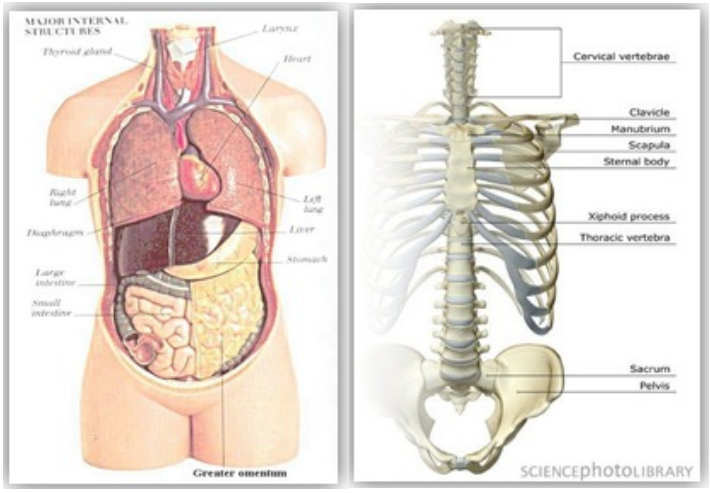


Anatomy of the chest

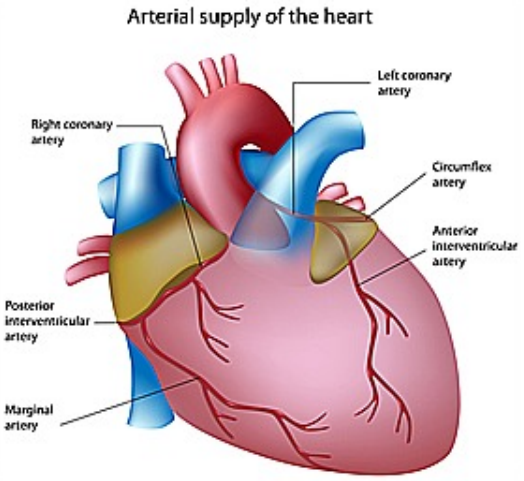
It is important to note that the human chest contains a number of vital structures including; the heart, lungs & major blood vessels

Other major organs including the liver, kidneys and digestive tract are also in close proximity

It is also important to remember that the human chest contains skin, bones, connective tissue and muscles.



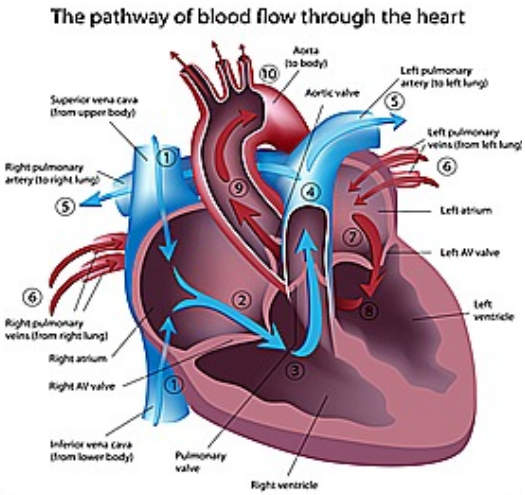
When looking at the anatomy of the heart it is important to consider:
The external anatomy which includes the major blood vessels (coronary arteries) responsible for perfusing the myocardium (heart muscle)
&
The internal anatomy which includes the different chambers (atrium and ventricle) and valves which prevent the backflow of blood.



During each heartbeat, myocardial contraction causes blood to flow into the right atrium where it is ejected into the right ventricle.

During contraction of the right ventricle blood is ejected into pulmonary circulation via the pulmonary artery before draining through the pulmonary veins into the left atrium

Blood is then pumped to the left ventricle where forceful contraction ejects blood to circulate around the body via the aorta.



Assessment

Primary survey

- The primary survey is systematic approach towards initial assessment which aims to rapidly identify any immediately life threatening conditions. This assessment is broken into 4 key categories:
  - **Airway**: look for secretions or foreign bodies, listen for auditory cues of obstruction
  - **Breathing**: look for chest movement, listen for breath sounds and feel for air movement
  - **Circulation**: look for skin colour or bleeding and feel for pulses.
  - **Disability**: assess neurological status, pupillary response and determine whether motor-sensory deficits are present.
- The primary survey allows us to identify and begin to correct any immediately life threatening conditions before moving on with our assessment of the patient.
- The primary survey can be attended very quickly and should form the basis of all patient assessment inside and outside the emergency department.
- A good place to start for those unfamiliar with this process is Australian Resuscitation Council's basic life support algorithm.
- This algorithm shows how dysfunction in any area of the primary survey gives us immediate priorities for treatment including CPR and advanced life support.

Initial assessment

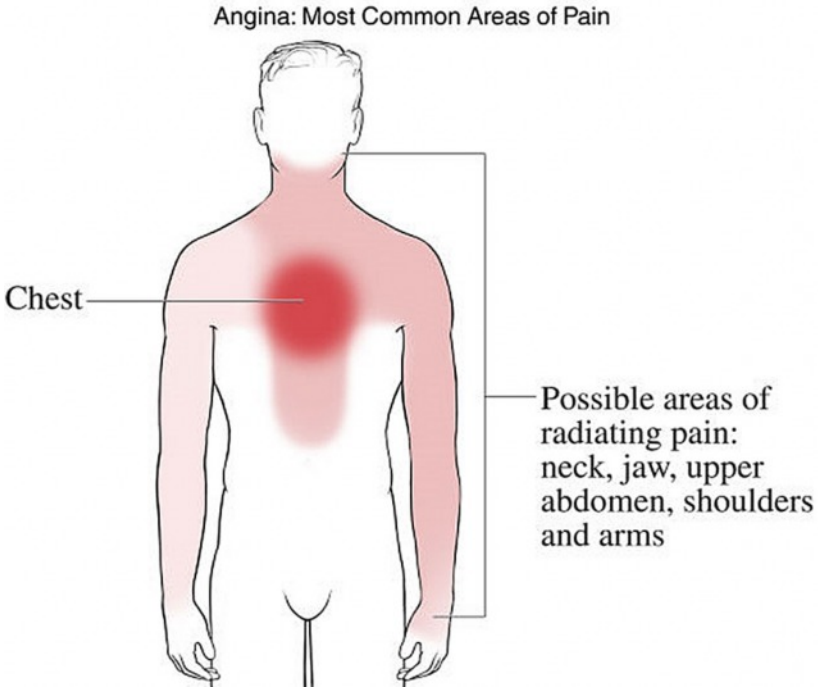
- Once the primary survey is completed. You should ensure you have collected a full set of vital signs including:
  - Pulse, Blood pressure, Respiration rate, Oxygen saturation & Temperature
- A baseline neurological assessment should be attended using the Glasgow Come Scale as a measurement.
- A brief patient history should be gathered including assessment of:
  - Presenting signs and symptoms
  - Complete medical history
  - Presence of major risk factors (hypertension, high cholesterol, diabetes, obesity etc)
  - List of current medication & allergies
- a brief physical exam should be attended including
  - Visualisation of the chest to identify any obvious injury of deformity (LOOK)
  - Auscultation of the chest (LISTEN)
  - Palpation the chest wall if required to identify tenderness or abnormalities such as subcutaneous emphysema or crepitus (crunching of bone on bone!) (FEEL)

Pain assessment

- It can be very difficult to differentiate cardiac chest pain from non-cardiac chest pain.
- The 'PQRST' pneumonic can be helpful in assessing a persons chest pain by identifying:
  - Provoking factors: was there anything that triggered, exacerbated or relieved the pain?
  - Quality: how does the patient describe the pain? i.e. sharp, aching, cramping, burning etc.
  - Region and Radiation: where is the pain? Does to radiate to any other part of the body?
  - Severity: how bad is the pain on a scale from worst to best? May be helpful to use a visual analogue scale which ranks pain on a scale of 1 (least) to 10 (most).
  - Time: when did the pain start? Is it constant or does it come and go?
- When assessing chest pain, quality is particularly important as many patients report feeling an ache, tightness or heaviness rather than pain. Some patients will actively deny pain but admit to feeling and abnormal sensation or pressure in the chest.
- For this reason it may be helpful to ask patients if they have chest 'discomfort' rather than chest pain.

Note

Patients experiencing acute myocardial infarction often report experiencing a feeling that something is going horribly wrong.
This is often referred to as a feeling of impending doom!
It is vital to consider the importance of communication and reassurance for these patients.



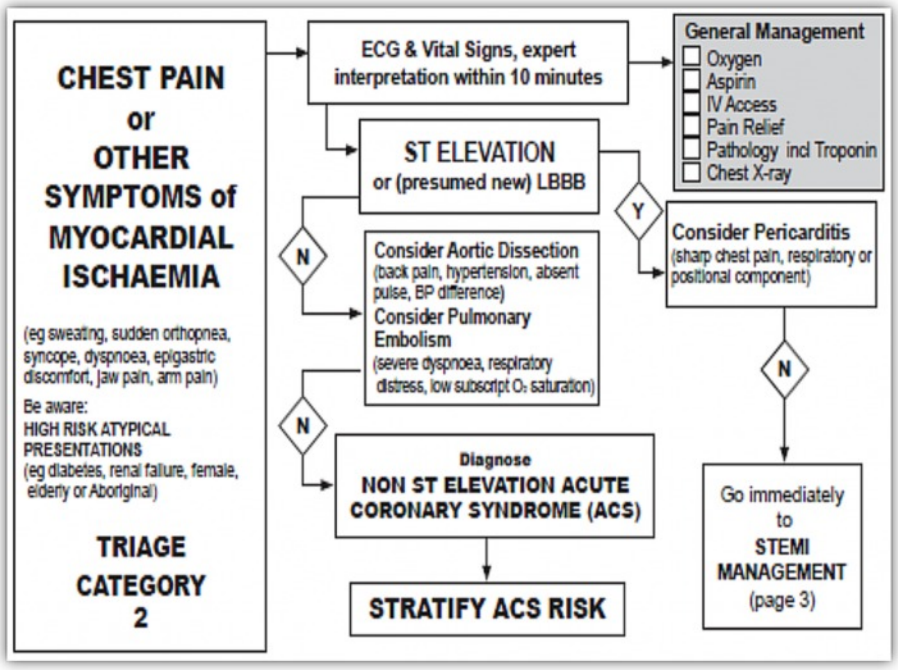
NSW chest pain pathway

- NSW health designed the chest pain pathway as a uniform guide detailing the minimum standard of care which should be provided to any patient presenting to NSW hospitals with chest pain.
- The pathway provides a flowchart for the assessment and treatment of chest pain as well as 2 separate diagrams detailing the treatment required for ischemic chest pain.
- Different versions of the pathway were developed for sites with primary PCI capability (i.e. John Hunter) and those without (i.e. Maitland, Mater, Belmont).

Important Notes

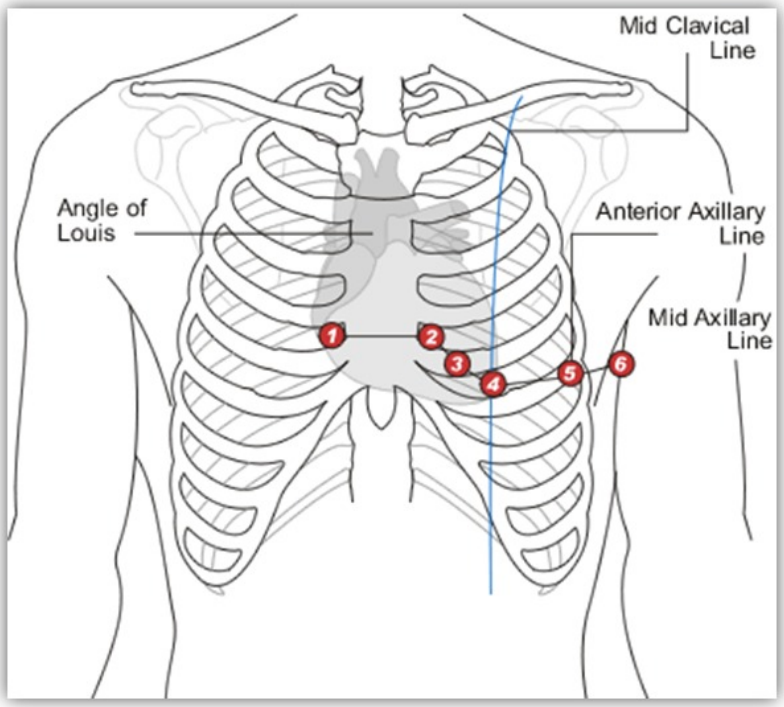
- Although this guide focuses predominantly on the management of chest pain according to the NSW health chest pain pathway, interventions relating to the primary survey including basic and advanced life support protocols always take precedence!
- This is particularly important when dealing with chest pain patients as they may be at an elevated risk of cardiorespiratory insufficiency or arrest.
- Further information about these resuscitation measures can be found in the *NSW health advanced life support package*
- It is essential to prioritise care i.e. if a patient is in respiratory distress, the application of supplemental oxygen may be required more immediately than an ECG.

The NSW chest pain pathway contains this flowchart detailing the immediate diagnostic and interventional requirements for any patient with undifferentiated chest pain. It also includes a list associated symptoms and other potential diagnosis.



Electrocardiographs (ECGs)

- In the case of chest pain, an electrocardiograph (ECG) is the most important diagnostic tool available!
- ECG's are non-invasive, can be attended quickly and can determine whether a patient requires other potentially life saving procedures!
- Certain ECG features allow us to detect myocardial ischemia in the early, intermediate and late stages as hypoxic or nectrotic cells within the myocardium will have altered conductive properties.
- The timing, location and pattern of these abnormalities can allow us to determine the location and severity of an ischemic injury to the myocardium.
- The following pages detail the most common ECG changes associated with myocardial ischemia in the order they would occur in the typical progression of an acute myocardial infarction.
- For further information on ECG interpretation please refer to the basic rhythm interpretation, advanced rhythm interpretation or advanced life support learning packages.



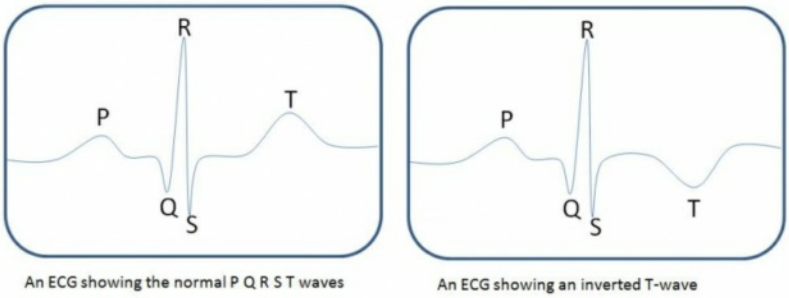
N.B. correct lead placement is essential for accurate interpretation of ECG data

T wave irregularities

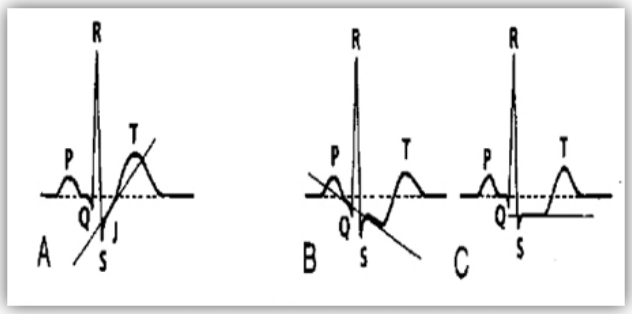
- T waves signify the period of ventricular repolarisation.
- T wave inversion is a normal variant in V1, and III and should always be present in aVR.
- *Ischemia* causes potassium ions to be lost from myocardial cells which causes abnormal or delayed ventricular repolarisation which translates to T wave inversion.
- New or dynamic T wave inversion is generally associated with acute ischemia or cardiac strain and should always be considered abnormal, whereas prolonged inversion can remain indefinitely following an infarction.
- T wave inversion related to myocardial ischemia most commonly occurs in contiguous leads (leads looking at the same area of the heart)
- T wave inversion can also be related to other pathologies i.e. A sudden rises in intercranial pressure or pulmonary embolism.

ST depression

- ST depression can be up-sloping, down-sloping or horizontal.
- Horizontal or down-sloping ST depression 0.5mm or greater at the J-point (the point where the QRS returns to the isoelectric line) in 2 or more contiguous leads is consistent with myocardial ischemia.
- Up-sloping ST depression is typically not specific for myocardial ischemia.
- Reciprocal ST depression refers to a depression of the ST segment in the leads opposite those effected in an ST elevation myocardial infarction.
- This is thought to be caused by both benign electrical mirroring and concurrent ischemia in remote areas as a result of occluded collaterals.
- Reciprocal depression occurs in 70% of inferior and 30% of anterior infarcts.



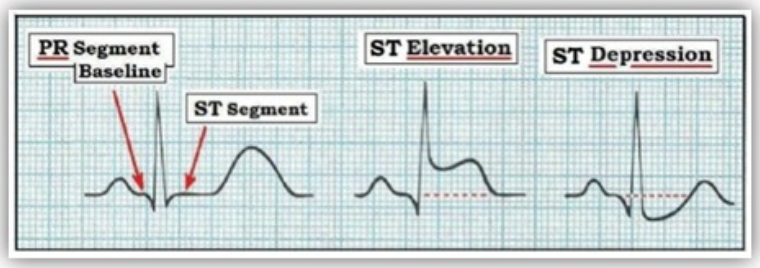
Normal complex vs T wave inversion

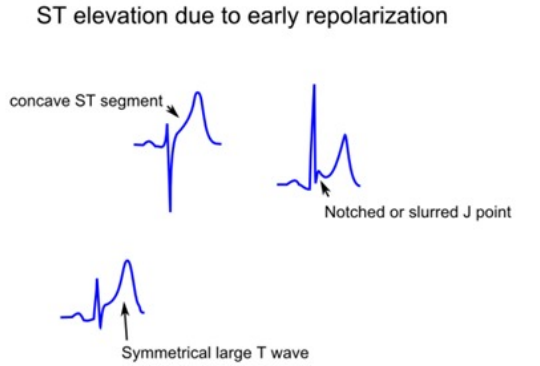


Up sloping A) vs down sloping B) and horizontal C) ST depression

ST elevation

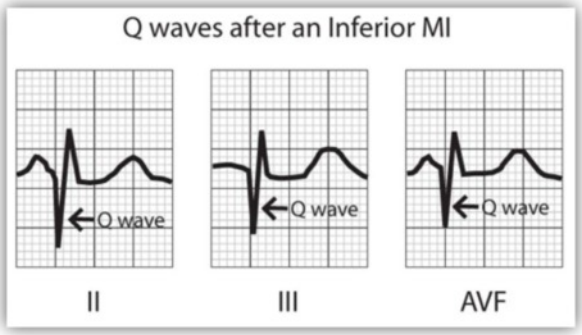
- ST elevation refers to an ST segment which remains elevated above the isoelectric line at the J point following the QRS complex.
- The ST segment signifies the end of ventricular systolic depolarisation. When myocardial cells are injured or inflamed they depolarise less effectively causing them to remain positively charged for longer. This residual positive charge causes the QRS to remain positive in relation to the neutral isoelectric line which graphically translates to ST elevation.
- ST elevation of ischemic origin is typically confined to an effected grouping of contiguous leads and is often accompanied by reciprocal ST depression.
- Widespread ST elevation can be seen in pericarditis, and other inflammatory conditions affecting the entire heart.
- ST elevation can easily be mistaken for benign early repolarisation or high take-off which are normal variants in younger people identified by slurred or notched J points, concave ST segments and prominent, symmetrical T waves.





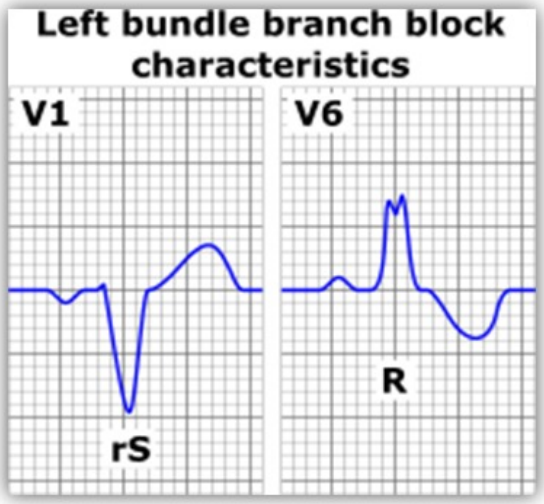
Pathological Q waves

- Pathological Q waves signify a degree of myocardial necrosis. These changes can take several hours to develop and once they appear they generally remain forever, as past a certain point cellular necrosis is irreversible.
- Q waves are considered pathological if they are more than 1mm wide, more than 2mm deep or more than ¼ the depth of the QRS complex.
- Small Q waves may be present in the leads viewing the left side of the heart, however, any right sided Q wave (V1-V3) should be viewed as pathological.



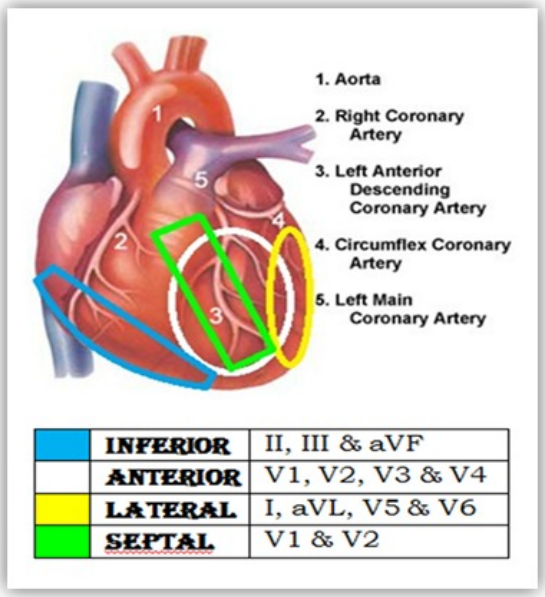
Left bundle branch block

- When the left bundle branch is blocked, electrical impulses travel down the right bundle branch stimulating contraction of the right ventricle before moving into the left.
- Although they can also appear independent of any pathology, LBBB are far more closely linked to the presence of serious cardiac conditions including cardiomyopathy and myocardial ischemia.
- As such, any patient presenting with a new LBBB should be thoroughly investigated for any potential pathological cause
- On a 12-lead ECG, the QRS complex of a patient with LBBB appears identical to the rhythm produced by an implantable pacemaker.
- Unlike RBBB, the delayed left ventricular contraction in LBBB can decrease cardiac output, particularly when associated with conditions such as heart failure.
- LBBB is defined as a QRS duration of 120milliseconds or longer with deep S wave ("W" shape) in V1 and an RSR complex ("M" shape) in V6



Identifying affected areas

- Different groupings of ECG leads correspond to different sections of the myocardium
- The picture on the right reflects the four main lead groupings and the four most commonly effected areas of the heart
- By identifying the effected area of the myocardium it is possible to identify which blood vessels may be involved



IV access and serology

- Intravenous access is important as it allows us to rapidly administer medication to treat the patient's symptoms and prevent further damage or deterioration.
- Blood testing is a vital part of assessing the patient with chest pain.
- Routine blood tests (FBC, EUC, LFT) allow us to identify and treat any issues or imbalances which may contribute to the patient's presentation.
- Venous blood gas testing allows us to rapidly assess basic blood counts, acid/base status, respiratory efficiency and electrolyte status and should be attended on all patients in respiratory distress or reporting palpitations.
- In the case of suspected pulmonary embolism, a blue top (sodium citrate) coags tube may be taken and held for possible D dimer testing which looks for cross linked fibrin degredation product (XDP) in circulating blood which may indicate the presence of a blood clot.
- The testing of serum cardiac enzymes is an important element of the assessment and risk stratification of myocardial infarction and acute coronary syndrome.

Troponin

- Troponin is a myofibrillar protein found in striated muscle cells including those in the myocardium.
- When these striated muscles are damaged troponin is released into the blood stream.
- There are many different tests for myocardial ischemia (CK MB, myoglobin, high sensitivity CRP and B-natiuretic peptide) why is troponin preferred?
  - It has a high level of specificity for measuring myocardial damage (95% specificity for myocardial ischemia after 8 hours)
  - Troponin is detectable in the blood stream 2-4 hours after myocardial injury which is significantly faster than other tests
  - Troponin levels remain elevated for longer than other tests following injury (remains detectable in the blood for up to 14 days)
- Classically two different forms; troponin I (TNI) & Troponin T (TNT)
- TNI favoured in diagnosis of MI as TNT can be chronically elevated in kidney disease
- **Normal TNI range < 0.01 nanograms/ml, infarct classified as TNI > 0.04 nanograms/ml**
- High sensitivity troponin (HST) is a test which is significantly more sensitive for the presence of serum troponin at the expense of cardiac specificity provided by TNI.
- High sensitivity troponin can be measured at baseline then repeated at 3 hours post pain for a comparative measurement or alternatively at 6 hours post pain for a maximal level measurement.
- HST is considered positive if greater than 26ng/L in men or 16ng/L in women or a greater than 50% increase from baseline measurement.
- Because of its high sensitivity/ low specificity attribute, negative readings are more definitive but positive reading should be closely investigated for alternative causes of troponin leaks.
- Implication for the patient? In the patient with chest pain, Troponin provides an important risk stratification tool in ACS.

Chest x-rays

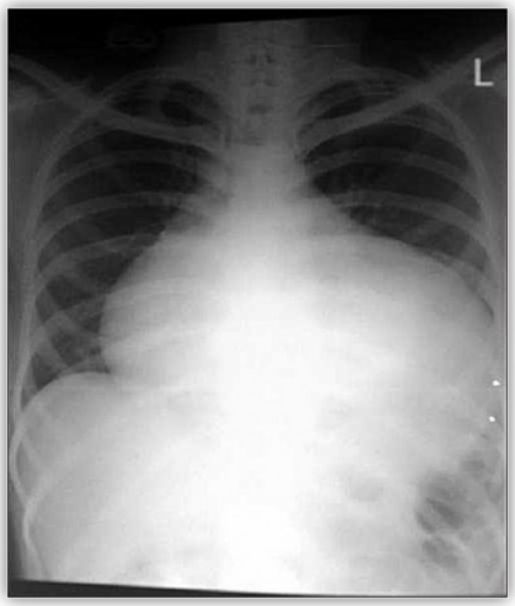
- Chest x-rays are performed routinely as a part of the initial assessment of chest pain.
- X-rays allow us to visualise any physical abnormalities within the patient's chest that could account for their pain.
- X-rays are essential in the process of disproving other serious or life threatening conditions.
- Chest x-ray should also be attended in all chest pain patients to assess for possible complications of AMI such as pulmonary odema.
- X-rays are integral in the diagnosis of: cardiac tamponade, pneumothorax, tension pneumothorax, pneumonia, aortic aneurysm, pleural effusion, cardiomegaly, rib fractures & lung masses.
- The following examples compare a normal, healthy chest to x-rays of several potentially life threatening conditions which may exhibit similar presentations to cardiac chest pain.

Normal chest

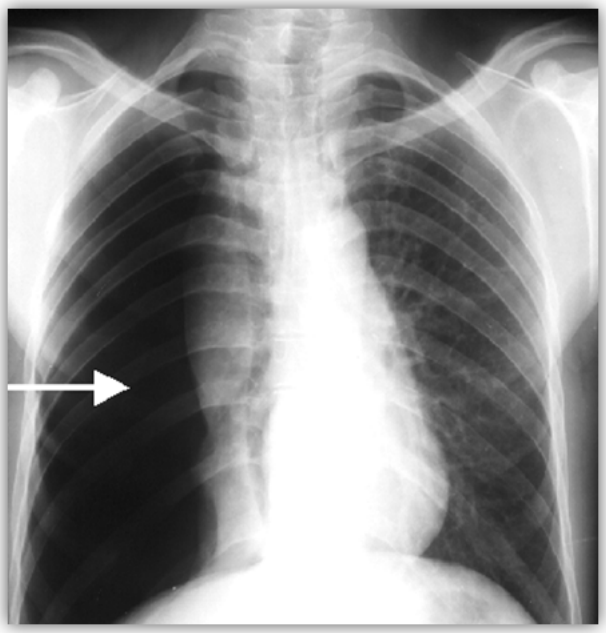
- Normal sized and shaped heart
- 2 completely inflated lungs
- Smooth, symmetrical diaphragm
- Central trachea
- Non-widened mediastinum
- Clear lung fields
- Intact ribs

Cardiomegaly

- The heart is grossly enlarged
- Defined as a heart which occupies more than 50% of the internal thoracic diameter
- Often associated with heart failure
- Commonly indicates cardiomyopathy, a condition in which the heart is chronically enlarged through hypertrophy or dilation.
- Also noted in pericardial effusion and tamponade!



Pneumothorax

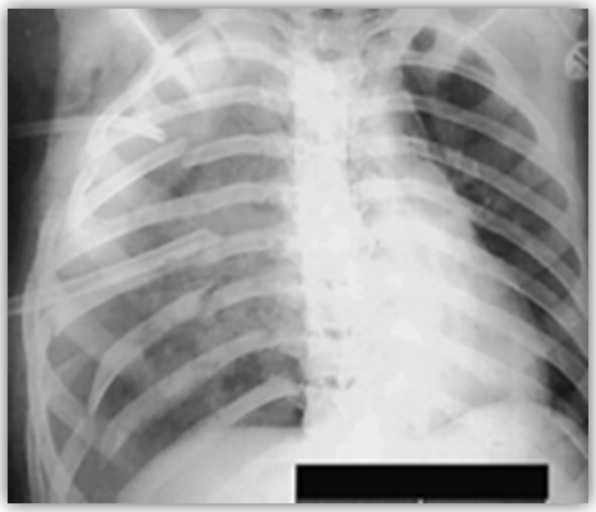


- One lung can be seen as inflated whilst the other is partially or totally collapsed
- Can be difficult to interpret
- Can be seen by following lung borders
- It may be helpful to compare the texture or opacity of the 2 lungs

Tension pneumothorax

- A pneumothorax which allows air to enter but not escape the thoracic cavity causing a build up of pressure which eventually compresses the heart and opposing lung
- Deviation of the trachea and mediastinum away from the tension
- Depression of the diaphragm on the side of the tension
- Tension pneumothorax is a life threatening medical emergency and one of the 8 reversible causes of cardiac arrest!
- Needle decompression or chest tube insertion required immediately

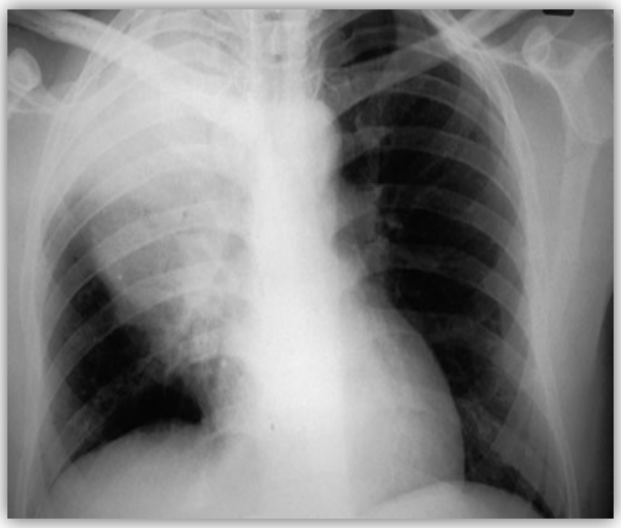
Rib fractures



- Can be identified by following the edges of each ribs to identify any breaks in the smooth edge of the bones.
- If 2 or more adjacent ribs are broken in 2 or more places it is referred to as a flail segment.
- Flail chest is often associated with pulmonary contusion, pain and mechanical dysfunction that can lead to respiratory failure.

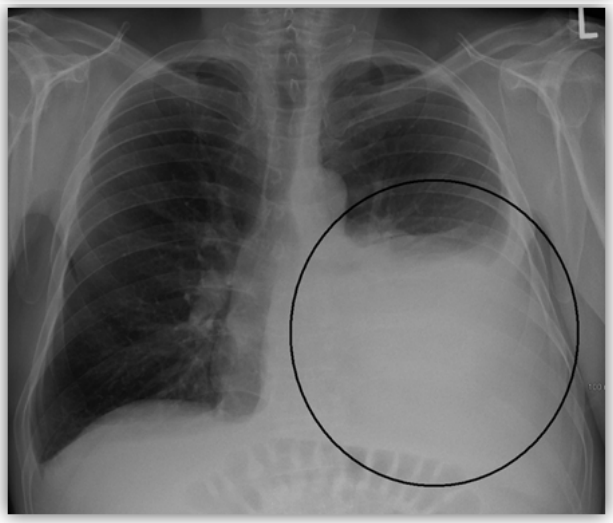
Pneumonia

- An acute infection of the lower respiratory tract
- Represented as an area of dense opacity
- Can be confined to individual lobes or diffuse throughout the lungs



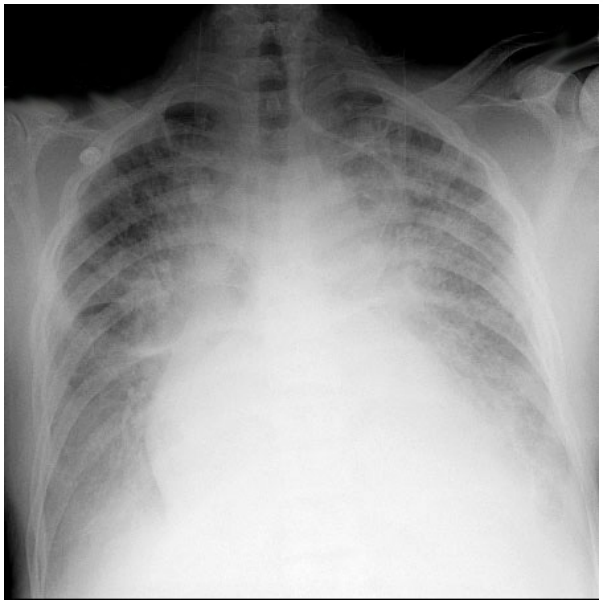
Pleural effusion

- The accumulation of fluid between the layers of the pleura which surround the lungs
- Often occuring in trauma, a collection of blood in the pleural space is referred to as a hemothorax.
- Seen as a solid opaque area between the diaphragm and the base of the lung



Pulmonary oedema

- The accumulation of fluid in the hollow spaces and parenchyma of the lungs.
- Caused by increased intravascular pressure in pulmonary circulation, often due to right heart failure.
- Seen as patchy opacity throughout both lungs representing fluid collection.



So it can be seen that through physical assessment, electrocardiography, serology and radiographic imaging of the chest a wide array of

Initial treatment

- While a patient is suffering chest pain it is important to continually monitor their vital signs for any sign of instability.
- Continuous cardiac monitoring should be applied to allow for rapid rhythm assessment should the patient become unstable.
- In the case of severe unrelieved chest pain with associated respiratory or circulatory distress, it may be desirable to attach an external defibrillator as this would allow:
  - Continuous cardiac monitoring
  - The ability to rapidly defibrillate or cardiovert if required
- The goals of initial treatment for patients with chest pain focus predominantly on symptom management and facilitating a safe path towards definitive treatment.
- Throughout NSW health, the assessment and management of chest pain is guided by the NSW Health chest pain pathway (attached to the end of this document.

Oxygen therapy

- For a long time supplemental oxygen therapy has been applied to patients presenting with chest pain of presumed ischemic origin, regardless of their oxygen saturation.
- The rationale behind this was that the patient was experiencing pain due to cellular hypoxia, and if you were able to hyper-oxygenate the patient's blood then the little amount of blood travelling through an occluded vessel or collateral would better perfuse the compromised section of the myocardium.
- While the application of supplemental oxygen has been a best practice recommendation for years, the evidence supporting its use has been inconsistent.
- Recent evidence has even begun to find that the risks associated with hyper-oxygenation of an ACS patient with normal saturations may outweigh any potential benefit.

Current oxygen evidence

- A recent systematic review by the Cochrane collaboration examined the results of 3 randomised controlled trials comparing the administration of oxygen and air to patients with chest pain. These trials involved 387 patients. Of the 387, 14 patients died. Of the 14 patients that died, nearly 3 quarters received oxygen therapy (Cabello et al, 2012).
- The authors of the different articles reviewed by Cochrane hypothesised that poor outcomes associated with supplemental oxygen may be attributed to:
  - The paradoxical effect of oxygen reducing coronary artery flow, increasing coronary vascular resistance, thus reducing stroke volume and cardiac output.
  - Increased vascular resistance from hyperoxemia.
  - Re-perfusion injury from increased oxygen free radicals.
- It was stressed that because the number of patients who died was so low in these studies that the results may be due to external factors or simple chance. For this reason the publishers suggested that further research into the area is required immediately.

Current recommendations

- In 2010, the national institute for health and clinical excellence issued the NICE clinical guideline 95 addressing the assessment and treatment of chest pain (National Institute of Health and Clinical Excellence, 2010).
- These guidelines recommended that oxygen therapy not be routinely administered to patients with chest pain.
- Instead, these guidelines recommend that oxygen should be administered to people with chest pain only if they are hypoxic.
- The Australian Resuscitation Council also acknowledge that there is limited evidence supporting the routine use of oxygen for patients with chest pain (Australian Resuscitation Council, 2011).
- In the NSW Health chest pain pathway, oxygen remains one of the recommended initial treatments.
- However, when considering emerging evidence, it would be more appropriate to assess the patient for indications that oxygen therapy would be of benefit (low o2 sats, cyanosis, breathlessness) rather than simply applying it habitually.
- The ARC's current recommendation is that oxygen be used to manage symptoms such as breathlessness or hypoxia, rather than being used as routine treatment for ACS (Australian Resuscitation Council, 2011).

1	In myocardial infarction and ACS, aim at an oxygen saturation of 94 to 98% or 88 to 92% if the patient is at risk of hypercapnic respiratory failure.
2	Patients with serious emergency conditions such as myocardial infarction and ACS should be monitored closely but oxygen therapy is not required unless the patient is hypoxaemic:
	• If hypoxaemic, the initial oxygen therapy is nasal cannulae at 2 to 6 l/min or simple face mask at 5 to 10 l/min unless oxygen saturation is < 85% (use reservoir mask) or if at risk from hypercapnia
	• The recommended initial target saturation range, unless stated otherwise, is 94% to 98%
	• If oximetry is not available, give oxygen as above until oximetry or blood gas results are available
	• If patients have COPD or other risk factors for hypercapnic respiratory failure, aim at a saturation of 88% to 92% pending blood gas results but adjust to 94% to 98% if the PaCO2 is normal (unless there is a history of respiratory failure requiring NIV or IPPV) and recheck blood gases after 30 to 60 minutes.

Anti-thrombotic drugs

- Aspirin or acetylsalicylic acid is a salicylate drug which is commonly used as an anti-inflammatory and anti-pyretic agent.
- Aspirin is also a widely utilised anti-platelet drug which inhibits the production of thromboxane, the substance which binds platelets together.
- Aspirin is given to patients suffering from potentially ischemic chest pain in an attempt to limit the growth of any existing thrombus and to reduce the risk of new thrombus causing re-infarction.
- Until recently it was widely understood that adults taking regular low dose aspirin would in turn lower their risk of heart attack or stroke. However, more heed is now being payed to the gastrointestinal complications caused by regular aspirin use and it is now being recommended only for people who are at risk of thrombotic disorders and when prescribed it is frequently being coupled a proton pump inhibitor to protect the gut (Hennekens, 2008)
- In addition to aspirin, other antithrombotic drugs such as clopidogrel, prasugrel, heparin and clexane may be administered in consultation with the consulting cardiology team.
- A single dose of 300mg of aspirin may be given orally by senior accredited emergency nurses to patients presenting with cardiac sounding chest pain as detailed in division of emergency medicines standing order for aspirin.
- Before administration patients should be asked about hypersensitivty to aspirin or NSAIDS as these drugs have been associated with complications of severe asthma and GI bleeding.
- Where possible patients should be encouraged to chew aspirin tablets before swallowing with water as the additional sublingual absorbtion has been demonstrated to hasten the antiplatelet effect of the drug compared to disolving it in water or swalloing whole tablets (Feldman & Cryer, 1999).

Pain relief

When dealing with chest pain, effective analgesia is particularly important as the presence of pain can indicate that the myocardium is being damaged!

Glyceryl Trinitrate

- Glyceryl trinitrate or nitro-glycerine is widely used in the treatment of a number of conditions including heart failure and chest pain due to myocardial ischemia.
- When GTN is absorbed by the body it is broken down into a metabolite called nitric oxide which acts as a profound vasodilator.
- By dilating the major coronary blood vessels, GTN effectively reduces pre-load and after-load allowing the heart to pump with less effort, thus requiring less oxygen.
- In the case of an occluded coronary artery it can also help to expand the internal diameter of the blood vessel which in turn allows more blood to reach the hypoxic section of the myocardium.
- Because of these 2 physiological effects, GTN is typically the first drug used to treat ischemic chest pain!
- GTN comes in a number of different preparations for administered via a number of different routes including: sublingual tablets, sublingual spray, transdermal patches and intravenous injections.
- Nitro-glycerine is also the active ingredient in dynamite!

Using GTN

- In the case of acute chest pain, 300 to 600mcg of sublingual GTN (half to one whole tablet) may be given every 3-4 minutes up to a maximum of 1200-1800mcg depending on the patient's tolerance and as directed by the treating physician.
- If chest pain persists after 2-3 tablets of sublingual GTN, narcotic analgesia should be considered.
- For persistent cardiac chest pain GTN may be considered for continuous administration via intravenous infusion.
- Due to its vasodilatory effects, GTN should be administered while patients are sitting or lying to avoid dizziness or fainting due to orthostatic hypotension.
- Patients may experience a headache after using GTN. In fact, one study found that patients with normal coronary arteries were 50% more likely to experience a headache from GTN than those with coronary artery disease (His et. al, 2004).

Narcotics

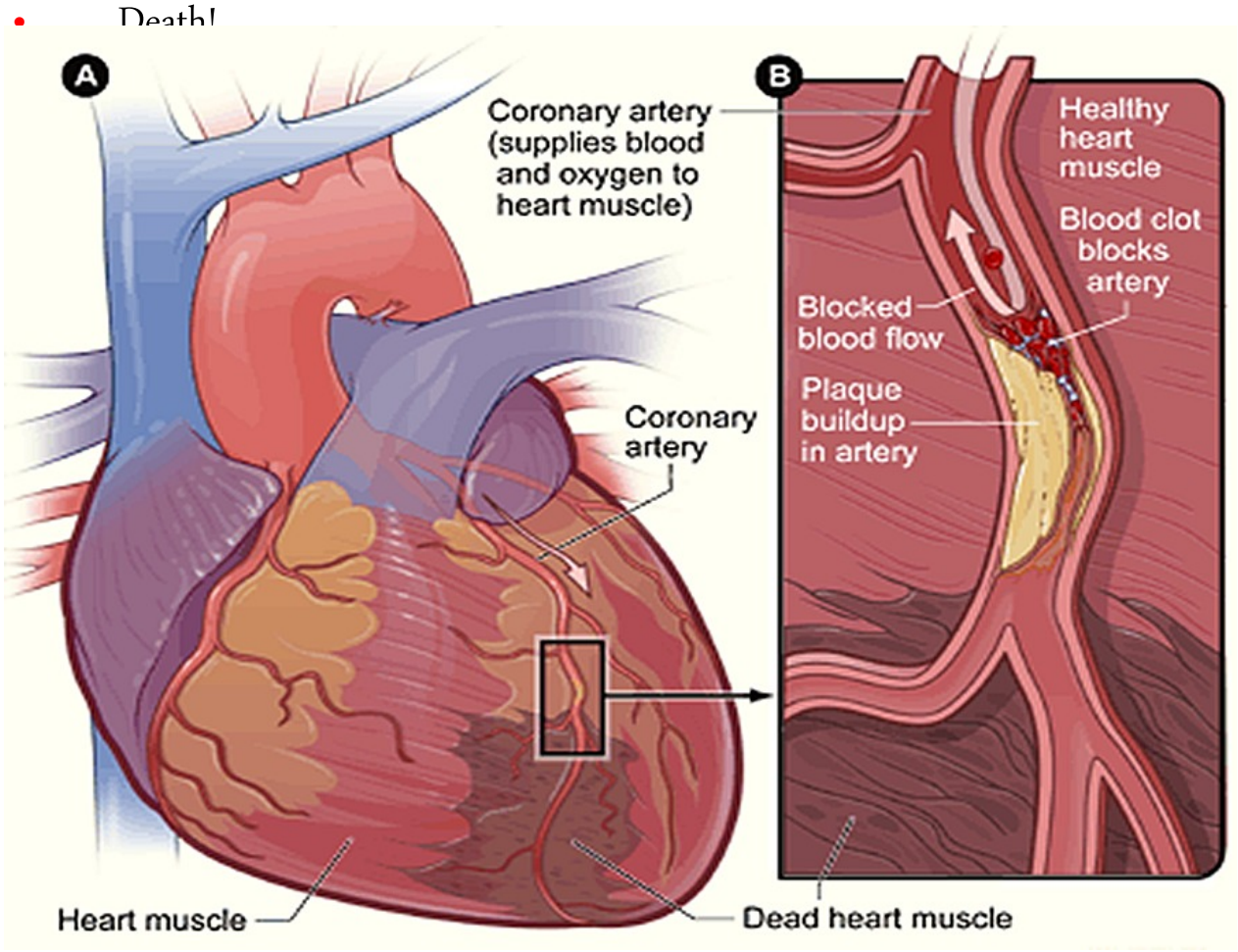
- If GTN is unsuccessful in completely removing a patient's chest pain, opioid medications are the next alternative.
- Morphine is a commonly used narcotic analgesia which works by blocking the receptors responsible for relaying pain messages within the central nervous system.
- Morphine is also a vasodilator which helps open coronary blood vessels allowing better blood flow while reducing pre and after-load.
- Although less evidence specifically addresses it's use, fentanyl can be used as a suitable alternative to morphine in the event of an allergy, however the additional vasodilatory effect is lessened.
- While narcotics remain the most effective method of managing ischemic chest pain which is unresponsive to nitrates, it is worth noting that a 2005 study by Meine et. al. found that the administration of morphine was associated with a higher risk of mortality in the NSTEMI patient.

Reassessment

- Because so much of the treatment provided to patients with chest pain focuses on the management of symptoms, it is vital that we continually reassess these patients to determine the effectiveness of our interventions.
- It is also important to continually assess these patients as they are often at a high risk of rapid deterioration.

What is a myocardial infarction?

- Myocardial infarction (MI) = heart attack!
- Refers to cellular necrosis of the myocardium resulting from restricted blood flow causing tissue hypoxia.
- Caused by:
  - / local plaque rupture stimulating platelet aggregation and forming a thrombus (blood clot), or;
  - / Debris released from an accessory plaque rupture forming an embolus (traveling blood clot) which travels and occludes a major vessel.
- Acute MIs are life threatening medical emergencies!
- Cellular necrosis inhibits the ability of the myocardium to conduct electrical impulses or physically contract which can in turn cause:
  - Lethal arrhythmias
  - Insufficient cardiac output
  - Insufficient organ perfusion
  - Death!



Classification of AMIs

- If a patient presents with clinical features of MI and has either contiguous ST-elevation or a new LBBB on their ECG they are categorised as having an ST-elevation MI (STEMI)
  - STEMI is diagnostically defined as ST elevation >1mm in 2 or more contiguous limb leads or ST elevation >2mm in 2 or more contiguous chest leads or presumed new LBBB (NSW Health chest pain pathway, 2011)
- If a patient fits the diagnostic criteria for myocardial infarction but does not have either of these features on their ECG, they are treated as a non ST-elevation MI (NSTEMI) or an acute coronary syndrome (ACS).
- All STEMI patients require immediate definitive treatment whilst ACS patients receive treatment according to their degree of risk as assessed using the ACS risk stratification matrix below.

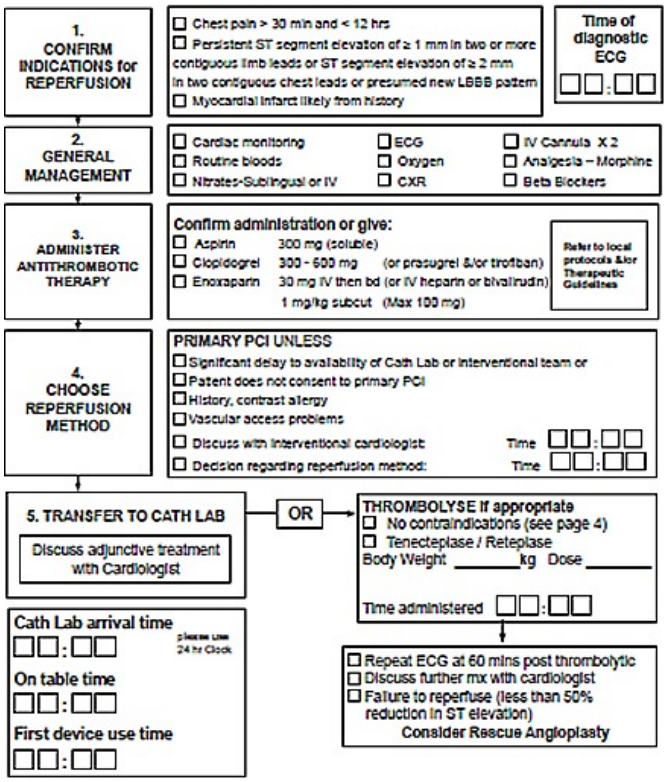
HIGH RISK Any of the following	**INTERMEDIATE RISK** Any of the following and no high risk features	**LOW RISK** Any of the following and no high or intermediate risk features
☐ ACS symptoms are repetitive or prolonged (> 10 min) & still present. ☐ Syncope ☐ History of chronic left ventricular systolic dysfunction (especially if known LVEF < 40%) OR current clinical evidence of LVF. ☐ Previous PCI/CABG < 6 months ☐ Diabetes + typical ACS symptoms ☐ Chronic renal failure + typical ACS symptoms ☐ Haemodynamic compromise (sustained SBP < 90 mmHg and / or new onset mitral regurgitation) ☐ Elevated Troponin (consider haemolysis, renal failure)	☐ ACS symptoms within 48 hrs that occurred at rest, or were repetitive or prolonged (but currently resolved) ☐ Previous PCI/CABG > 6 months ☐ Known coronary heart disease- Esp if prior AMI or known coronary lesion > 50% stenosis ☐ Two or more risk factors of: Hypertension, family history, active smoking or hyperlipidaemia ☐ Chronic renal failure (especially if known GFR < 60 mL/min) + atypical ACS symptoms ☐ Diabetes + atypical ACS symptoms ☐ Age > 65 years	☐ Presentation with clinical features consistent with ACS without intermediate- risk or high-risk features.
☐ Persistent or dynamic ECG changes of • ST depression ≥ 0.5 mm or • new T wave inversion ≥ 2 mm ☐ Transient ST elevation (≥ 0.5 mm) in more than two contiguous leads ☐ Sustained VT	☐ ECG is not normal and has changed from previous pain free ECG but does not contain high risk changes.	☐ ECG Normal or unchanged from previous pain free ECG
All cases to be discussed with Senior Medical Officer		

Definitive treatment at a primary PCI site

- A PCI facility is any hospital or health care facility with the equipment and capacity to perform percutaneous coronary interventions or coronary angioplasty.
- PCI facilities all have a cardiac catheterisation laboratory or similar area in which PCI can be performed.
- They are also required to have access to on-call staff to perform the procedure 24 hours a day.
- These facilities must have the capability to perform emergency cardiothoracic surgery in the event that PCI fails and emergency coronary artery bypass surgery is required, however several recent articles exist demonstrating acceptable results from PCI facilities without on site cardiothoracic surgical support. (Alversano et. al., 2002)
- PCI facilities allow patients experiencing myocardial infarction to receive gold standard revascularising treatment in a timely manner.

PCI site STEMI management

- As is always the case with chest pain, patients presenting to PCI site should have vital signs and an ECG reviewed by a senior doctor within 10 minutes.
- Once a diagnosis of STEMI has been established the NSW health chest pain pathway should be used to determine whether reperfusion therapy is indicated.
- While the patient is prepared for reperfusion, general observation and management should continue i.e. vital sign and ECG monitoring, pain relief & oxygen therapy if required.
- If not already administered, the patient should receive antithrombotic therapy as directed by the cardiology team.
- If PCI is available and not contraindicated it should be attended in the cardiac catheterisation laboratory without delay.
- If for any reason PCI is not available or appropriate, thrombolysis should be considered for administration as soon as possible.
- Following reperfusion therapy the patient should be closely monitored under the care of the cardiology team in a suitable high acuity patient care area i.e. CCU, ICU, HDU



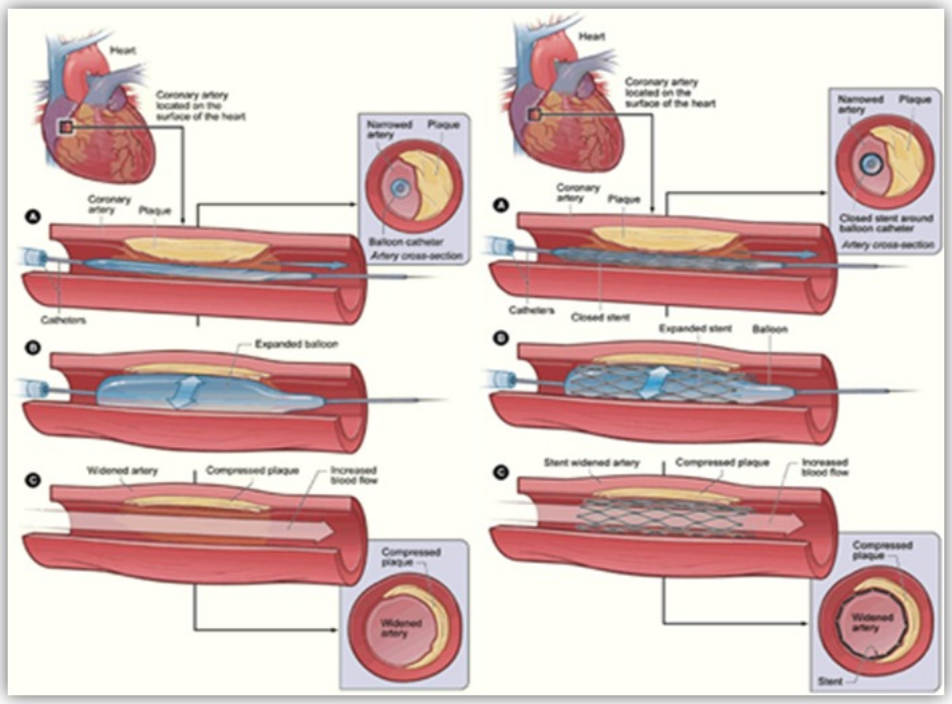
What is PCI?

- Percutaneous coronary intervention: also know as angioplasty, is an invasive revascularising treatment for myocardial infarction and coronary artery stenosis.
- PCI allows the physician to accurately and definitively visualise any occlusions of the coronary vessels before attempting to restore myocardial blood flow.
- PCI is regarded as the gold standard treatment for STEMI, providing better outcomes than thrombolysis whilst limiting the risk of complications.
- Compared with thrombolytic therapy, the benefits of primary PCI include a reduction in the risk of stroke, a reduction in the frequency of reinfarction and an increase in the frequency of infarct-related artery patency, resulting in improved in-hospital and long-term survival (Weaver et. al., 1997).
- PCI has also been demonstrated to be a time dependant intervention with better results being associated the early initiation of PCI following first medical contact (Moscucci & Eagle, 2006).
- It should also be noted that significantly worse outcomes are noted when PCI is attended 90 minutes or more from first medical contact (Moscucci & Eagle, 2006).

The actual size of an angioplasty catheter with balloon inflated

How is it done?

- Local anaesthetic and light sedation administered as required.
- An introducer needle is inserted into a peripheral artery: this is typically the femoral artery, however a growing evidence base supports the safety of PCI attended from the radial artery.
- A radiopaque guide wire is gently advanced through the introducer needle and through the arterial system to the aortic arch.
- The needle is then replaced with a flexible plastic sheath in order to reduce the risk of injury to the artery.
- A guiding catheter then inserted over the guide wire and the guide wire is removed.
- The catheter is advanced to the entrance to the coronary artery and radiopaque contrast is injected into the vessels. Angiogram pictures taken using an x-ray machine.
- Once a blockage is identified a flexible guide wire is advanced through the narrowed artery.
- The angioplasty catheter is then advanced over the top of the guide wire.
- Once in place, the balloon tip of this angioplasty catheter is expanded, mechanically forcing the artery open and crushing the atherosclerotic plaque outwards.
- If a stent is used, it is expanded as the balloon inflates and is left behind to strengthen the vessel when the balloon is deflated.

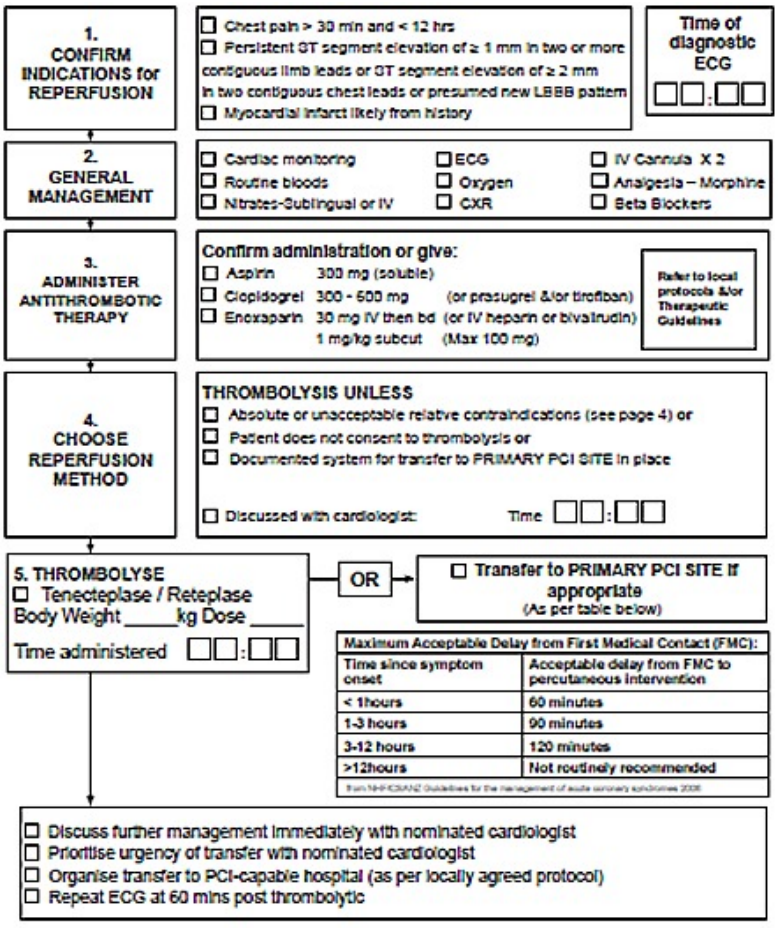


Definitive treatment at a non PCI site

- Any facility without the ability to perform primary PCI is referred to as a non-PCI site.
- As PCI remains the gold standard for the treatment of STEMI, many non PCI sites have pre established rapid transfer procedures in place to enable STEMI patients at hospitals nearby a primary PCI site to receive primary angioplasty.
- For facilities without PCI capability or an established rapid transfer procedure, chemical thrombolysis is the first choice for definitive treatment of STEMI

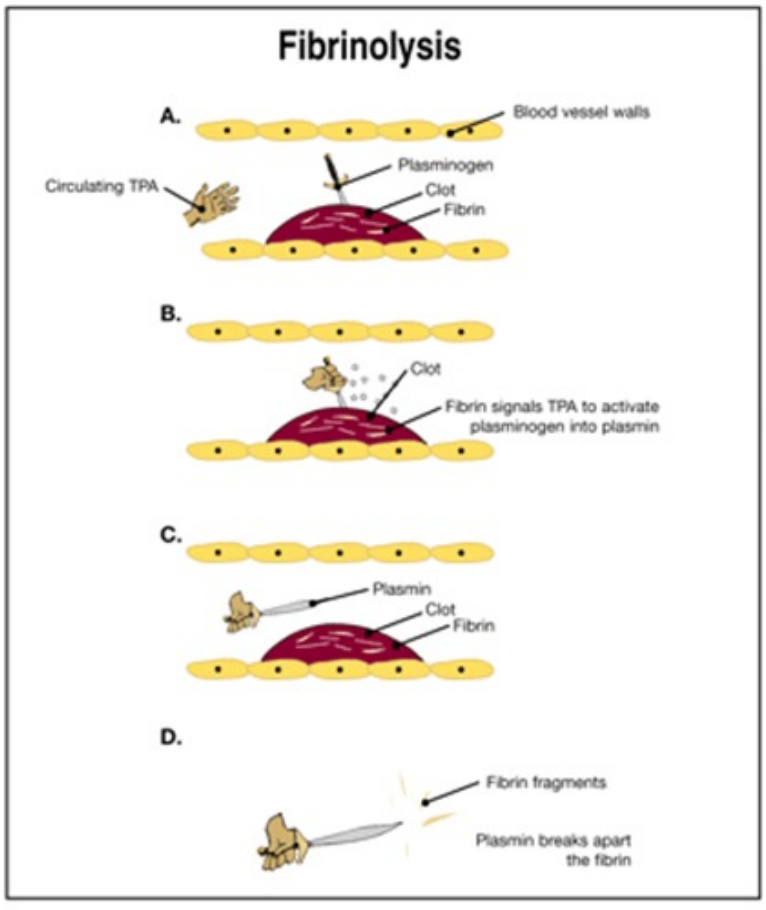
Non PCI site STEMI management

- As is always the case with chest pain, patients presenting to PCI site should have vital signs and an ECG reviewed by a senior doctor within 10 minutes.
- Once a diagnosis of STEMI has been established the NSW health chest pain pathway should be used to determine whether reperfusion therapy is indicated.
- While the patient is prepared for reperfusion, general observation and management should continue i.e. vital sign and ECG monitoring, pain relief & oxygen therapy if indicated.
- If not already administered, the patient should receive antithrombotic therapy as directed by the cardiology team.
- If a STEMI patient is seen at a non-PCI site, there may be an option to utilise an established rapid transfer system to move the patient to a primary PCI site for angiography. If the patient is stable and meets the timing criteria the accepting cardiologist should be contacted immediately regarding transfer.
- If no transfer system is in place or the patient does not fit the criteria, thrombolysis should be considered as the most appropriate option for reperfusion.
- Following reperfusion therapy the patient should be closely monitored under the care of the cardiology team in a suitable high acuity patient care area i.e. CCU, ICU, HDU



What is thrombolysis?

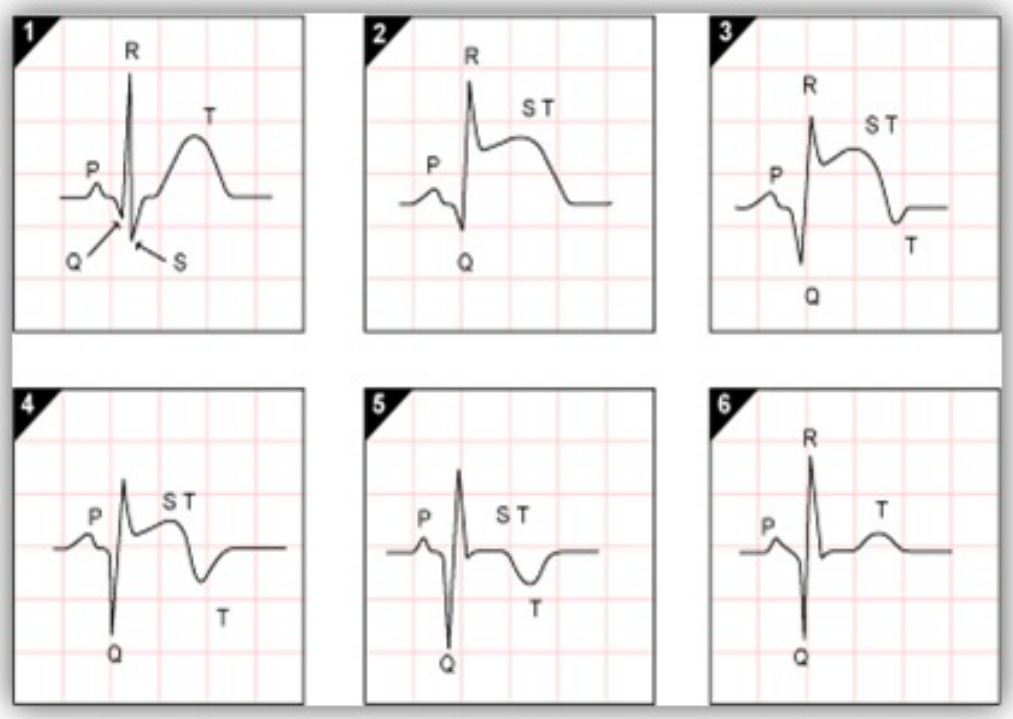
- Thrombolysis refers to the breakdown or destruction of blood clots or thrombus through the use of medication.
- This is achieved by the administration of tissue plasminogen activator (tPA) which helps to convert plasminogen to plasmin which actively accelerates **fibrinolysis** (the destruction of the structural bonds which exist within blood clots).
- Originally, thrombolytic agents were derived from the streptococcus bacteria (streptokinase)
- Thrombolytic drugs have even been made from proteins isolated from human urine (urokinase) (Stump. et. al, 1986).
- Most recently, thrombolytic agents have been produced using recombinant biochemistry which combines naturally occurring human tPA with DNA of another source in order to alter its properties.
- All thrombolytic agents have been associated with numerous major complications with an especially high incidence of bleeding.
- Of the 3 major preparations of recombinant tPA (alteplase, reteplase & tenecteplase), tenecteplase is favoured in STEMI for a number of reasons...



Tenectaplase

- Tenecteplase is distributed under the trade name Metalyse.
- It is a recombinant tPA which modifies natural human tPA using a cell line found in Chinese hamster ovaries (Metalyse.com, 2012).
- The human tPA in tenecteplase is modified by the addition of asparagine, glutamine and 4 alanines which are aimed at decreasing fibrin binding and increasing fibrin specificity (Metalyse.com, 2012).
- Tenecteplase has been associated with fewer major haemorrhagic complications than other forms of rtPA (Militello, 2001).
- Tenecteplase is able to be administered over 5-10 seconds whereas reteplase takes 2 minutes and alteplase takes 90 minutes (McAuley, 2012).
- Tenecteplase has a half-life of 20-24 minute meaning that it can be administered as a single bolus dose whereas reteplase may require a second administration 30 minutes post the first (McAuley, 2012).
- Multiple studies have demonstrated that tenecteplase is associated with outcomes that are consistently as good or better than other forms of tPA with regards to reducing mortality (Militello, 2001).
- Tenecteplase is expensive! Single doses costing anywhere between $1700-2300 each!

ECG progression in STEMI after reperfusion therapy



1. Normal sinus complex. The ST segment is on the isoelectric line. At the onset of pain the ECG would be normal but the ST segment would soon start to change. In this picture, the T wave has grown taller.
2. Within an hour the ST segment would be noticeably elevated, indicating the onset on myocardial necrosis. (tissue death). This is the point at which we would be aiming to administer the thrombolytic (clot-busting) drug or PCI.
3. After around an hour (variable to the patient) of ischemia, cells begin to permanently die which translates to Q waves
4. If reperfusion therapy is administered, we would be looking for specific changes on the ECG. A 50% reduction in ST segment elevation is a good indicator of success. In this picture, the ST elevation has reduced by more than 50% from picture 2. We would expect to see these changes within 90 minutes of administering thrombolysis. You can also see the T wave inversion is much deeper. This is a good sign of reperfusion. (blood flow returning to the damaged area.)
5. 24 hours later, the ST segment may have returned to the isoelectric line. In this picture you can see the ST segment is back on the isoelectric line but the T wave remains inverted. It may stay inverted for days, weeks or months.
6. In some cases, after a few months the ECG looks relatively normal. Compare picture 6 with picture 1. They look much the same but for the deep Q wave in picture 6. A deep Q wave is an indicator of myocardial tissue death and will remain on the ECG permanantly. A "pathological" Q wave is not "time-specific". It may be there from a previous heart attack and therefore is not part of the criteria for evaluating an **Acute** Myocardial Infarction.

Questions

1. Which of the following is the leading worldwide cause of death?

- Heart disease
- Stroke
- Asthma
- The Ebola virus

2. Name the three vital structures contained within the chest?

3. Describe the process of pain assessment in the patient presenting with chest pain?

4. Which of the following is the most important diagnostic tool in the assessment of chest pain?

- MRI
- ECG
- X-ray
- Urinalysis

5. Which of the following ECG changes indicates the presence of potentially irreversible myocardial necrosis?

- T wave inversion
- ST elevation
- Right budnle branch block
- Pathological Q waves

6. Left bundle branch blocks are characterised by the presence of a deep, wide S wave ("W" pattern) in V1 and an RSR complex ("M" pattern) in V6.

- True
- Flase

7. Leads II, III and aVF on an ECG relate to which of the following areas of the myocardium?

- Inferior
- Septal
- Anterior
- Lateral.

8. Which of the following coronary arteries supplies the inferior region of the myocardium?

- Left anterior descending artery
- Right coronary artery
- Left circumflex artery

9. What blood test is used for risk stratification and classification of acute coronary syndromes?

10. Name 2 diagnosis that can be identified using chest x-ray?

11. Above what range is a high sensitivity troponin reading considered positive in men?

12. Considering the current evidence and the recommendations of the Australian Resuscitation Council, when should oxygen therapy be applied to patients presenting with chest pain?

13. What dose of the anti-thrombotic drug aspirin should be given to patients experiencing chest pain?

- 100mg
- 300mg
- 900mg
- 2g

14. Describe how glyceryl trinitrate (GTN) acts to reduce ischemic chest pain?

15. If GTN is unsuccessful in relieving chest pain, which of the following drugs should be considered as second line therapy?

- Paracetamol
- Morphine
- NSAIDS
- Noradrenaline

16. What are the 2 pathological mechanisms which can cause myocardial infarction?

17. STEMI is diagnostically defined as ST elevation >1mm in 2 or more contiguous limb leads or ST elevation >2mm in 2 or more contiguous chest leads or presumed new LBBB.

- True
- False

18. PCI or primary angioplasty is considered the gold standard of treatment for STEMI.

- True
- False

19. Name one major complication associated with thrombolysis?

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Other resources

Internet Resources

- www.lifeinthefastlast.com/ecg-library/
- http://www.ecglibrary.com/ecghome.html
- http://ecg-interpretation.blogspot.com.au/
- http://geekymedics.com/
- http://en.ecgpedia.org/

Iphone/Ipad apps

- Anesoft “ECG Rhythms”
- CIPM “ECG in motion”
- Medical Joyworks “Prognosis: cardiology”

Policy Directive

Department of Health, NSW
73 Miller Street North Sydney NSW 2060
Locked Mail Bag 961 North Sydney NSW 2059
Telephone (02) 9391 9000 Fax (02) 9391 9101
http://www.health.nsw.gov.au/policies/

Chest Pain Evaluation (NSW Chest Pain Pathway)

Document Number	PD2011_037
Publication date	09-Jun-2011
Functional Sub group	Clinical/ Patient Services - Governance and Service Delivery Clinical/ Patient Services - Medical Treatment
Summary	The Policy outlines the minimum standards for the management of patients presenting with Chest Pain or other symptoms of myocardial ischaemia. NOTE: This Policy also applies to Local Health Networks until Local Health Districts commence on 1 July 2011.
Author Branch	Health Services Performance Improvement Branch
Branch contact	James Dunne 9391 9555
Applies to	Local Health Networks, Board Governed Statutory Health Corporations, Network Governed Statutory Health Corporations, NSW Ambulance Service, Public Hospitals
Audience	All staff involved in the management and risk stratification of patients who present with chest pain
Distributed to	Public Health System, Divisions of General Practice, Government Medical Officers, Health Associations Unions, NSW Ambulance Service, NSW Department of Health, Tertiary Education Institutes
Review date	09-Jun-2016
Policy Manual	Patient Matters
File No.	
Status	Active

Director-General

This Policy Directive may be varied, withdrawn or replaced at any time. Compliance with this directive is **mandatory** for NSW Health and is a condition of subsidy for public health organisations.

NSW GOVERNMENT Health

POLICY STATEMENT

IMPLEMENTATION OF MINIMUM STANDARDS FOR CHEST PAIN EVALUATION (NSW CHEST PAIN PATHWAY)

PURPOSE

The policy mandates the implementation of minimum standards for chest pain evaluation, by all hospitals in the NSW Health system for patients presenting to Emergency Departments with chest pain. Compliance with these minimum standards for chest pain evaluation will improve the management of patients by guiding clinicians through risk stratification and outlining the best practice management. Facilities may continue to use existing local Pathways provided that they meet all of the minimum standards and are in active use in emergency departments.

Facilities who do not use an existing Chest Pain Pathway that meets the minimum standards must implement the standard NSW Chest Pain Pathway. The NSW Chest Pain Pathway aligns with the National Heart Foundation/Cardiac Society of Australia and New Zealand Guidelines for the management of acute coronary syndromes.

MANDATORY REQUIREMENTS

1. All facilities with Emergency Departments must have and use a pathway that meets the following minimum standards for chest pain patients:
    - Assigns triage category 2
    - Includes risk stratification
    - ECGs are taken **and** reviewed
    - Troponin levels are taken **and** reviewed
    - Vital signs are taken **and** documented
    - Critical times are documented (symptom onset, presentation)
    - Aspirin is given, unless contraindicated
    - A Senior Medical Officer is assigned to provide advice and support on chest pain assessment and initial management, 24/7
    - A nominated Cardiologist is assigned to provide advice on further management 24/7
    - The pathway gives instruction regarding atypical chest pain presentations
    - High risk alternate diagnosis listed for consideration e.g. Aortic Dissection, Pulmonary Embolism & Pericarditis.
    - Sites that do not have 24/7 PCI capability must have Thrombolysis as the default STEMI management strategy unless there is an existing documented system for transfer.

2. All facilities who do not use an existing Chest Pain Pathway that meets the minimum standards must implement the standard NSW Chest Pain Pathway that matches their facility (i.e. only sites that can provide 24/7 Primary PCI are able to use the Primary PCI site Pathway) as the minimum standard.

IMPLEMENTATION

ROLES AND RESPONSIBILITIES

NSW Department of Health:

- Review the minimum standards of a Chest Pain Pathway in line with relevant national guidelines and best practice evidence.
- Develop and make accessible implementation support tools.
- Evaluate Chest Pain Pathway implementation and performance against the minimum standards across the NSW Health system.

LHN Chief Executives:

- Ensure effective implementation of the minimum standards for chest pain evaluation in all LHN Emergency Departments
- Report minimum standards for chest pain evaluation implementation to the LHN Governing Council
- Report Chest Pain Pathway implementation and performance against the minimum standards to NSW Department of Health as requested

LHN Directors of Clinical Governance:

- Direct a LHN gap analysis against the chest pain evaluation minimum standards
- Develop and lead implementation strategy
- Coordinate appropriate educational resources for clinicians
- Evaluate LHN Chest Pain Pathway implementation and performance against the minimum standards
- Investigate RCA incidents relating to the minimum standards for chest pain evaluation

Facility General Managers and Heads of Cardiology and Emergency Departments:

- Direct a local gap analysis against the chest pain evaluation minimum standards
- Implement the chest pain evaluation minimum standards locally
- Evaluate and monitor local implementation and performance against the chest pain evaluation minimum standards
- Coordinate local education requirements for clinicians
- Coordinate local rostering to ensure that a senior clinician is available to assist 24/7 as per the chest pain evaluation minimum standards or utilise documented referral network

Clinicians:

- Comply with the minimum standards of chest pain evaluation
- Escalate management of deteriorating patients as per Between the Flags (PD2010_026)
- In Emergency Departments that do not have a medical officer accessible 24/7, it will be necessary to implement processes where the nurse in charge of the ED signs the Chest Pain Pathway form in place of the medical officer.

REVISION HISTORY

Version	Approved by	Amendment notes
June 2011 (PD2011_037)	Dr Tim Smyth, Deputy Director-General, HSQPID	New Policy

ATTACHMENTS

1. NSW Chest Pain Pathway: Primary PCI Site
2. NSW Chest Pain Pathway: Non Primary PCI Site

NSW GOVERNMENT Health

Facility:

FAMILY NAME	MRN
GIVEN NAME	☐ MALE ☐ FEMALE
D.O.B. _____ / _____ / _____	M.O.
ADDRESS	
LOCATION / WARD	

COMPLETE ALL DETAILS OR AFFIX PATIENT LABEL HERE

CHEST PAIN PATHWAY

PRIMARY PCI SITE

Date of Presentation ☐☐/☐☐/☐☐☐☐ Time ☐☐:☐☐ Time of Symptom Onset: ☐☐:☐☐

CHEST PAIN or OTHER SYMPTOMS of MYOCARDIAL ISCHAEMIA

(eg sweating, sudden orthopnea, syncope, dyspnoea, epigastric discomfort, jaw pain, arm pain)

Be aware:
HIGH RISK ATYPICAL PRESENTATIONS
(eg diabetes, renal failure, female, elderly or Aboriginal)

TRIAGE CATEGORY 2

ECG & Vital Signs, expert interpretation within 10 minutes

General Management
- ☐ Oxygen
- ☐ Aspirin
- ☐ IV Access
- ☐ Pain Relief
- ☐ Pathology incl Troponin
- ☐ Chest X-ray

ST ELEVATION or (presumed new) LBBB

Y → **Consider Pericarditis** (sharp chest pain, respiratory or positional component)

N → **Consider Aortic Dissection** (back pain, hypertension, absent pulse, BP difference)
Consider Pulmonary Embolism (severe dyspnoea, respiratory distress, low subscript O_2 saturation)

N → **Diagnose NON ST ELEVATION ACUTE CORONARY SYNDROME (ACS)**

N → Go immediately to **STEMI MANAGEMENT** (page 3)

STRATIFY ACS RISK

HIGH RISK Any of the following	INTERMEDIATE RISK Any of the following and no high risk features	LOW RISK Any of the following and no high or intermediate risk features
≤ ACS symptoms are repetitive or prolonged (> 10 min) & still present. ≤ Syncope ≤ History of chronic left ventricular systolic dysfunction (especially if known LVEF < 40%) OR current clinical evidence of LVF. ≤ Previous PCI/CABG < 6 months ≤ Diabetes + typical ACS symptoms ≤ Chronic renal failure + typical ACS symptoms ≤ Haemodynamic compromise (sustained SBP < 90 mmHg and / or new onset mitral regurgitation) ≤ Elevated Troponin (consider haemolysis, renal failure)	≤ ACS symptoms within 48 hrs that occurred at rest, or were repetitive or prolonged (but currently resolved) ≤ Previous PCI/CABG > 6 months ≤ Known coronary heart disease- Esp if prior AMI or known coronary lesion > 50% stenosis ≤ Two or more risk factors of: Hypertension, family history, active smoking or hyperlipidaemia ≤ Chronic renal failure (especially if known GFR < 60 mL/min) + atypical ACS symptoms ≤ Diabetes + atypical ACS symptoms ≤ Age > 65 years	≤ Presentation with clinical features consistent with ACS without intermediate- risk or high-risk features.
≤ Persistent or dynamic ECG changes of ● ST depression ≥ 0.5 mm or ● new T wave inversion ≥ 2 mm ≤ Transient ST elevation (≥ 0.5 mm) in more than two contiguous leads ≤ Sustained VT	≤ ECG is not normal and has changed from previous pain free ECG but does not contain high risk changes.	≤ ECG Normal or unchanged from previous pain free ECG

All cases to be discussed with Senior Medical Officer

Recommended Management on page 2

This tool is intended as a guideline for clinicians to provide quality patient care. It is not intended, nor should it replace, individual clinical judgement. Some patients with co-morbidities or patients not suitable for invasive investigations may be appropriately managed medically.

NSW GOVERNMENT Health

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CHEST PAIN PATHWAY

PRIMARY PCI SITE

Contraindications and cautions for thrombolysis use in STEMI[1]

Absolute contraindications:

Risk of bleeding

- Active bleeding or bleeding diathesis (excluding menses)
- Significant closed head or facial trauma within 3 months
- Suspected aortic dissection (including new neurological symptoms)

Risk of intracranial haemorrhage

- Any prior intracranial haemorrhage
- Ischaemic stroke within 3 months
- Known structural cerebral vascular lesion (eg, arteriovenous malformation)
- Known malignant intracranial neoplasm (primary or metastatic)

Relative contraindications:

Risk of bleeding

- Current use of anticoagulants: the higher the international normalised ratio (INR), the higher the risk of bleeding
- Non-compressible vascular punctures
- Recent major surgery (< 3 weeks)
- Traumatic or prolonged (> 10 minutes) cardiopulmonary resuscitation
- Recent (within 4 weeks) internal bleeding (eg, gastrointestinal or urinary tract haemorrhage)
- Active peptic ulcer

Risk of intracranial haemorrhage

- History of chronic, severe, poorly controlled hypertension
- Severe uncontrolled hypertension on presentation (> 180 mmHg systolic or > 110 mmHg diastolic)
- Ischaemic stroke more than 3 months ago, dementia, or known intracranial abnormality not covered in contraindications

Other

- Pregnancy

[1] Adapted from NHF/CSANZ Guidelines for the management of acute coronary syndromes 2006

Contraindications to Exercise Testing (ACC/AHA Guidelines)[2]

Absolute

- Recurrent chest pain
- Acute myocardial infarction, within 2 days
- High-risk unstable angina
- Uncontrolled cardiac arrhythmias causing symptoms or haemodynamic compromise
- Symptomatic severe aortic stenosis
- Uncontrolled symptomatic heart failure
- Acute pulmonary embolus or pulmonary infarction
- Acute myocarditis or pericarditis
- Acute aortic dissection

Relative

- Critical left main coronary stenosis
- Moderate stenotic valvular heart disease
- Electrolyte abnormalities
- Systolic hypertension > 200 mmHg
- Diastolic hypertension > 100 mmHg
- Tachyarrhythmias or bradyarrhythmias
- New onset atrial fibrillation
- Hypertrophic cardiomyopathy and other forms of outflow obstruction
- Mental or physical impairment leading to the inability to exercise adequately
- High-degree atrioventricular block
- Resting ECG which will make EST interpretation difficult (eg LBBB, LVH with strain, Ventricular pacing, Ventricular preexcitation.)

[2] Gibbons etal, Circulation 106:1883,2002

Abbreviations:

ACS – Acute Coronary Syndrome
CABG – Coronary Artery Bypass Graft
ECG – Electrocardiogram
EST – Exercise Stress Test
FMC – First Medical Contact
GTN – Glyceryl trinitrate
LBBB – Left Bundle Branch Block
LVF – Left Ventricular Failure
LVH – Left Ventricular Hypertrophy
PCI – Percutaneous Coronary Intervention
SMO – Senior Medical officer
STEMI – ST Elevation Myocardial Infarction

BINDING MARGIN - NO WRITING

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CHEST PAIN PATHWAY

PRIMARY PCI SITE

Recommended Further Management Refer to drug protocols &/or Therapeutic Guidelines

HIGH RISK

ADMIT or TRANSFER

- ≤ Continuous cardiac monitoring & frequent vital signs
- ≤ Repeat ECG immediately if symptoms recurs
- ≤ Repeat ECG 8 hrs post onset of symptoms
- ≤ Repeat Troponin at 8 hrs if 1st sample negative *
- ≤ ECG/Troponin review by medical officer

Antiplatelet therapy

- ≤ Yes
- ≤ No

} Discuss with cardiologist /SMO

If no reason_____

_____

Betablocker

- ≤ Yes
- ≤ No

If no reason_____

_____

Anticoagulant

- ≤ Yes
- ≤ No

If no reason_____

_____

Symptomatic treatment of ongoing pain/hypertension

- ≤ IV GTN (titrate against pain & BP)
- ≤ IV Morphine
- ≤ Refer to nominated cardiologist for further management

INTERMEDIATE RISK

RESTRATIFY

- ≤ Continuous cardiac monitoring & frequent vital signs
- ≤ Repeat ECG immediately if symptoms recur
- ≤ Repeat ECG 8 hrs post onset of symptoms
- ≤ Repeat Troponin at 8 hrs if 1st sample negative *
- ≤ ECG/Troponin review by medical officer

Refer for Exercise Stress Test ** if :

- ≤ No further chest pain/symptoms **and**
- ≤ 2 negative Troponin tests **and**
- ≤ No new ECG changes and
- ≤ No contraindications to stress test

(page 4)

Restratify to High Risk if:

- ≤ Recurrent ischaemic chest pain **or**
- ≤ Positive Troponin **or**
- ≤ New ECG changes **or**
- ≤ Positive stress test

Restratify to Low Risk & Discharge if:

- ≤ Negative stress test **or**
- ≤ Stress test available within 72 hrs** **and**
- ≤ No further chest pain/symptoms
- ≤ Repeat ECG & vital signs, if stable discharge

NB: ** If stress test is not available within 72 hrs of discharge, treatment plan should be guided by nominated SMO/Cardiologist

Pharmacological stress test or CT coronary angiography may be indicated

LOW RISK

DISCHARGE

- ≤ Regular vital signs
- ≤ Repeat ECG immediately if symptoms recur
- ≤ Repeat ECG 8 hrs post onset of symptoms
- ≤ Repeat Troponin at 8 hrs if 1st sample negative *
- ≤ ECG/Troponin review by medical officer

_____

Restratify Risk if:

- ≤ Recurrent ischaemic chest pain **or**
- ≤ Positive Troponin **or**
- ≤ New ECG changes

If low Risk ACS

- ≤ Discharge
- ≤ Follow up GP/LMO within 3-5 days of D/C
- ≤ Consider Specialist follow up
- ≤ Consider discharge on Aspirin (discuss with SMO)
- ≤ Vital signs prior to discharge

If unlikely cardiac cause

Consider alternative diagnosis

Exit Pathway

***If a high sensitivity troponin assay is used, the testing interval may be reduced to 3 hours, provided the second sample is taken at least 6 hours after symptom onset.**

Medical Officer: Print name & sign_____ **Date**_____

Medical Officer Designation_____

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BINDING MARGIN - NO WRITING

NH606600 - 120511

NSW GOVERNMENT Health

Facility:

FAMILY NAME	MRN
GIVEN NAME	☐ MALE ☐ FEMALE
D.O.B. _____ / _____ / _____	M.O.
ADDRESS	
LOCATION / WARD	

COMPLETE ALL DETAILS OR AFFIX PATIENT LABEL HERE

CHEST PAIN PATHWAY
PRIMARY PCI SITE

STEMI MANAGEMENT

1. CONFIRM INDICATIONS for REPERFUSION

- ☐ Chest pain > 30 min and < 12 hrs
- ☐ Persistent ST segment elevation of ≥ 1 mm in two or more contiguous limb leads or ST segment elevation of ≥ 2 mm in two contiguous chest leads or presumed new LBBB pattern
- ☐ Myocardial infarct likely from history

Time of diagnostic ECG

☐☐:☐☐

2. GENERAL MANAGEMENT

☐ Cardiac monitoring	☐ ECG	☐ IV Cannula X 2
☐ Routine bloods	☐ Oxygen	☐ Analgesia – Morphine
☐ Nitrates-Sublingual or IV	☐ CXR	☐ Beta Blockers

3. ADMINISTER ANTITHROMBOTIC THERAPY

Confirm administration or give:

- ☐ Aspirin 300 mg (soluble)
- ☐ Clopidogrel 300 - 600 mg (or prasugrel &/or tirofiban)
- ☐ Enoxaparin 30 mg IV then bd (or IV heparin or bivalirudin) 1 mg/kg subcut (Max 100 mg)

Refer to local protocols &/or Therapeutic Guidelines

4. CHOOSE REPERFUSION METHOD

PRIMARY PCI UNLESS

- ☐ Significant delay to availability of Cath Lab or interventional team or
- ☐ Patient does not consent to primary PCI
- ☐ History, contrast allergy
- ☐ Vascular access problems
- ☐ Discuss with Interventional cardiologist: Time ☐☐:☐☐
- ☐ Decision regarding reperfusion method: Time ☐☐:☐☐

5. TRANSFER TO CATH LAB

Discuss adjunctive treatment with Cardiologist

OR

THROMBOLYSE if appropriate

- ☐ No contraindications (see page 4)
- ☐ Tenecteplase / Reteplase

Body Weight _____kg Dose _____

Time administered ☐☐:☐☐

- ☐ Repeat ECG at 60 mins post thrombolytic
- ☐ Discuss further mx with cardiologist
- ☐ Failure to reperfuse (less than 50% reduction in ST elevation)

Consider Rescue Angioplasty

Cath Lab arrival time

☐☐:☐☐ please use 24 hr Clock

On table time

☐☐:☐☐

First device use time

☐☐:☐☐

Time to Revascularisation (TIMI 3 flow) Yes / No Time ☐☐:☐☐

☐ 0-30 mins ☐ 31-45 mins ☐ 46-60 mins ☐ 61-75 mins ☐ 76-90 mins
☐ >90 mins Reason for delay

Medical Officer: Print name & sign_____ **Date**_____

Medical Officer Designation_____

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CHEST PAIN PATHWAY

NON PRIMARY PCI SITE

Date of Presentation ☐☐/☐☐/☐☐☐☐ Time ☐☐:☐☐ Time of Symptom Onset: ☐☐:☐☐

CHEST PAIN or OTHER SYMPTOMS of MYOCARDIAL ISCHAEMIA

(eg sweating, sudden orthopnea, syncope, dyspnoea, epigastric discomfort, jaw pain, arm pain)

Be aware:
HIGH RISK ATYPICAL PRESENTATIONS
(eg diabetes, renal failure, female, elderly or Aboriginal)

TRIAGE CATEGORY 2

ECG & Vital Signs, expert interpretation within 10 minutes

General Management
- ☐ Oxygen
- ☐ Aspirin
- ☐ IV Access
- ☐ Pain Relief
- ☐ Pathology incl Troponin
- ☐ Chest X-ray

ST ELEVATION or (presumed new) LBBB

Y

Consider Pericarditis
(sharp chest pain, respiratory or positional component)

N

Go immediately to **STEMI MANAGEMENT** (page 3)

N

Consider Aortic Dissection
(back pain, hypertension, absent pulse, BP difference)
Consider Pulmonary Embolism
(severe dyspnoea, respiratory distress, low subscript O_2 saturation)

N

Diagnose
NON ST ELEVATION ACUTE CORONARY SYNDROME (ACS)

STRATIFY ACS RISK

HIGH RISK **Any of the following**	**INTERMEDIATE RISK** **Any of the following and no high risk features**	**LOW RISK** **Any of the following and no high or intermediate risk features**
≤ ACS symptoms are repetitive or prolonged (> 10 min) & still present. ≤ Syncope ≤ History of chronic left ventricular systolic dysfunction (especially if known LVEF < 40%) OR current clinical evidence of LVF. ≤ Previous PCI/CABG < 6 months ≤ Diabetes + typical ACS symptoms ≤ Chronic renal failure + typical ACS symptoms ≤ Haemodynamic compromise (sustained SBP < 90 mmHg and / or new onset mitral regurgitation) ≤ Elevated Troponin (consider haemolysis, renal failure)	≤ ACS symptoms within 48 hrs that occurred at rest, or were repetitive or prolonged (but currently resolved) ≤ Previous PCI/CABG > 6 months ≤ Known coronary heart disease- Esp if prior AMI or known coronary lesion > 50% stenosis ≤ Two or more risk factors of: Hypertension, family history, active smoking or hyperlipidaemia ≤ Chronic renal failure (especially if known GFR < 60 mL/min) + atypical ACS symptoms ≤ Diabetes + atypical ACS symptoms ≤ Age > 65 years	≤ Presentation with clinical features consistent with ACS without intermediate- risk or high-risk features.
≤ Persistent or dynamic ECG changes of ● ST depression ≥ 0.5 mm or ● new T wave inversion ≥ 2 mm ≤ Transient ST elevation (≥ 0.5 mm) in more than two contiguous leads ≤ Sustained VT	≤ ECG is not normal and has changed from previous pain free ECG but does not contain high risk changes.	≤ ECG Normal or unchanged from previous pain free ECG

All cases to be discussed with Senior Medical Officer

Recommended Management on page 2

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Facility:

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ADDRESS	
LOCATION / WARD	

COMPLETE ALL DETAILS OR AFFIX PATIENT LABEL HERE

CHEST PAIN PATHWAY

NON PRIMARY PCI SITE

Contraindications and cautions for thrombolysis use in STEMI[1]

Absolute contraindications:

Risk of bleeding

- Active bleeding or bleeding diathesis (excluding menses)
Significant closed head or facial trauma within 3 months
- Suspected aortic dissection (including new neurological symptoms)

Risk of intracranial haemorrhage

- Any prior intracranial haemorrhage
- Ischaemic stroke within 3 months
- Known structural cerebral vascular lesion (eg, arteriovenous malformation)
- Known malignant intracranial neoplasm (primary or metastatic)

Relative contraindications:

Risk of bleeding

- Current use of anticoagulants: the higher the international normalised ratio (INR), the higher the risk of bleeding
- Non-compressible vascular punctures
- Recent major surgery (< 3 weeks)
- Traumatic or prolonged (> 10 minutes) cardiopulmonary resuscitation
- Recent (within 4 weeks) internal bleeding (eg, gastrointestinal or urinary tract haemorrhage)
- Active peptic ulcer

Risk of intracranial haemorrhage

- History of chronic, severe, poorly controlled hypertension
- Severe uncontrolled hypertension on presentation (> 180 mmHg systolic or > 110 mmHg diastolic)
- Ischaemic stroke more than 3 months ago, dementia, or known intracranial abnormality not covered in contraindications

Other

- Pregnancy

[1] Adapted from NHF/CSANZ Guidelines for the management of acute coronary syndromes 2006

Contraindications to Exercise Testing (ACC/AHA Guidelines)[2]

Absolute

- Recurrent chest pain
- Acute myocardial infarction, within 2 days
- High-risk unstable angina
- Uncontrolled cardiac arrhythmias causing symptoms or haemodynamic compromise
- Symptomatic severe aortic stenosis
- Uncontrolled symptomatic heart failure
- Acute pulmonary embolus or pulmonary infarction
- Acute myocarditis or pericarditis
- Acute aortic dissection

Relative

- Critical left main coronary stenosis
- Moderate stenotic valvular heart disease
- Electrolyte abnormalities
- Systolic hypertension > 200 mmHg
- Diastolic hypertension > 100 mmHg
- Tachyarrhythmias or bradyarrhythmias
New onset atrial fibrillation
Hypertrophic cardiomyopathy and other forms of outflow obstruction
- Mental or physical impairment leading to the inability to exercise adequately
- High-degree atrioventricular block
Resting ECG which will make EST interpretation difficult (eg LBBB, LVH with strain, Ventricular pacing, Ventricular preexcitation.)

[2] Gibbons etal, Circulation 106:1883,2002

Abbreviations:

ACS – Acute Coronary Syndrome
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LBBB – Left Bundle Branch Block
LVF – Left Ventricular Failure
LVH – Left Ventricular Hypertrophy
PCI – Percutaneous Coronary Intervention
SMO – Senior Medical officer
STEMI – ST Elevation Myocardial Infarction

BINDING MARGIN - NO WRITING

NSW GOVERNMENT Health

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ADDRESS	
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CHEST PAIN PATHWAY

NON PRIMARY PCI SITE

Recommended Further Management Refer to drug protocols &/or Therapeutic Guidelines

HIGH RISK

ADMIT or TRANSFER

≤ Continuous cardiac monitoring & frequent vital signs

≤ Repeat ECG immediately if symptoms recurs

≤ Repeat ECG 8 hrs post onset of symptoms

≤ Repeat Troponin at 8 hrs if 1st sample negative *

≤ ECG/Troponin review by medical officer

Antiplatelet therapy

≤ Yes

≤ No

} Discuss with cardiologist /SMO

If no reason_____

_____

Betablocker

≤ Yes

≤ No

If no reason_____

_____

Anticoagulant

≤ YES

≤ No

If no reason_____

_____

Symptomatic treatment of ongoing pain/hypertension

≤ IV GTN (titrate against pain & BP)

≤ IV Morphine

≤ Refer to nominated cardiologist for further management

INTERMEDIATE RISK

RESTRATIFY

≤ Continuous cardiac monitoring & frequent vital signs

≤ Repeat ECG immediately if symptoms recur

≤ Repeat ECG 8 hrs post onset of symptoms

≤ Repeat Troponin at 8 hrs if 1st sample negative *

≤ ECG/Troponin review by medical officer

Refer for Exercise Stress Test ** if :

≤ No further chest pain/symptoms **and**

≤ 2 negative Troponin tests **and**

≤ No new ECG changes and

≤ No contraindications to stress test (page 4)

Restratify to High Risk if:

≤ Recurrent ischaemic chest pain **or**

≤ Positive Troponin **or**

≤ New ECG changes **or**

≤ Positive stress test

Restratify to Low Risk & Discharge if:

≤ Negative stress test **or**

≤ Stress test available within 72 hrs** **and**

≤ No further chest pain/symptoms

≤ Repeat ECG & vital signs, if stable discharge

NB: ** If stress test is not available within 72 hrs of discharge, treatment plan should be guided by nominated SMO/Cardiologist

Pharmacological stress test or CT coronary angiography may be indicated

LOW RISK

DISCHARGE

≤ Regular vital signs

≤ Repeat ECG immediately if symptoms recur

≤ Repeat ECG 8 hrs post onset of symptoms

≤ Repeat Troponin at 8 hrs if 1st sample negative *

≤ ECG/Troponin review by medical officer

_____

Restratify Risk if:

≤ Recurrent ischaemic chest pain **or**

≤ Positive Troponin **or**

≤ New ECG changes

If low Risk ACS

≤ Discharge

≤ Follow up GP/LMO within 3-5 days of D/C

≤ Consider Specialist follow up

≤ Consider discharge on Aspirin (discuss with SMO)

≤ Vital signs prior to discharge

If unlikely cardiac cause

Consider alternative diagnosis

Exit Pathway

***If a high sensitivity troponin assay is used, the testing interval may be reduced to 3 hours, provided the second sample is taken at least 6 hours after symptom onset**.

Medical Officer: Print name & sign_____ **Date**_____

Medical Officer Designation_____

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BINDING MARGIN - NO WRITING

120511

NSW GOVERNMENT Health

Facility:

FAMILY NAME	MRN
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CHEST PAIN PATHWAY
NON PRIMARY PCI SITE

STEMI MANAGEMENT

1. CONFIRM INDICATIONS for REPERFUSION

- ☐ Chest pain > 30 min and < 12 hrs
- ☐ Persistent ST segment elevation of ≥ 1 mm in two or more contiguous limb leads or ST segment elevation of ≥ 2 mm in two contiguous chest leads or presumed new LBBB pattern
- ☐ Myocardial infarct likely from history

Time of diagnostic ECG

☐☐:☐☐

2. GENERAL MANAGEMENT

- ☐ Cardiac monitoring
- ☐ Routine bloods
- ☐ Nitrates-Sublingual or IV
- ☐ ECG
- ☐ Oxygen
- ☐ CXR
- ☐ IV Cannula X 2
- ☐ Analgesia – Morphine
- ☐ Beta Blockers

3. ADMINISTER ANTITHROMBOTIC THERAPY

Confirm administration or give:

- ☐ Aspirin 300 mg (soluble)
- ☐ Clopidogrel 300 600 mg (or prasugrel &/or tirofiban)
- ☐ Enoxaparin 30 mg IV then bd (or IV heparin or bivalirudin)
  1 mg/kg subcut (Max 100 mg)

Refer to local protocols &/or Therapeutic Guidelines

4. CHOOSE REPERFUSION METHOD

THROMBOLYSIS UNLESS

- ☐ Absolute or unacceptable relative contraindications (see page 4) **or**
- ☐ Patient does not consent to thrombolysis **or**
- ☐ Documented system for transfer to PRIMARY PCI SITE in place

☐ Discussed with cardiologist: Time ☐☐:☐☐

5. THROMBOLYSE

☐ Tenecteplase / Reteplase

Body Weight _____kg Dose _____

Time administered ☐☐:☐☐

OR → ☐ **Transfer to PRIMARY PCI SITE if appropriate**
(As per table below)

Maximum Acceptable Delay from First Medical Contact (FMC):	
Time since symptom onset	**Acceptable delay from FMC to percutaneous intervention**
< 1hours	**60 minutes**
1-3 hours	**90 minutes**
3-12 hours	**120 minutes**
>12hours	**Not routinely recommended**

from NHF/CSANZ Guidelines for the management of acute coronary syndromes 2006

- ☐ Discuss further management immediately with nominated cardiologist
- ☐ Prioritise urgency of transfer with nominated cardiologist
- ☐ Organise transfer to PCI-capable hospital (as per locally agreed protocol)
- ☐ Repeat ECG at 60 mins post thrombolytic

Medical Officer: Print name & sign_____ **Date**_____

Medical Officer Designation_____

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