CLINICAL PRACTICE GUIDELINES

MANAGEMENT OF

ACUTE ST SEGMENT ELEVATION

MYOCARDIAL INFARCTION [STEMI]

2007 - (2nd Edition)
This guideline is meant to be a guide for clinical practice, based on the best available evidence at the time of development. Adherence to this guideline may not necessarily guarantee the best outcome in every case. Every health care provider is responsible for the management of his/her unique patient based on the clinical picture presented by the patient and the management options available locally.

This guideline was issued in 2007 and will be reviewed in 2010 or sooner if new evidence becomes available.

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http://www.moh.gov.my  
http://www.acadmed.org.my

This is an update to the Clinical Practice Guideline on ST Elevated MI (published 2001). This CPG supersedes the previous CPG on ST Elevated MI (2001)
MESSAGE FROM THE DIRECTOR GENERAL OF HEALTH

In this new millennium, there has been an exponential increase of scientific knowledge and publications. Indeed, one of the greatest challenges of modern day medical practice is to keep pace with this information and applying them into routine clinical practice.

The publication of the Malaysian CPG on STEMI by the National Heart Association of Malaysia, Academy of Medicine and Ministry of Health Malaysia is therefore very timely. Apart from the updates in the clinical scientific knowledge, adaptations have been made for the local setting. With this CPG, patients presenting to any of the health care institutions in the country with STEMI will be assured of the latest standards of clinical practice. However, for any CPG to be a success, it has to be utilized optimally and updated from time to time.

I would like to commend to the expert panel for the time and effort taken by them in updating this CPG and for health care practitioners to translate knowledge in this CPG into routine clinical practice, for the benefit of our fellow Malaysians.

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Rationale and Process of Development of this CPG

Rationale

Acute Myocardial Infarction is a major health problem, with relatively high morbidity and mortality. Although mortality has been reduced, it is still substantial. In the management of established acute myocardial infarction, time is the essence.

The Clinical Practice Guidelines (CPG) in the Management of ST Elevated Myocardial Infarction (STEMI) was developed to provide a clear and concise approach to all clinicians on the current concepts in the management of acute MI patients. In Malaysia, a significant number of acute MI patients are managed by non-cardiologists. We feel that it is important to summarise and adapt relevant clinical trial data and current treatment strategies to our local practice.

The 1st CPG on STEMI was published in 2001. Since then, there have been many new developments in the management of STEMI. Thus an update on the latest and current guidelines in the 1st revision on the CPG in STEMI would be most appropriate.

This CPG has been prepared by a panel of committee members appointed by the National Heart Association of Malaysia (NHAM), Academy of Medicine (AOM) and Ministry of Health (MOH). The committee members comprise of cardiologists and general physicians from the government, private sector and the Universities.

Objectives

These guidelines are intended to provide awareness and education in

• early recognition of STEMI
• evidence-based practice for the management of STEMI
• secondary prevention following STEMI

with the intention of reducing the morbidity and mortality associated with STEMI

Process

Evidence was obtained by systematic review of current medical literature on STEMI using search engines available online. The other international guidelines on STEMI were also taken into consideration. After many in-depth discussions, the draft was completed by the members of the Expert Panel and submitted to key health personnel in major hospitals of the Ministry of Health, Malaysia, universities and private sector for review and approval.

The level of recommendation and the grading of evidence used in this CPG was adapted from the American Heart Association and the European Society of Cardiology. The evidence supporting the recommendation was
graded as A if the data was derived from multiple randomized clinical trials involving a large number of individuals or meta-analyses. Evidence was graded as B if the data was derived from a single randomized clinical trial or limited to non randomized clinical trials or observational data registries. Evidence was graded C if the recommendation was based on consensus of expert opinion or case studies only.

In certain conditions where there are no clinical trials but nevertheless the practice is recommended based on years of clinical experience and is thus well supported even though the evidence is ranked as C. An example is anticoagulation in the presence of a large mobile left ventricular thrombus. The levels of recommendation were ranked as I, IIa, IIb or III as outlined in page V.

Clinical Questions Addressed
• How do you diagnose STEMI?
• What is the best strategy to treat STEMI patients based on the current evidence available?
• How to reduce the risk of a subsequent cardiovascular event?
• How to treat the following special groups?
  - Elderly
  - Diabetics
  - Women

Target Group:
These guidelines are developed for all healthcare providers involved in the management of STEMI in adults.

Target Population:
These guidelines are developed to treat all adults with STEMI
LEVELS OF EVIDENCE AND GRADES OF RECOMMENDATION

<table>
<thead>
<tr>
<th>LEVELS OF EVIDENCE</th>
<th>DESCRIPTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Conditions for which there is evidence and/or general agreement that a given procedure/therapy is beneficial, useful and/or effective.</td>
</tr>
<tr>
<td>II</td>
<td>Conditions for which there is conflicting evidence and/or divergence of opinion about the usefulness/efficacy of a procedure/therapy.</td>
</tr>
<tr>
<td>II-a</td>
<td>Weight of evidence/opinion is in favor of its usefulness/efficacy.</td>
</tr>
<tr>
<td>II-b</td>
<td>Usefulness/efficacy is less well established by evidence/opinion.</td>
</tr>
<tr>
<td>III</td>
<td>Conditions for which there is evidence and/or general agreement that a procedure/therapy is not useful/effective and in some cases may be harmful.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>GRADES OF RECOMMENDATION</th>
<th>DESCRIPTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Data derived from multiple randomized clinical trials or meta analyses</td>
</tr>
<tr>
<td>B</td>
<td>Data derived from a single randomized clinical trial or large non randomized studies</td>
</tr>
<tr>
<td>C</td>
<td>Only consensus of opinions of experts, case studies or standard of care</td>
</tr>
</tbody>
</table>

(Adapted from the American Heart Association and the European Society of Cardiology)
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1. INTRODUCTION

Cardiovascular disease remains an important cause of death in Malaysia accounting for 20-25% of all deaths in government hospitals from 2000-2005. Following an acute myocardial infarction (AMI) the mortality rate was about 20% in 2004.1 This high rate could have been due to late presentation and diagnosis, leading to delayed treatment. Occasionally the diagnosis could have been missed. In developed countries the mortality rate following an AMI had decreased to less than 9% by the early 2000’s.2

Much progress has been made in the management of AMI, especially in the last two decades with the discovery that early reperfusion therapy results in myocardial salvage and a significant reduction in morbidity and mortality. The majority of deaths occur soon after the onset of symptoms, the “pre-hospital phase”. To reduce these deaths, the public needs to be educated of the symptoms of an infarct and on the need to seek medical attention immediately. Healthcare personnel should be sensitized and trained to deal with these patients urgently and appropriately. Emergency Departments in hospitals need to develop critical pathways and management strategies to maximize treatment benefits.

Guidelines help in the management of patients. All the recommendations stated in this guideline may not be available to all eligible patients. Patient care should be individualized and sound clinical judgement plays an important role in decision making.

2. TERMINOLOGY

Acute coronary syndrome is a clinical spectrum of ischemic heart disease ranging from unstable angina, non-ST-elevation myocardial infarction (NSTEMI) to ST-elevation myocardial infarction (STEMI) depending upon the degree and acuteness of coronary occlusion.(see Figure 1)

STEMI: Myocardial infarction due to acute total occlusion of the coronary artery
NSTEMI: Myocardial infarction due to acute sub-total occlusion of the coronary artery

2.1 Pathogenesis of STEMI

STEMI is necrosis of heart muscle due to inadequate blood supply following an acute total coronary occlusion. This occlusion is usually due to atherosclerotic plaque rupture, fissuring or ulceration with superimposed thrombosis and coronary vasospasm. Rarely, it may result from non-atherosclerotic arterial disease such as coronary vasospasm alone, coronary embolism or vasculitis³.
2.2 Clinical Diagnosis of STEMI

It is diagnosed by:

i. Clinical history of ischaemic type chest pain
   - New onset ST-segment elevation of:
     - \( \geq 0.1 \text{ mV} \) in 2 contiguous limb leads, or V4 to V6
     - \( \geq 0.2 \text{ mV} \) in 2 contiguous precordial leads V1 to V3
   - Presumed new left-bundle branch block

ii. ECG changes – The following are integral to the diagnosis of STEMI:
   - Evidence of myocardial injury or necrosis as indicated by elevated serum cardiac biomarkers

2.2.1 History

A good history is vital in raising the clinical suspicion and making a diagnosis of STEMI. Chest pain of STEMI is typically retrosternal, severe, crushing, squeezing or pressing in nature, lasting more than 30 minutes, associated with profuse sweating, nausea, vomiting and shortness of breath. It is sometimes described as chest tightness only. The pain may radiate to the jaw or down the left upper limb. It may occur at rest or with activity. Occasionally the pain may be burning in nature and of lesser severity. It may be in the epigastric region and be misinterpreted as indigestion or heart burn. Rarely may it be localized to the back in the interscapular region only resulting in a misdiagnosis.

Other atypical presentations include unexplained nausea and vomiting, weakness, dizziness, lightheadedness and syncope, which may occur in the presence or absence of chest pain.
Diabetics, the elderly and females may not present with typical chest pains. Common presenting symptoms in these patients are dyspnoea and atypical chest pains.

Other important points to note in the history are the presence of:
- previous history of ischemic heart disease, percutaneous coronary intervention (PCI) or coronary artery bypass surgery (CABG)
- risk factors for atherosclerosis
- symptoms suggestive of previous transient ischemic attack or other forms of cerebrovascular disease
- symptoms suggestive of peripheral vascular disease

2.2.2 Electrocardiographic changes

The diagnosis of STEMI depends upon the presence of characteristic ECG changes. These evolving ECG changes are hyperacute changes of a tall peaked T-wave, ST segment elevation followed by the development of Q-wave, return of the ST segment to isoelectric and T-wave inversion. The cut off points for new or presumed new ST segment elevation are ≥ 0.2mV in leads V1, V2, or V3 and ≥ 0.1mV in other leads. This should be present in 2 or more contiguous leads. The presence of new onset or presumably new left bundle branch block (LBBB) in a patient with typical type chest pain indicates an infarct.

However in some cases especially when the patient presents early, the ECG may be normal or equivocal. In patients with ongoing chest pain and in whom the clinical index of suspicion of STEMI is high, 12 lead ECG tracings repeated at close intervals of at least 15 minutes might show evolving changes. Comparison with previous ECG’s may also be helpful in such situations.

In patients with an inferior infarct, one should look for associated posterior, lateral and RV infarct. The latter requires right sided chest leads for diagnosis.

For localization of infarct see Table 1.

<table>
<thead>
<tr>
<th>Location</th>
<th>Leads</th>
<th>ECG findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anteroseptal</td>
<td>V1 – V3</td>
<td>ST elevation, Q wave</td>
</tr>
<tr>
<td>Extensive anterior</td>
<td>V1 – V6</td>
<td>ST elevation, Q wave</td>
</tr>
<tr>
<td>Posterior</td>
<td>V7 – V8</td>
<td>T elevation, Q wave</td>
</tr>
<tr>
<td>Posterior</td>
<td>V1 – V2</td>
<td>ST depression, Tall R wave</td>
</tr>
<tr>
<td>Anterolateral</td>
<td>I, AVL, V5 – V6</td>
<td>ST elevation, Q wave</td>
</tr>
<tr>
<td>Inferior</td>
<td>II, III, AVF</td>
<td>ST elevation, Q wave</td>
</tr>
<tr>
<td>Right Ventricular</td>
<td>V4R, V5R</td>
<td>ST elevation, Q wave</td>
</tr>
</tbody>
</table>

2.2.3 Serum Cardiac Biomarkers

A rise and fall in the levels of serum cardiac biomarkers support the diagnosis of STEMI. One should not, however, wait for the results of these biomarkers before initiating reperfusion therapy. These cardiac biomarkers include:

- Cardiac troponins (cTnT and cTnI)
• Creatine kinase-Myocardial Band (CK-MB)
• Creatine kinase (CK)
• Myoglobin
• Fatty Acid Binding Proteins

For the relative timing, rate of rise, peak value, duration of elevation and properties of these cardiac biomarkers following STEMI, see Figure 2 and Table 2.

Cardiac troponins and CK-MB are the most specific cardiac biomarkers. It takes about 3-8 hours after STEMI for them to rise. Thus, too early a measurement may result in a misleadingly low level.

For the diagnosis of STEMI, the value of CK-MB should be twice the upper limit of normal. Persistently elevated values of CK-MB are almost never due to myocardial necrosis. CK-MB rises early and falls early. Hence, CK-MB measurements are useful for the diagnosis of reinfarction. CK is not as sensitive or as specific as CK-MB. Nevertheless, it is also useful for the diagnosis of STEMI and reinfarction.

A single measurement of a raised Troponin T or I (99th percentile of the values for a reference control group) is sufficient to indicate myocardial necrosis. They are useful in detecting myocardial infarction in patients presenting with atypical histories and non diagnostic ECG’s. Elevated troponin levels are more important for the diagnosis of NSTEMI than STEMI. Troponins may remain elevated for up to 14 days. Therefore it is not useful for the diagnosis of reinfarction.

It is recommended that measurement of cardiac biomarkers be done at periodic intervals, at hospital admission and again at 12-24 hours. This would help to establish or exclude the diagnosis and may be useful for an estimation of infarct size.

AST and LDH levels are not sensitive or specific for AMI with frequent false positive elevations.

Figure 2: Time Course of Elevation of Serum Cardiac Biomarkers after STEMI

(Reproduced with permission from “Clinical Implications of the new definition of myocardial infarction”. John K French, Harvey D White; Heart 2004;90:99–106)
### 2.2.4 Other Diagnostic Modalities.
Imaging techniques such as chest radiography, echocardiography, multi-slice computed tomography (MSCT) and radionuclide techniques are useful investigations in the patient presenting with acute chest pain. They help to:

- Rule out or confirm the presence of acute infarction or ischaemia.
- Identify non-ischaemic conditions causing chest pain such as valvular heart disease, pulmonary embolism, aortic dissection and pneumothorax.
- Identify mechanical complications of acute infarction.
- Provide prognostic information.

Echocardiography is a particularly useful bedside imaging technique in difficult diagnostic situations.

### 2.2.5 Difficult Diagnosis

Sometimes the diagnosis of myocardial infarction is difficult. About 2-8% of patients presenting with chest pains to the emergency department have been misdiagnosed and sent home. The morbidity and mortality in these patients is high. To reduce this misdiagnosis, we suggest the following measures be taken in all patients presenting with chest pains:

- They should be given priority in the emergency department and attended to urgently.
- Myocardial ischemia or infarction should be excluded in all these patients.
- Clinical suspicion should be high in all patients with predisposing risk factors for atherosclerosis.
- A careful history will often help in making the diagnosis.
- An ECG should be done as soon as possible in all patients with chest pains especially when the clinical suspicion of AMI is high. The threshold for doing an ECG in a patient presenting with chest pain should be low.
- Where the initial ECG is non-diagnostic, it should be repeated and compared with old ECG's. Thus it is important to maintain good clinical records that can be rapidly retrieved.

#### Table 2: Properties of Serum Cardiac Biomarkers

<table>
<thead>
<tr>
<th>Protein</th>
<th>First detection*</th>
<th>Duration of detection</th>
<th>Sensitivity</th>
<th>Specificity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fatty acid binding protein</td>
<td>1.5 – 2 hours</td>
<td>8 – 12 hours</td>
<td>+++</td>
<td>++</td>
</tr>
<tr>
<td>Myoglobin</td>
<td>1.5 – 2 hours</td>
<td>8 – 12 hours</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>CK-MB</td>
<td>2 – 3 hours</td>
<td>1 – 2 days</td>
<td>+++</td>
<td>+++</td>
</tr>
<tr>
<td>Troponin I</td>
<td>3 – 4 hours</td>
<td>7 – 10 days</td>
<td>++++</td>
<td>++++</td>
</tr>
<tr>
<td>Troponin T</td>
<td>3 – 4 hours</td>
<td>7 – 14 days</td>
<td>++++</td>
<td>++++</td>
</tr>
<tr>
<td>CK</td>
<td>4 – 6 hours</td>
<td>2 – 3 days</td>
<td>++</td>
<td>++</td>
</tr>
<tr>
<td>Aspartate Transaminase</td>
<td>6 – 10 hours</td>
<td>3 – 5 days</td>
<td>++</td>
<td>+</td>
</tr>
<tr>
<td>LDH</td>
<td>6 –10 hours</td>
<td>5 – 7 days</td>
<td>++</td>
<td>+</td>
</tr>
</tbody>
</table>

* Hours after symptom onset.

CK, creatine kinase; LDH, lactate dehydrogenase

(Reproduced with permission from "Clinical Implications of the new definition of myocardial infarction". John K French, Harvey D White; Heart 2004;90:99–106)
Cardiac biomarkers especially the troponins, are helpful in ruling in or ruling out a myocardial infarction.

Where the diagnosis is unclear but the clinical suspicion is high, these patients should be observed in the emergency department for a few hours and the resting ECG and cardiac biomarkers repeated to look for serial changes. If these remain stable, then the patient may be sent home but asked to return for an early review in the outpatient clinic.

In addition, the hospital needs to:

- educate all medical staff on the importance of early detection and treatment of AMI because this results in myocardial salvage and improved patient outcomes.
- have regular refresher courses on ECG interpretation.
- implement critical pathways for patients presenting with chest pains to the emergency department.

Recommendations:

- The public and allied health care personnel should be educated on the importance of early diagnosis and the benefits of early reperfusion therapy.

- STEMI is diagnosed by the clinical history of chest pain, ECG changes of ST elevation/ LBBB and elevated serum cardiac biomarkers.

- Atypical presentations can occur in the elderly, women and in diabetic patients.

- If the initial ECG is non-diagnostic, it may need to be repeated at frequent intervals to detect evolving changes of STEMI.

- Too early a measurement can sometimes result in a misleadingly low level of serum cardiac biomarkers.
3. PRE-HOSPITAL MANAGEMENT

Immediate measures to be taken in suspected cases of STEMI

3.1 For the general public:
- Seek immediate medical attention at the nearest hospital.
- Call for an ambulance (dial 991 or hospital direct line if known) or get someone to take you immediately to the nearest hospital.
- Do not drive yourself.
- If not on regular aspirin and with no history of allergy, chew and swallow one 300mg tablet of aspirin immediately.

3.2 For Patients with known CHD:
- If the pain is suggestive of STEMI (see section 2.2.1), take one dose of sublingual GTN and be rapidly transported to the hospital.
- If the pain is not severe, take one tablet of GTN and repeat every 5 minutes for a maximum of 3 doses. If the pain still persists after 15 minutes, go to the hospital.

3.3 For the general practitioner / family physician:
- Ask patient to chew and swallow one 300mg tablet of aspirin.
- Give sublingual GTN.
- If the ECG shows ischemic changes, give 300mg of clopidogrel if available.
- Wherever possible, set up intravenous access.
- Pain relief with intravenous opiates (IV morphine 3-5mg slowly).
- Avoid intramuscular injections since this could result in intramuscular hematomas if fibrinolytic agents are subsequently administered.
- Call an ambulance or ask the patient’s relative or friend to send the patient immediately to the nearest hospital.

3.4 For Allied Health Care Personnel:

Immediate measures to be taken when there is an ambulance call:-
- Note nature of complaint.
- Obtain name of caller, address and telephone number.
- If possible, request that a relative or friend wait at a strategic place to help locate the patient.
- Dispatch an adequately equipped ambulance with trained paramedics immediately.
- Patient should be given oxygen and aspirin (if he has not taken) and transported to hospital.
• Upon reaching the hospital, the patient should be taken directly to the Emergency Department.

An ECG should be done as soon as possible. In hospitals where networking facilities are available, allied health care personnel in the ambulance should relay/transmit the ECG to the call centre for review by the Specialist, for consideration of pre-hospital fibrinolysis or primary PCI.

Allied Health care personnel should be trained:-
• to identify patients at high risk of developing CHD.
• to identify patients presenting with AMI.
• on the importance of early referral and treatment.
• in basic and advanced cardiopulmonary resuscitation (CPR).

**Recommendations:**

- Patients with suspected STEMI should be given sublingual GTN, aspirin and clopidogrel.
- These patients should be rapidly transported to the hospital.
4. IN-HOSPITAL MANAGEMENT
Early management of STEMI is directed at:
• Pain relief
• Establishing early reperfusion
• Treatment of complications - arrhythmias

4.1 Initial Recognition and Management

When the patient with suspected STEMI reaches the emergency department, evaluation and initial management should take place promptly (FAST TRACK - RED ZONE) because the benefits of reperfusion therapy is greater the earlier it is initiated.

A quick targeted history should be taken and vital signs noted. The diagnosis should be confirmed with an ECG, which should be done as soon as possible, preferably within 10 minutes of the patient’s arrival in the emergency department. It is important to relieve pain and quickly assess the patient’s suitability for reperfusion by either fibrinolytic therapy or primary PCI.

The following should be done immediately and concomitantly in the emergency department (see flow chart 1, page 44):

- Assessment and stabilization of the patient’s haemodynamics.
- Sublingual GTN if chest pain persists (unless systolic blood pressure (SBP) < 90 mmHg).
- Continuous ECG monitoring.
- 300mg of aspirin chewed and swallowed if not given earlier.
- Clopidogrel at a dose of 300mg should be given if not given earlier.
- Oxygen by nasal prongs / facemask.
- Venous access established and blood taken for cardiac biomarkers, full blood count, renal profile, glucose and lipid profile. Preferably 2 intravenous lines should be set up.
- Pain relief - morphine should be administered intravenously at 2-5mg every 5-15 minutes until pain is relieved. Watch for evidence of toxicity – hypotension and respiratory depression. Anti-emetics (IV metoclopromide 10mg or promethazine 25mg) should be given.
- Intramuscular injections should be avoided.
- Assessment for reperfusion strategy.
4.2 Reperfusion Strategies

- Early and prompt reperfusion is crucial as **TIME LOST** is equivalent to **MYOCARDIUM LOST**. 12,13,14.

- Despite overwhelming data showing that prompt reperfusion therapy improves survival 12,13, it is still widely underutilized and delayed.

- Most studies indicate that primary PCI is superior to fibrinolytic therapy as a reperfusion strategy. 15,16.

- However, in patients who present within 3 hours of symptom onset and are at low risk, both treatment strategies appear to have similar benefits. 17,18.

In the majority of our hospitals, fibrinolytic therapy is more readily available and constitutes the main reperfusion strategy. If both choices are available, the following factors help guide the choice of reperfusion strategies:

- Time from symptom onset to first medical contact
- Time delay to PCI (time from hospital arrival to balloon dilatation – door to balloon time)
- Time to hospital fibrinolysis (time from hospital arrival to administration of fibrinolytic therapy – door to needle time)
- Contraindications to fibrinolytic therapy
- High risk patients

The best reperfusion strategy will depend upon:

**A) Time from onset of symptoms**

- **Early presentation (within 3 hours)**

  - If both treatment options are readily available, they have been shown to be equally effective 17,18 except for the following situations where primary PCI is the preferred strategy:
    - fibrinolytic therapy is contraindicated
    - in high-risk patients
    - PCI time delay [(door-to-balloon time) – (door-to-needle time)] is less than 60 minutes 19

- **Late presentation (3 to 12 hours)**

  - Primary PCI is preferred 15,16. The door to balloon time should be within 90 min if the patient presents at a PCI capable facility 19.
    - If transferred from a center with no PCI facilities, it should be less than 2 hours. (including transfer delay) 20
    - If the time delay to primary PCI is longer than as mentioned, then fibrinolytic therapy should be given.

- **Very late presentation (> 12 hours)**

  - Both primary PCI and fibrinolytic therapy are not routinely recommended in patients who are asymptomatic and haemodynamically stable 12.
However, reperfusion therapy would still be beneficial in patients with persistent ischaemic symptoms, haemodynamic or electrical instability. In this subgroup, primary PCI is the preferred strategy.

**B) Contraindications to fibrinolytic therapy**

See section 4.2.1.2

**C) High risk patients**

These include patients with:
- Large infarcts
- Anterior infarcts
- Cardiogenic shock
- Elderly patients
- Post revascularization (post CABG and post PCI)
- Post infarct angina

Primary PCI is the preferred strategy in these patients\(^{21,22,23}\).

The goals of time to reperfusion therapy should be within:
- 30 minutes door to needle time\(^{12,13}\)
- 90 minutes door to balloon time\(^{19}\)

**4.2.1 Fibrinolytic Therapy**

Fibrinolytic therapy has been shown to reduce mortality when given within the appropriate time frame. When given within 1 hour from time of onset of symptoms, it is most beneficial and has been shown to be able to abort the infarction and reduce mortality by up to 50%\(^{12,17}\).

The door-to-needle time should be within 30 mins. Strategies should be put in place to achieve this target. Fibrinolytic therapy should be made available in all hospitals and there should be protocols to initiate it in the emergency department.

Pre-hospital fibrinolytic therapy has been shown to achieve faster reperfusion\(^{17,24}\).

Patients presenting with a low blood pressure (SBP < 90mm Hg) should receive inotropic support prior to fibrinolytic therapy.

**4.2.1.1 Indications**

Fibrinolytic therapy should only be given to patients with STEMI. It has no role and may even be detrimental in patients with NSTEMI\(^{12,25}\).

**4.2.1.2 Contraindications**

**Absolute contraindications**

*Risk of Intracranial haemorrhage*
- Any history of intracranial haemorrhage
- Ischaemic stroke within 3 months
Known structural cerebral vascular lesion (e.g. arteriovenous malformation)
Known intracranial neoplasm

**Risk of bleeding**
- Active bleeding or bleeding diathesis (excluding menses)
- Significant head trauma within 3 months
- Suspected aortic dissection

**Relative contraindications**

**Risk of intracranial haemorrhage**
- Severe uncontrolled hypertension on presentation (BP > 180/110 mm Hg)*
- Ischaemic stroke more than 3 months ago
- History of chronic, severe uncontrolled hypertension

**Risk of Bleeding**
- Current use of anticoagulation in therapeutic doses (INR > 2)
- Recent major surgery < 3 weeks
- Traumatic or prolonged CPR > 10 minutes
- Recent internal bleeding (e.g. gastrointestinal or urinary tract haemorrhage) within 4 weeks
- Non-compressible vascular puncture
- Active peptic ulcer

**Others**
- Pregnancy
- Prior exposure (>5 days and within 12 months of first usage) to streptokinase (if planning to use same agent)

* The blood pressure should be reduced prior to institution of fibrinolytic therapy.

### 4.2.1.3 Choice of Fibrinolytic Agent

Presently the agents available in Malaysia are:

**Streptokinase**

This is the most widely used agent. It is not fibrin specific and is less efficacious than fibrin selective agents.\(^{26,27}\) Despite having a lower risk of intracranial haemorrhage, the reduction in mortality is less than with fibrin specific agents.\(^{26,28}\)

Streptokinase is antigenic and promotes the production of antibodies. Thus the utilization of this agent for re-infarction is less effective if given again 5 days after the first administration.\(^{29}\)

PCI or fibrin specific agents should then be considered.

**Regimen:**
- 1.5 mega units in 100 ml normal saline or 5% dextrose over 1 hour.

Other regimens are:
- 1.5 mega units over 20 minutes, or
- 0.75 mega unit bolus and then repeated at the same dose after an interval of 50 minutes if there is no clinical reperfusion.
The last 2 regimens achieve a higher coronary artery patency rate and are associated with lower in-hospital mortality. However they are associated with a higher incidence of hypotension.

**Alteplase**

This agent is fibrin specific and achieves better reperfusion at 90 min as compared to streptokinase. However there is a higher rate of reocclusion. Thus heparin needs to be given for 48 hours.

**Regimen:**
- For patients > 65 kg: 15 mg bolus; then 50 mg over 30 min and 35 mg over the next 60 min
- For patients < 65 kg: 15 mg bolus; then 0.75 mg/kg over 30 min and 0.5 mg/kg over the next 60 min

**Second generation fibrin specific agents: Tenecteplase, Reteplase**

Tenecteplase has been recently introduced in Malaysia. These second generation fibrin specific agents are as efficacious as alteplase. Tenecteplase has been shown to have a slightly lower bleeding risk as compared to alteplase. The main advantage of using these agents is that they are easier to administer. They are given as single or double bolus injections and also do not induce antibody production.

**Regimen:**
- Tenecteplase (TNK-tPA) single i.v. bolus
  - 30 mg if < 60 kg
  - 35 mg if 60 to < 70 kg
  - 40 mg if 70 to < 80 kg
  - 45 mg if 80 to < 90 kg
  - 50 mg if > 90 kg

Heparin needs to be given for 48 hours.

**4.2.1.4 Indicators of Successful Reperfusion**

There is no sensitive bedside clinical method to reliably detect successful reperfusion. Some useful guides are:

- resolution of chest pain (may be confounded by the use of narcotic analgesics).
- early return of ST segment elevation to isoelectric line or a decrease in the height of the ST elevation by 50% in the lead that records the highest ST elevation therapy within 60-90 mins of initiation of fibrinolytic therapy.
- early peaking of CK and CK-MB levels.
- restoration and/or maintenance of haemodynamic and/or electrical stability

The occurrence of ‘reperfusion arrhythmias’ is not a reliable indicator of successful reperfusion. An exception is accelerated idioventricular rhythm and sudden sinus bradycardia which have been correlated with a patent infarct related coronary artery after fibrinolytic therapy or primary PCI.
4.2.1.5 Failed Fibrinolysis

Failure of fibrinolytic agents to open up the occluded infarct related artery is manifested as continuing chest pain, persistent ST segment elevation and hemodynamic instability. These patients are more likely to develop complications such as heart failure and arrhythmias.

The treatment of choice for these patients is rescue PCI\(^36\). They should not be given a second dose of a fibrinolytic agent. This is because there has been no difference in event free survival demonstrated if these patients are given a repeat dose of a fibrinolytic agent or if they are treated conservatively\(^36\).

4.2.2. Percutaneous Coronary Intervention (PCI)

4.2.2.1 Primary PCI

Primary PCI is the reperfusion strategy of choice as indicated earlier (section 4.2)\(^15,16\) It should be performed promptly by experienced operators and in centers performing a sufficient number of primary PCI procedures.

4.2.2.2 Facilitated PCI

Current evidence indicates that strategies combining fibrinolytic therapy is associated with higher reperfusion rates and TIMI flow. However, it is associated with higher mortality and bleeding rates. It is therefore not recommended\(^37,38\).

4.2.2.3 Rescue PCI

Rescue PCI may be considered in patients who have failed fibrinolytic therapy or have recurrent chest pain and/or ischaemic complications. Those who may benefit are patients with:

- ongoing chest pains
- haemodynamic and electrical instability
- cardiogenic shock in patient < 75 years old, within 36 hours of STEMI and <18 hours of shock whose coronary anatomy is suitable for revascularization
- heart failure and onset of chest pain within 12 hours
- cardiogenic shock – In these high risk patients, those who are <75 years of age and who present within 36 hours of STEMI and <18 hours of shock may be considered for rescue PCI if their coronary anatomy is suitable for revascularization

This strategy is associated with high mortality and morbidity rates. As such, patients should be individually evaluated.

4.2.2.4 Delayed PCI (> 72 hours after fibrinolytic therapy)

Routine angiography and PCI in asymptomatic and stable patients post fibrinolytic therapy is controversial\(^39,40\).
Recommendations:

- Patients presenting to the hospital need to be rapidly evaluated for prompt reperfusion therapy.

- Reperfusion therapy with either primary PCI or fibrinolytic therapy reduces mortality and is useful in patients presenting within 12 hours from onset of symptoms.

- We should aim to achieve a door-to-needle time of less than 30 min and a door-to-balloon time of less than 90 min.

- Fibrinolytic therapy if given within 3 hours of onset of symptom gives equivalent results as primary PCI.

- The preferred strategy is primary PCI if the time of onset of symptoms is between 3-12 hours.

- The treatment of choice for failed fibrinolysis is rescue PCI.
4.3 CARDIAC CARE UNIT MANAGEMENT

4.3.1 General Measures

A period of at least 12 hours of complete bed rest is recommended following admission to CCU. Patients with uncomplicated infarcts are encouraged to ambulate early. Those with haemodynamic instability will need a longer period of monitoring.

Sedatives may be useful.

Use of bedside commodes and assisted bedside washing should be safe in most patients.

The Valsalva maneuver has been shown to precipitate dangerous haemodynamic and electrocardiographic changes particularly in the young and thus prevention of constipation with stool softeners is encouraged.

4.3.2 Monitoring

The general condition of the patient, vital signs, pulse oximetry and the ECG should be continuously monitored following STEMI, looking for complications.

4.3.3 Concomitant Therapy

4.3.3.1 Oxygen

Oxygen is indicated in the presence of hypoxemia. In uncomplicated cases, its use should probably be limited to the first 24 hours. Oxygen, via nasal prongs, at 2 - 4 litres/min is usually adequate. One should aim to maintain the oxygen saturation above 95%.

4.3.3.2 Antiplatelet Agents

A) Aspirin

Aspirin is indicated in all patients at diagnosis and should be continued indefinitely unless contraindicated. The initial dose of 100-300mg should be followed by a maintenance dose of 75 - 150mg daily.

B) Clopidogrel

Clopidogrel, when given together with aspirin and fibrinolytic therapy in STEMI, has been shown to reduce the odds of an occluded infarct related artery, death or reinfarction without increasing the risk of bleeding or cerebrovascular accidents. A loading dose of 300 mg should be given followed by a maintenance dose of 75 mg daily. We recommend treatment for at least 1 month after fibrinolytic therapy. Following PCI, a longer period of dual antiplatelet therapy (up to 12 months) is necessary particularly when drug-eluting stents are used.
4.3.3.3 β-blockers

Current recommendations are to use oral β-blockers early in all patients without specific contraindications. In patients with asymptomatic LV dysfunction (LV ejection fraction < 40%) and not in overt heart failure, carvedilol has been shown to reduce the frequency of death and recurrent AMI. When indicated, it should be started in patients who are hemodynamically stable after 48 hours.

Contraindications to β-blockers:
1) Bradycardia < 60/minute
2) SBP < 100mmHg
3) Pulmonary congestion with crepitations beyond the lung bases
4) Signs of peripheral hypoperfusion
5) Second or third degree atrio-ventricular (AV) block
6) Asthma or chronic obstructive airway disease (COAD)
7) Severe peripheral vascular disease

Table 3: Recommended dosages of β-blockers in STEMI

<table>
<thead>
<tr>
<th>Type</th>
<th>Initiation dose</th>
<th>Target dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metoprolol</td>
<td>25mg bd</td>
<td>100mg bd</td>
</tr>
<tr>
<td>Atenolol</td>
<td>25mg od</td>
<td>100mg od</td>
</tr>
<tr>
<td>Propranolol</td>
<td>5mg tds</td>
<td>80mg tds</td>
</tr>
<tr>
<td>Carvedilol</td>
<td>3.125mg bd</td>
<td>25mg bd</td>
</tr>
</tbody>
</table>

4.3.3.4 Angiotensin Converting Enzyme Inhibitors (ACEI) and Angiotensin Receptor Blockers (ARB)

Early use of ACEI (within 24 hours) following STEMI has been shown to improve survival. ACEI should be started when the blood pressure is stable and systolic blood pressure (SBP) remains above 100mmHg.

The benefits of ACEI are greatest in patients with:
- Heart failure
- Anterior infarcts
- Asymptomatic left ventricular dysfunction (LV ejection fraction < 40% on echocardiography)

In patients who cannot tolerate ACEI, the ARB, valsartan, has been shown to have a similar survival benefit.

Contraindications to ACEI and ARB therapy:
1) Systolic BP < 100mmHg
2) Established contraindications e.g. bilateral renal artery stenosis, worsening renal function.
4.3.3.5 Nitrates

The routine use of nitrates has not been shown to have a survival benefit\(^{51,52}\).

Nitrates can be considered in patients with:

- Continuing chest pain and/or ischemia
- Heart failure
- Hypertension

In the acute stage, IV nitrates are recommended because of their rapid onset of action, ease of titration and potential for prompt termination in the event of side effects. After the first 48 hours, oral or topical nitrates may be continued in patients with persisting ischemia and/or heart failure.

Contraindications to nitrate therapy:

1) Hypotension (SBP < 90mmHg)
2) RV infarction
3) History of phospho-diesterase 5 inhibitors ingestion depending upon the half-life of the agent. (Appendix 3)

4.3.3.6 Calcium Channel Blockers

There is no data to support the routine use of calcium channel blockers post STEMI\(^ {53,54}\).

However they may be used as adjunctive therapy in patients with hypertension and/or on-going ischaemia despite \(\beta\)-blockers and nitrates.

In patients who cannot tolerate \(\beta\)-blockers, verapamil or diltiazem may be used for secondary prevention\(^ {55}\).

Calcium channel blockers should be avoided in patients with LV dysfunction, pulmonary congestion, bradycardia and AV block.

### Table 4: Recommended dosages of ACEI and ARB in STEMI

<table>
<thead>
<tr>
<th>Type</th>
<th>Initiation dose</th>
<th>Target dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Captopril</td>
<td>6.25mg bd –tds</td>
<td>25 - 50mg tds</td>
</tr>
<tr>
<td>Ramipril</td>
<td>2.5mg bd</td>
<td>10mg od</td>
</tr>
<tr>
<td>Enalapril</td>
<td>2.5 - 5mg od</td>
<td>10mg od</td>
</tr>
<tr>
<td>Lisinopril</td>
<td>5mg od</td>
<td>10mg od</td>
</tr>
<tr>
<td>Perindopril</td>
<td>2mg od</td>
<td>4mg od</td>
</tr>
<tr>
<td>Valsartan</td>
<td>80mg od</td>
<td>160mg bd</td>
</tr>
</tbody>
</table>
Table 5: Recommended doses of Nitrates in STEMI

<table>
<thead>
<tr>
<th>Compound</th>
<th>Route</th>
<th>Dosage</th>
<th>Time of Onset</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nitroglycerine, Glyceryl/ trinitrate</td>
<td>Intravenous</td>
<td>5 - 200µg/min*</td>
<td>1 minute</td>
</tr>
<tr>
<td></td>
<td>Sublingual</td>
<td>0.3 - 0.6mg, can repeat up to 3 times at 5 minute intervals</td>
<td>2 minute</td>
</tr>
<tr>
<td></td>
<td>GTN Spray</td>
<td>0.4 - 0.8mg per metered dose, no more than 3 sprays at 5 minute intervals</td>
<td>2 minute</td>
</tr>
<tr>
<td>Isosorbide dinitrate</td>
<td>Transdermal patch</td>
<td>0.2 - 0.8mg over 12 hours on, then 12 hours off</td>
<td>1 - 2 hours</td>
</tr>
<tr>
<td></td>
<td>Intravenous</td>
<td>1.25 - 5mg / hour</td>
<td>1 minute</td>
</tr>
<tr>
<td></td>
<td>Transdermal patch</td>
<td>2.5 - 10mg</td>
<td>3 – 4 minutes</td>
</tr>
<tr>
<td></td>
<td>Oral</td>
<td>10 – 20mg, 2 – 3 times daily</td>
<td>30-60 minutes</td>
</tr>
<tr>
<td>Isosorbide mononitrate</td>
<td>Oral</td>
<td>20 - 30mg, 2 - 3 times daily, up to 120mg in divided doses</td>
<td>30 - 60 minutes</td>
</tr>
</tbody>
</table>

* The dose of IV nitrates should be titrated every 5 - 10 minutes until symptoms and/or ischaemia is relieved and the desired haemodynamic response is obtained

4.3.3.7 Antithrombotics

The antithrombotics that have been studied in STEMI are:
- Unfractionated heparin
- Low molecular weight heparin
- Synthetic pentasaccharide – fondaparinux

Heparin is indicated in patients with:

I,C - post infarct angina
I,C - atrial fibrillation
I,C - mural thrombus
I,C - extensive anterior infarction
I,A - post fibrin-specific fibrinolytic agent 31,32,33
IIIa,B - post non fibrin-specific fibrinolytic agent 31

4.3.3.7.1 Unfractionated heparin (UFH)

Unfractionated heparin is administered as a bolus of 60units/kg (maximum 4000units) followed by an infusion rate of 12units/kg/hour (maximum 1000units/hour) adjusting the dose to maintain the aPTT (activated partial thromboplastin time) of 1.5 to 2.5 times control.
**B) Low molecular weight heparin (LMWH)**

Low molecular weight heparin is given subcutaneously twice a day. LMWH was associated with better clinical outcomes as compared to UFH when given following fibrinolytic therapy in STEMI [56,57]. This benefit was seen with both fibrin-specific [58,59] and non-fibrin specific [57,60] agents, but at an increased risk of bleeding. These studies however, were done prior to the usage of clopidogrel in STEMI.

In patients >75 years of age and with renal impairment (serum creatinine > 200umol/L in women and >250umol/L in men), UFH is preferable to LMWH [59].

<table>
<thead>
<tr>
<th>Heparin</th>
<th>Dosage</th>
<th>Duration of therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Enoxaparin</td>
<td>&lt; 75 yr: 30mg IV bolus, sc 1.0mg/kg bd</td>
<td>Until hospital discharge</td>
</tr>
<tr>
<td></td>
<td>≥ 75 yr: No bolus, sc 0.75mg/kg bd</td>
<td></td>
</tr>
<tr>
<td>UFH</td>
<td>60U/kg bolus (max 4000 U)</td>
<td>≥ 48 hours</td>
</tr>
<tr>
<td></td>
<td>Infusion 12U/kg/h (max 1000 U/h)</td>
<td></td>
</tr>
</tbody>
</table>

Patients given aspirin, clopidogrel, fibrinolytic therapy and LMWH have been found to have a higher rate of patency of the infarct related artery without an increase in the risk of bleeding complications in one substudy [61].

We caution the use of all 4 agents (aspirin, clopidogrel, fibrinolytic therapy and UFH/LMWH) together until the efficacy and safety of the combination is established in future trials.

**C) Synthetic pentasaccharide**

A trial on fondaparinux at a dose of 2.5mg/kg per day, given for 8 days, or the duration of index hospitalization to patients who were given fibrinolytic agents or who were not reperfused, was shown to reduce death or reinfarction at 30 days when compared to UFH. The risk of bleeding was not increased [62].

**4.3.3.8 Glycoprotein IIb/IIIa Receptor Inhibitors**

Glycoprotein IIb/IIIa receptor inhibitors are used mainly in the setting of primary PCI. In primary PCI, the glycoprotein IIb/IIIa receptor inhibitor, abciximab, has been shown to improve patient outcomes [63,64].

**4.3.3.9 Statins**

Recent data has shown that statins started within 24 hours of admission or continued after admission leads to a reduction in major adverse cardiac events [65,66,67].
4.3.3.10 Aldosterone Antagonists

Eplerenone, a selective aldosterone receptor antagonist, when added to β-blockers and ACE-I, has been shown to reduce mortality and hospitalizations when given to patients post myocardial infarction with impaired LV function and mild HF.

4.3.3.11 Others – Magnesium, Lignocaine, Glucose – Insulin Potassium Infusions

Magnesium and Lignocaine are not recommended for routine use in patients with STEMI.

Although earlier studies and meta-analysis seem to indicate that Glucose-Insulin–Potassium infusions are beneficial, more recent studies have not shown reduction in mortality or infarct size.

Recommendations:

- All patients should receive 100 - 300 mg aspirin and 300 mg clopidogrel followed by a maintenance dose of 75-150 mg of aspirin long term and 75 mg of clopidogrel daily for at least a month.
- All patients should be on β blockers if there are no specific contraindications.
- Other medications that have been shown to improve survival if given early are ACE inhibitors (or ARB if ACE intolerant) and statins.
4.4 Complications of STEMI
These are:
- arrhythmias
- left ventricular dysfunction and shock
- mechanical complications
- right ventricular infarction
- others e.g. pericarditis

4.4.1 Arrhythmias
These include:

A) Tachyarrhythmias
- Pulseless ventricular tachyarrhythmias.
  This includes pulseless ventricular tachycardia and ventricular fibrillation. Defibrillate immediately. Early VF occurs within the first 48 hours and is due to electrical instability. Late VF is associated with large infarcts and poor pump function and carries a poor prognosis (refer to algorithm 1)
  [Shockable waves refers to the presence of recognizable organized or disorganized cardiac rhythms on continuous ECG monitoring while non shockable waves refers to the absence of any heart rhythm on ECG monitoring]
  - Stable Ventricular Tachycardia (VT).
    Ventricular tachycardia in the setting of STEMI may arise from either ischaemia or from myocardial scar resulting from the infarct. Treatment of ischaemia may result in the termination of the tachycardia. (refer to algorithm 2).
  - Ventricular Premature Contractions (VPC).
    These are often benign and do not require treatment. Correct underlying ischaemia, hypoxia and electrolyte disturbances.
  - Accelerated Idioventricular Rhythm (AIVR).
    These do not require any treatment. This is a sign suggestive of successful reperfusion.
  - Atrial fibrillation (AF).
    This is more commonly seen in the elderly and is associated with large infarcts and atrial infarcts. It denotes a poorer prognosis\(^7\) and carries an increased risk of thromboembolism. (refer to algorithm 3)

B) Bradyarrhythmias
These are:
- Sinus bradycardia.
  This does not require treatment unless associated with symptoms and/or hypotension.
- Atrio-ventricular Block (AV Block).
  First degree and second degree type 1 (Mobitz 1) do not need treatment. Patients with second degree type 2 (Mobitz 2) and complete AV block may not require treatment if haemodynamically stable.
In patients who are haemodynamically unstable, arrangement must be made for urgent temporary pacing. Atropine may be given in the interim.(maximum 3 mg) (refer to algorithm 4)

In patients with anterior infarcts, second degree and complete AV block carry a worse prognosis. They may require urgent temporary pacing (refer to algorithm 4).

• Asystole/Pulseless Electrical Activity

Asystole/ Pulseless electrical activity can be differentiated from pulseless ventricular tachyarrhythmias by the presence of shockable waves on ECG monitoring. (refer to Algorithm 1)

4.4.2 Left Ventricular Dysfunction and Shock

4.4.2.1 Presentation:

The clinical manifestation of left ventricular dysfunction varies from asymptomatic to cardiogenic shock. An important prognostic indicator is LV function which can be assessed objectively using echocardiography.

A useful clinical classification is the Killip’s Classification\textsuperscript{74,75} (table 7)

<table>
<thead>
<tr>
<th>KILLIP CLASS\textsuperscript{75}</th>
<th>CLINICAL FEATURES</th>
<th>APPROXIMATE PROPORTION OF PATIENTS WITH AMI (%)	extsuperscript{74}</th>
<th>30 day - MORTALITY (%)\textsuperscript{74}</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>No signs of LV failure</td>
<td>71</td>
<td>7</td>
</tr>
<tr>
<td>II</td>
<td>S3 gallop, bibasal crackles</td>
<td>23</td>
<td>20</td>
</tr>
<tr>
<td>III</td>
<td>Acute pulmonary oedema</td>
<td>3.7</td>
<td>39</td>
</tr>
<tr>
<td>IV</td>
<td>Cardiogenic shock</td>
<td>2.4</td>
<td>70</td>
</tr>
</tbody>
</table>

4.4.2.2 Differential diagnoses of LV dysfunction and shock:

Differential diagnoses are:

• Pump failure due to extensive myocardial infarction
• Mechanical complications
• Right ventricular infarction
• Hypovolemia
• Arrhythmias
• Drugs
• Aortic root dissection.
4.4.2.3 Investigations:

Investigations that may be helpful in making the diagnosis and in the management includes:

- Chest radiograph
- ECG
- Echocardiography
- Arterial blood gases
- Pulmonary artery catheter

4.4.2.4 Management:

A) Heart Failure

Acute management includes the following:

- Oxygen therapy
- Diuretics
- Intravenous nitroglycerine
- Intravenous morphine
- Inotropes if hypotensive.

Refer to the Malaysian Clinical Practice Guidelines on Heart Failure 2007 on the management of Acute Cardiogenic Pulmonary Edema.

B) Cardiogenic shock

Cardiogenic shock is defined as a systolic BP of < 90 mmHg associated with signs of tissue hypoperfusion, and central filling pressure (PCWP) is >20mmHg or cardiac index is <1.8L/min/m². This condition is associated with a very high mortality rate.

Emergency PCI may be life-saving and should be considered early. Intra-aortic balloon pump may be useful.

When cardiogenic shock is due to a mechanical defect, urgent surgical repair is indicated. Pre-operative coronary angiography and subsequent coronary artery bypass graft (CABG) surgery in these patients remain an issue of debate. The decision must be individualized.

4.4.3 Mechanical complications

These include the following:

- free wall rupture
- ventricular septal rupture
- papillary muscle rupture

The diagnosis should be suspected in patients with sudden clinical deterioration and suggested by the presence of new murmurs or diminished heart sounds. The diagnosis can be confirmed by echocardiography. In these patients early surgery should be considered.
4.4.4 Right Ventricular Infarction (RVI)
Patients with RVI may have varying clinical presentation, from asymptomatic to cardiogenic shock. Haemodynamically significant RVI complicates approximately 5-10% of all STEMI. It occurs in 30 – 50% of patients with infero-posterior STEMI and is associated with a significantly higher mortality. RVI can also occur in patients with extensive anterior STEMI.

Clinical Diagnosis
The presence of RVI should be sought in all patients with inferior STEMI. The clinical triad of hypotension, clear lung fields and elevated jugular venous pressure in the setting of inferior STEMI is suggestive of RVI.
ST elevation in the right precordial leads (V4R) is the most specific finding in diagnosing RVI. However, this ECG finding may be transient, often resolving within 8-10 hours.

Management
Treatment strategies depend on the severity of peripheral hypoperfusion and the degree of co-existing LV dysfunction. Drugs that reduce the preload, such as nitrates and diuretics should be avoided.
Management includes:
- Optimization of intravenous fluid (saline or colloids)
- Inotropes

Failure to respond to these measures usually indicates concomitant LV dysfunction. These patients require more aggressive management with afterload reducing agents such as nitroprusside and intra-aortic balloon pump.

4.4.5 Others

4.4.5.1 Chest pain post STEMI
Chest pain post STEMI may be due to reinfarction, ischaemia or pericarditis. Non-cardiac causes must also be considered.

A) Reinfarction
Reinfarction occurs in about 3-4% of patients who had undergone fibrinolytic therapy and received aspirin. Reinfarction may be diagnosed by:
- recurrence of ischaemic type chest pain
- recurrence of ST segment elevation of at least 0.1mV in at least contiguous leads and/or
- re-elevation of serum cardiac biomarkers especially CK

Death, severe heart failure and arrhythmias are more common in these patients. They should be considered for rescue PCI.

B) Post-infarct angina
Early recurrent angina, especially after successful reperfusion may occur in up to 20% of patients. The ECG in these patients may show
ST segment changes or pseudo-normalisation of inverted T-waves. These patients should be sent for early coronary angiography with a view to revascularization.

C) Pericarditis

Pericarditis secondary to STEMI may produce pain as early as the first day and as late as 6 weeks\(^{80}\). The pain classically becomes worse on deep inspiration and may be relieved when the patient sits up and leans forward. A pericardial rub may be detected.

Dressler’s syndrome (post MI syndrome) usually occurs 2-10 weeks after STEMI. This is immunologically mediated\(^{81}\). It is treated with aspirin 600mg 3-4 times a day. Steroids and NSAIDS are best avoided in the first 4 weeks of STEMI\(^{82}\).

4.4.5.2 LV Thrombus and Arterial Embolism

LV mural thrombus has been identified in about 20-40% of patients with STEMI. The majority of these occur following anterior or large infarcts. Anticoagulation therapy for a minimum of 3-6 months is advocated in these patients\(^{83}\).

4.4.5.3 Deep Venous Thrombosis (DVT)

In high risk patients (prolonged bed rest, heart failure, unable to mobilize), prophylactic anti-coagulation therapy (subcutaneous heparin 5000 units bd, LMWH – e.g. enoxaparin 40mg od) may be considered until the patient is ambulant.

4.5 Urgent/Emergent CABG surgery:

Urgent/emergent CABG surgery should be considered in the following situations:

- At the time of surgical repair of post-infarction ventricular septal defect (VSD) or mitral valve regurgitation (see section 4.4.3).
- Patients with failed reperfusion whose coronary anatomy and clinical profile are suitable.

These patients should be supported with intra-aortic balloon pump. In general, CABG surgery in this group of patients carries a very high in-hospital mortality rate.
5. RISK STRATIFICATION POST-STEMI

Risk stratification serves to prognosticate and identify appropriate treatment strategies. Risk stratification starts from admission and is a continuing process.

Poor prognostic indicators include:
- older persons ( > 65 years)
- female gender
- previous MI
- anterior MI
- inferior MI with RV involvement
- diabetes mellitus
- ECG changes in multiple leads
- persistent or recurrent ischaemia as manifested by post-infarction angina or ST segment depression at rest
- hypotension
- heart failure
- atrial fibrillation and late (after 48 hours) ventricular arrhythmias
- presumably new LBBB

The above high risk patients should be considered for early coronary angiography. All other patients should be risk stratified early. This helps:
- to identify patients who are likely to reinfarct or develop other complications such as heart failure.
- in the rehabilitation of patients. Low risk patients may be allowed to return to their former activities early.

Risk stratification may be done by assessing:
- Left ventricular function
  - clinical, chest X-ray, echocardiogram, radionuclide studies or cardiac MRI
- Presence of myocardial ischaemia -
  - clinical (recurrent angina)
  - exercise stress testing in asymptomatic patients.
- This may be done from day 5 post-STEMI (sub-maximal stress test with a target heart rate of 70% of maximum predicted heart rate) up to 6 weeks post-STEMI (maximal with a target heart rate of 90% of maximum predicted heart rate for age or symptom limited).
- If the pre-discharge sub-maximal stress test is negative, the patient should be subjected to a maximal or symptom limited stress test within 6 weeks after discharge.
- For those who cannot exercise, consider dobutamine stress echocardiogram, radionuclide perfusion studies or cardiac MRI.
- Presence of malignant ventricular arrhythmias

The presence of angina, an abnormal stress test or late ventricular arrhythmias necessitates early coronary angiography with a view to revascularization.

In patients with poor LV function, myocardial viability studies (dobutamine stress echocardiogram, radionuclide perfusion studies or cardiac magnetic resonance imaging) would help to differentiate
scarred from viable ischaemic myocardium. The latter patients would benefit from revascularization.

Patients with palpitations, near faints and syncope require comprehensive evaluation. This includes:
- serum electrolytes
- resting ECG
- 24 hour ambulatory ECG recording
- evaluation of LV function
- assessment for reversible myocardial ischaemia
- coronary angiography

In these patients, reversible causes such as electrolyte disturbances and ischaemia should be corrected.

The following medications have been shown to reduce the incidence of sudden death:

- β-blockers\(^{84,85}\)
- ACEI\(^{86}\).
- Aldosterone antagonist, eplerenone\(^{68}\).
- Statins\(^{87,88}\).

The following patients should be considered for an ICD:

- Secondary prevention in patients with resuscitated sudden cardiac death\(^{89,90}\).
- Primary prevention in patients with LV dysfunction (EF < 30%). The ICD should be implanted 30 days post STEMI and 3 months post revascularisation\(^{91,92,93}\).

6. DURATION OF HOSPITALIZATION

The duration of hospital stay following STEMI will depend on the extent of myocardial damage, the patient and the presence of co-morbidity.

Asymptomatic patients with uncomplicated STEMI may be discharged after 3-5 days\(^94\) particularly if patient has been successfully reperfused. Patients with significant left ventricular dysfunction or other complications may require a longer hospital stay.
7. SECONDARY PREVENTION

Patients who survive the initial course of STEMI are at increased risk of morbidity and mortality because of reinfarction and the development of other complications. Thus, it is very important that efforts be made to reduce this risk.

These measures include:

7.1 Cessation of smoking

Smoking cessation reduced CHD mortality by 36% as compared to those who continue smoking\(^95\). This lifestyle change confers a risk reduction which is at least as great as other pharmacological interventions.

Trials of nicotine replacement therapy using either transdermal nicotine patch or nicotine chewing gum have proven to greatly increase abstention rates after cessation. Such pharmacological programmes, as well as physician-guided counseling, are cost-effective and should be encouraged\(^96\).

7.2 Diet

Dietary intervention has been shown to reduce cardiac event rates post STEMI\(^97\):

Recommendations include:

- Total calorie intake should be tailored to the desirable body weight
- Wherever possible, substitute saturated and trans fat with polyunsaturated fat.
  Use more high fibre food and whole grains instead of rapidly digested carbohydrates.
- Take more fruits, nuts and vegetables.
- Increased intake of omega 3 – fatty acids (1g daily) is beneficial\(^{96,99}\). Eat fish at least twice a week.

7.3 Regular Exercise

Recommended exercises include brisk walking, jogging, cycling, swimming or other aerobic activity for at least 30 to 60 minutes on most days of the week. It should be supplemented with an increase in daily life-style activities such as walking up stairs whenever possible\(^{100}\).

7.4 Control of Hypertension

After STEMI, prognosis is affected by both the pre existing and the subsequent blood pressure. The higher the pre existing blood pressure, the higher the fatality rate\(^{101}\). The target blood pressure should be < 130/80mmHg. Drugs of choice include β- blockers, ACEIs and Valsartan\(^52\) (if ACEI intolerant).

7.5 Good Glycemic control

Good control of the blood glucose is important\(^{102}\). Target fasting blood glucose should be < 6.0mmol/l and HBA1C < 6.5%
7.6 Antiplatelet Agents

- Aspirin - Aspirin should be prescribed at 75-150mg daily as a maintenance dose unless contraindicated.\(^{9,103}\).

In patients who cannot tolerate aspirin, alternatives include:

- Clopidogrel, 75mg daily \(^{10,11}\)

- Ticlopidine, 250mg bd \(^{11}\)

Dual anti-platelet therapies should be given for at least one month post fibrinolytic therapy for secondary prevention.\(^{10,11}\)

Following primary PCI a longer period of dual antiplatelet therapy is necessary particularly if drug-eluting stents are used.

7.7 β-blockers

In general, β-blockers should be continued indefinitely in all patients if there are no contraindications.\(^{32,43}\).

7.8 ACE Inhibitors and ARB

ACEI should be continued indefinitely in all patients if there are no contraindications.\(^{45}\)

In ACEI intolerant patients, the ARB valsartan may be used.\(^{52}\)

7.9 Lipid-lowering therapy

Statins should be started soon after admission and continued indefinitely. Target LDL cholesterol should be < 2.0mmol/L.\(^{104,105,106}\)

Recent studies indicate that lowering LDL cholesterol even further (< 1.8mmol/L) confers greater benefits.\(^{67,107,108}\).

7.10 Oral Anticoagulant (warfarin)

Long term therapy should be considered for patients with persistent atrial fibrillation.

Warfarin should be given for 3-6 months in patients with LV thrombus.

7.11 Others

- Hormone replacement therapy is not beneficial for secondary prevention.\(^{109,110}\).

- Postmenopausal women who were taking hormone replacement therapy at the time of STEMI should discontinue it.

- Vitamin E and anti-oxidants have no clinical benefit.\(^{111,112}\).

- Garlic, lecithin, Vitamin A and C are not beneficial.
8. SPECIAL GROUPS

8.1 STEMI in the elderly

Patients above the age of 75 years have a much higher in-hospital as well as one year mortality. This may be due to their atypical and delayed presentations, co-morbidities and under utilization of aggressive therapeutic strategies.

Atypical features include silent ischaemia, dyspnoea, syncope and acute confusion. Specific cardiac biomarkers such as CK-MB and troponins should be measured.

Management

A) Primary PCI

This is the preferred reperfusion strategy if facilities are available.

B) Fibrinolytic

There is an increased risk of intracranial haemorrhage in the elderly with the use of fibrin specific fibrinolytic agents as reperfusion strategy. Therefore streptokinase is the preferred agent. Despite this, the absolute benefits of fibrinolytic therapy in this age group are almost twice that of younger patients with substantial decrease in mortality.

C) Concomitant Therapy

These patients have similar or greater benefits with the use of aspirin, β-blockers, ACEI and statins as younger patients.

D) Risk Stratification

This has to be individualized taking into consideration the biological age rather than the chronological age of the patient. The presence of on-going ischaemia, symptomatic malignant arrhythmias and a depressed LV function are bad prognostic indicators and would generally necessitate a more aggressive approach. Both PCI and CABG surgery, when indicated, can be carried out in the elderly with acceptable morbidity and mortality by experienced operators. The risks are however higher than in younger patients.

8.2 STEMI in Diabetics

Diabetic patients have higher in-hospital mortality (about 1.5 to 2 times) than non-diabetics following an STEMI. The prognosis is worse in diabetic women. There is a higher frequency of atypical and silent presentations in these patients.

Management

Diabetic patients should be treated in a similar manner as non-diabetics.

8.3 STEMI in Women

The perception that women are “protected” against ischaemic heart disease leads to under recognition of this condition. Atypical and delayed presentation, late diagnosis, older age at presentation and high prevalence of co-morbidities result in a higher mortality in women.
Women are thus high risk patients and should be treated aggressively.

9. CARDIAC REHABILITATION IN STEMI

All patients’ post STEMI (including those post PCI or CABG surgery) should undergo comprehensive cardiac rehabilitation. This programme aims at improving the long-term prognosis and optimizing the physical, psychological and social well-being of the patient. It comprises prescribed exercise training and education, counseling, risk factor modification and behavioral interventions.

Cardiac rehabilitation should start in the cardiac care unit, continue to out-patient settings and extend to community care. It is a proven effective intervention and every effort must be made to ensure minimal dropouts so as to maximize beneficial effects of the programme.

10. FUTURE DEVELOPMENTS

There is ongoing research in various aspects of myocardial preservation in the acute phase of STEMI utilizing stem cells, myocytes, growth factors and other agents.

11. CHECK LISTS FOR FOLLOW-UP VISIT

The following should be assessed at each follow-up visit:

- Delineate the presence or absence of cardiac symptoms and determine the functional class of the patients.
- Evaluate patients’ psychosocial (anxiety & depression) status and the social integration and support network.
- Review pre discharge risk assessment and planned workup
  - Evaluation for further cardiac ischaemia
  - LV function
- Re-evaluate the list of all current medications and optimize their doses.
- Actively review the following issues with the patient and family members
  - Principles of secondary prevention
  - CPR training
  - A plan for appropriate recognition and response to a potential acute cardiac event
- Treat to target.
  - Blood pressure: <130/80 mmHg
  - Lipids: LDL < 2.6mmol/L
  - Diabetic control: Fasting blood glucose < 6.0mmol/L
    HbA1C < 6.5%
  - Achieve and maintain ideal body weight and waist circumference
10. SUMMARY

- CHD is an important cause of death in Malaysia.
- The diagnosis of STEMI depends on the presence of ischaemic type chest pain and ST elevation in the resting ECG or new onset LBBB.
- **TIME LOST IS MYOCARDIUM LOST**, thus early diagnosis and treatment is important.
- Early management of STEMI involves pain relief, stabilization of haemodynamics and assessment for reperfusion.
- The occluded infarct-related artery should be opened as soon as possible either by primary PCI or fibrinolytic therapy. Failed fibrinolysis necessitates rescue PCI.
- Concomitant pharmacotherapy includes aspirin, clopidogrel, β-blockers, ACEI/ARB and statins.
- Complications of STEMI include arrhythmias, LV dysfunction and shock.
- Urgent/emergent CABG surgery should be considered in patients with mechanical complications or failed reperfusion.
- High-risk patients should have early coronary angiography with view to revascularization. The others should be risk stratified according to the presence or absence of ischaemia, arrhythmias and LV function.
- Secondary prevention is important and includes the use of aspirin, β-blockers, ACEI/ARB and statins.
- All patients should be encouraged to undergo cardiac rehabilitation.
Flow chart 1: MANAGEMENT OF PATIENTS PRESENTING WITH STEMI

Chest Pain

ECG
Cardiac Biomarkers

STEMI

Sublingual GTN
Continuous ECG monitoring
Oxygen
Aspirin
Clopidogrel
Analgesia

Concomitant initial management includes:

Assessment for reperfusion:

Onset of symptoms:

< 3 hrs

3-12 hours

> 12 hours

Preferred option:

Primary PCI**
or Fibrinolytic

Primary PCI***

Medical Therapy ± Anti thrombotics

Second option:

Fibrinolytics

Primary PCI*

Concomitant Therapy:

Anti Thrombotics*

β - Blockers
ACEI/ARB
Statins
Nitrates*
Calcium antagonists *

* when clinically indicated

** Preferred option in:
- high risk patients,
- presence of contraindications to fibrinolytic therapy and/or
- PCI time delay [(door to balloon time) – (door to needle time)] of
  less than 60 minutes

*** If door to balloon time is within 90 minutes
Algorithm 1: PULSELESS ARRHYTHMIAS

**PULSELESS ARREST**
- BLS Algorithm: Call for help, give CPR
- Give oxygen when available
- Attach monitor/defibrillator when available

2. Check rhythm: Shockable rhythm?
   - Shockable
   - Not Shockable

3. **VF/VT**
   - Give 1 shock
     - Manual biphasic: device specific (typically 120 to 200 J)
     - Note: if unknown, use 200 J
     - AED: device specific
     - Monophasic: 360 J
     - Resume CPR immediately

4. Give 5 cycles of CPR*  
   - Check rhythm: Shockable rhythm?
   - No

5. Continue CPR while defibrillator is charging
   - Give 1 shock
     - Manual biphasic: device specific (same as first shock or higher dose)
     - Note: if unknown, use 200 J

9. **Asystole/PEA**
   - Resume CPR immediately for 5 cycles
     - When IV/IO available, give vasopressor
       - Epinephrine 1 mg IV/IO
       - Repeat every 3 to 5 min or
         - May give 1 dose of vasopressin 40 U IV/IO to replace first or second dose of epinephrine
       - Consider atropine 1 mg IV/IO for asystole or slow PEA rate
       - Repeat every 3 to 5 min (up to 3 doses)

10. Give 5 cycles of CPR*
   - Check rhythm: Shockable rhythm?

11. **Continue page 36**
Resume CPR immediately after the shock
When IV/IO available, give vasopressor
during CPR (before or after the shock)
- Epinephrine 1 mg IV/IO
  Repeat every 3 to 5 min
  or
- May give 1 dose of vasopressin 40 U IV/IO
to replace first or second dose of epinephrine

8. Continue CPR while defibrillator is charging
Give 1 shock
- Manual biphasic: device specific
  (same as first shock or higher dose)
  Note: if unknown, use 200 J
- AED: device specific
- Monophasic: 360 J
Resume CPR immediately after the shock
Consider antiarrhythmics: give during CPR
(before or after the shock)
  - amiodarone (300 mg IV/OI once, then
    consider additional 150 mg IV/OI once)
  - lidocaine (1 to 1.5 mg/kg first dose then
    0.5 to 0.75 mg/kg IV/OI, maximum 3 doses or
    3 mg/kg)
Consider magnesium, loading dose
  1 to 2 g IV/OI for torsades de pointes
After 5 cycles of CPR, go to Box 5 above

12. If asystole, go to Box 10
- If electrical activity,
  check pulse. If no pulse, go to Box 10
- If pulse present, begin postresuscitation care

13. During CPR
- Push hard and fast (100/min)
- Ensure full chest recoil
- Minimize interruptions in chest compressions
  - One cycle of CPR: 30 compressions
    then 2 breaths; 5 cycles ≈ 2 min
  - Avoid hyperventilation
  - Secure airway and confirm placement
  - After an advanced airway is placed,
    rescuers no longer deliver “cycles”
    of CPR. Give continuous chest com-
    pressions without pauses for breaths
    Give 8 to 10 breaths/minute. Check
    rhythm every 2 minutes.
  - Rotate compressors every
    2 minutes with rhythm checks
  - Search for and treat possible
    contributing factors:
    - Hypovolemia
    - Hypoxia
    - Hydrogen ion (acidosis)
    - Hypo-/hyperkalemia
    - Hypoglycemia
    - Hypothermia
    - Toxins
    - Tamponade, cardiac
    - Tension pneumothorax
    - Thrombosis (coronary or
      pulmonary)
    - Trauma

(Reproduced with permission from 2005
American Heart
Association Guidelines
for Cardiopulmonary
Resuscitation and
Emergency
Cardiovascular Care,
Circulation. 2005;112:
IV-58 – IV-66.)
**Algorithm 2: Stable Ventricular Tachycardia**

- Assess and support ABCs
  - Give Oxygen
  - Monitor ECG

**Hemodynamically Stable**

**Monomorphic VT**
- i/v Amiodarone 150 mg over 10 mins
- Repeat as needed up to 2.2 gm/24 hrs

- Successful Cardioversion
  - Oral Amiodarone
- Failed Cardioversion
  - Electrical Cardioversion

**Polymorphic VT**
- Check electrolytes and correct accordingly
  - Stop all arrhythmic drugs (if any)
  - i/v Magnesium
  - Overdrive Pacing
  - i/v Isoprenaline

**Algorithm 3: Atrial Fibrillation**

- Search and treat identifiable underlying causes

**Hemodynamic Stability**

**Stable**
- Normal LV function
  - Rate or rhythm control
    - Rate control
    - Beta blockers
    - Calcium blockers
  - Rhythm control
    - i/v amiodarone followed by oral amiodarone
  - Anticoagulation
    - If persistent after 48 hours or if cardioversion is contemplated

**Impaired LV function**
- Rhythm control preferably
  - i/v amiodarone followed by oral amiodarone
  - Anticoagulation
  - Cardioversion

**Unstable**
- Pre-medicate for cardioversion
  - Anticoagulation

**Electrical Cardioversion**

- Successful CV
  - Oral Amiodarone
- Unsuccessful CV
  - i/v Amiodarone
Algorithm 4: Bradycardia

Bradycardia
- Slow (absolute bradycardia ≤ 60bpm)
  or
- Relatively slow (rate less than expected relative to the underlying condition/cause)

- Assess ABCD
- Vital signs monitoring
- Search for underlying causes e.g. electrolytes, drugs
  And treat accordingly

Serious symptoms due to bradycardia?

No
Type II second degree AV block or Third degree AV block?

No
Haemodynamically stable

Observe

Yes
Transvenous pacing

Yes
Intervention sequence
- Atropine 0.5 to 1mg
- Dopamine 5 to 20mcg/kg/min
- Epinephrine 2 to 20mcg/kg/min
- Transcutaneous pacing if available
# APPENDIX 1: GRADES OF RECOMMENDATION AND LEVEL OF EVIDENCE FOR ACUTE THERAPY OF STEMI

<table>
<thead>
<tr>
<th>RECOMMENDATION</th>
<th>Grade of Recommendation</th>
<th>Level of Evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Reperfusion Therapy</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Primary PCI: Strategy of choice if:</td>
<td>I</td>
<td>A</td>
</tr>
<tr>
<td>• Door to balloon time &lt; 90 min</td>
<td>I</td>
<td>A</td>
</tr>
<tr>
<td>• There are contraindications to fibrinolysis</td>
<td>I</td>
<td>A</td>
</tr>
<tr>
<td>• High Risk patients</td>
<td>I</td>
<td>A</td>
</tr>
<tr>
<td>*Fibrinolytic Therapy: Strategy of choice if:</td>
<td>I</td>
<td>A</td>
</tr>
<tr>
<td>• If the door to balloon time is &gt; 90 min</td>
<td>I</td>
<td>A</td>
</tr>
<tr>
<td>• No contraindications to fibrinolysis</td>
<td>I</td>
<td>A</td>
</tr>
<tr>
<td><strong>Concomitant Pharmacotherapy</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aspirin: loading dose of 300 mg followed by maintenance dose of 75 mg – 150 mg daily</td>
<td>I</td>
<td>A</td>
</tr>
<tr>
<td>Clopidogrel: Loading dose of 300 mg followed by maintenance dose of 75 mg daily(for at least 1 month)</td>
<td>I</td>
<td>A</td>
</tr>
<tr>
<td>Anti-thrombotic (heparin) to be given to patients:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Who received fibrin selective lytic agents</td>
<td>I</td>
<td>A</td>
</tr>
<tr>
<td>• With Atrial Fibrillation</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>• With Mural Thrombus</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>• With Large Infarcts</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>• Routine administration to patients following fibrinolysis (not on dual antiplatelet therapy)</td>
<td>II a</td>
<td>B</td>
</tr>
<tr>
<td>β-blockers: For all patients if no contraindications.</td>
<td>I</td>
<td>A</td>
</tr>
<tr>
<td>ACE Inhibitors: For all patients with no contraindications</td>
<td>I</td>
<td>A</td>
</tr>
<tr>
<td>Statins: For all patients if no contraindications</td>
<td>I</td>
<td>A</td>
</tr>
<tr>
<td>Calcium Channel Blockers: Not for routine use</td>
<td>III</td>
<td>A</td>
</tr>
<tr>
<td>Nitrates and Magnesium: Not for routine use</td>
<td>III</td>
<td>A</td>
</tr>
</tbody>
</table>

*please refer to flow chart 1 for details*
<table>
<thead>
<tr>
<th>STRATEGY</th>
<th>GRADE OF RECOMMENDATION</th>
<th>LEVEL OF EVIDENCE</th>
<th>COMMENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoking Cessation</td>
<td>I</td>
<td>C</td>
<td>At least 30-60 min most days of the week</td>
</tr>
<tr>
<td>Exercise</td>
<td>I</td>
<td>C</td>
<td></td>
</tr>
<tr>
<td>CONCOMITANT PHARMACOTHERAPY</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aspirin</td>
<td>I</td>
<td>A</td>
<td>Maintenance dose: 75-150 mg daily</td>
</tr>
<tr>
<td>Clopidogrel</td>
<td>I</td>
<td>A</td>
<td>Maintenance dose 75 mg daily to be given for 1 month following fibrinolytic therapy and for longer periods post primary PCI</td>
</tr>
<tr>
<td>Anti-coagulants (warfarin)</td>
<td>I</td>
<td>C</td>
<td>Long term therapy for patients in AF; 3-6 months for pts with mural thrombus</td>
</tr>
<tr>
<td>β-Blockers</td>
<td>I</td>
<td>A</td>
<td>Consider long term therapy for all patients if no contraindications</td>
</tr>
<tr>
<td>ACEI</td>
<td>I</td>
<td>A</td>
<td>Started on first day and continued long term for all pts if no contraindications</td>
</tr>
<tr>
<td>ARB</td>
<td>I</td>
<td>B</td>
<td>For ACEI intolerant pts, consider Valsartan</td>
</tr>
<tr>
<td>Statins</td>
<td>I</td>
<td>A</td>
<td>Aim for an LDL-C &lt;2.6mmol/l (the lower the better)</td>
</tr>
</tbody>
</table>
**Appendix 3: Pharmacokinetics of Phosphodiesterase 5 inhibitors**

<table>
<thead>
<tr>
<th></th>
<th>Sildenafil (Viagra)</th>
<th>Vardenafil (Levitra)</th>
<th>Tadalafil (Cialis)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset of action</td>
<td>27 min</td>
<td>26 min</td>
<td>45 min</td>
</tr>
<tr>
<td>Duration of action</td>
<td>4 hours</td>
<td>4-5 hours</td>
<td>Up to 36 hours</td>
</tr>
<tr>
<td>Time to peak action</td>
<td>60 min</td>
<td>60 min</td>
<td>30 min-6 hours</td>
</tr>
</tbody>
</table>
REFERENCES:

1. Number of discharges and deaths due to circulatory system by age group and sex. Information and Documentation System Unit. Annual Report. Ministry of Health Malaysia 2004


8. Guidelines for clinical use of cardiac imaging. Report of the American College of Cardiology/American Heart Association Task Force on Assessment of Diagnostic and Therapeutic Cardiovascular Procedures (Committee on Radionuleide Imaging), developed in collaboration with the American College of Nuclear Cardiology. J Am Coll Cardiol 1995; 25 : 521-47.


16. Aversano T, Aversano LT, Passamani E et al for the Atlantic Cardiovascular Patient Outcomes Research Team (C-POR). Thrombolytic therapy vs primary percutaneous coronary intervention for myocardial infarction in patients presenting to hospitals without onsite cardiac surgery: a randomized controlled trial. JAMA 2002; 287: 1943-51


84. Nutall SL, Toescu V, Kendall MJ. Beta blockers have a key role in reducing morbidity and mortality after infarction. BMJ 2000;320:581


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