Management of Patients with ST-Elevation Myocardial Infarction (STEMI)

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In-hospital mortality (STEMI)

In-hospital mortality dramatically decreased under the last 30 years:

- CCU + defibrillator: 30
- Thrombolysis: 15
- Primary PCI: 10
- 90-s: 7

60-s 70-s 80-s 90-s
Figure 13.1 Terminology used in acute coronary syndromes. Reproduced with permission from Hamm et al. Lancet 2001; 358: 1533–1538 [151]
Prizmetal's or 'variant' angina

Angina associated with ST elevation may be due to coronary artery vasospasm.
This may occur with or without a fixed coronary abnormality and may be indistinguishable from an acute MI until changes resolve rapidly with GTN as pain is relieved.
Options for Transport of Patients With STEMI and Initial Reperfusion Treatment

**EMS Transport**
- Onset of symptoms of STEMI
- Encourage 12-lead ECGs.
- Consider prehos. fibrinolytic if capable and EMS-to-needle within 30 min.

**Hospital fibrinolysis:**
- Door-to-Needle within 30 min.

**Not PCI capable**

**EMS Triage Plan**

**PCI capable**

**Inter-Hospital Transfer**

**GOALS**
- Patient
  - Dispatch 1 min.
  - EMS 8 min.
  - Prehospital fibrinolysis: EMS-to-needle within 30 min.
- EMS Transport
  - EMS 5 min.
  - EMS on-scene
    - EMS-to-needle within 30 min.
  - EMSTransport
    - EMS-to-balloon within 90 min.
    - Patient self-transport
    - Hospital door-to-balloon within 90 min.

**Golden Hour = first 60 min.**

**Total ischemic time: within 120 min.**

Antman EM, et al. J Am Coll Cardiol 2008. Published ahead of print on December 10, 2007. Available at http://content.onlinejacc.org/cgi/content/full/j.jacc.2007.10.001. Figure 1.
Transmission of Patient Data

Cellular Site

EMS

ED
Cath Lab

HOSPITAL
Important
Evolution of Heart Attack

<table>
<thead>
<tr>
<th>Time after onset</th>
<th>Onset</th>
<th>&lt;20-40 min.</th>
<th>30 min.</th>
<th>1 hour</th>
<th>2 hours</th>
<th>6 hours</th>
<th>24 hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extent of necrosis</td>
<td>0%</td>
<td>0%</td>
<td>10%</td>
<td>30%</td>
<td>50%</td>
<td>90%</td>
<td>100%</td>
</tr>
</tbody>
</table>
The diagnosis of acute MI requires 2 out of the following 3 features:

- A history of cardiac-type ischaemic chest pain.
- Evolutionary changes on serial ECGs.
- A rise in serum cardiac markers.
MI diagnosis

Note that 50-60% of pts will not have a diagnostic ECG on arrival and up to 17% will have an entirely normal initial ECG. Late presentation does not improve diagnostic accuracy of the ECG.
History

The classic presentation is of sudden onset, severe, constant central chest, which radiates to the arms, neck, or jaw. This may be similar to previous angina pectoris, but is much more severe and unrelieved by GTN. The pain is usually accompanied by one or more associated symptoms: sweating, nausea, vomiting, breathlessness.

Atypical presentation is common.
Have a high level of suspicion. Many pts describe atypical pain, some attributing it to indigestion (be wary of new onset 'dyspeptic' pain). Up to a third of pts with acute MI do not report any chest pain. These pts tend to be older, are more likely to be female, have a history of diabetes or heart failure, and have a higher mortality.
These patients may present with:

- LVF.
- Collapse or syncope (often with associated injuries eg head injury).
- Confusion.
- Stroke.
- An incidental ECG finding at a later date.
Examination

Examination and initial resuscitation (maintain SpO2 in normal range. IV cannula, analgesia) go hand in hand. The patient may be pale, sweaty, and distressed. Examination is usually normal unless complications have supervened (e.g. arrhythmias, LVF). Direct initial examination towards searching for these complications and excluding alternative diagnoses:
Examination

- Check pulse. BP and monitor trace (?arrhythmia or cardiogenic shock).
- Listen to the heart (murmurs or 3rd heart sound).
- Listen to the lung fields (?LVF, pneumonia, ptx).
- Check peripheral pulses (?aortic dissection).
- Check legs for evidence of deep vein thrombosis (?PE).
- Palpate for abdominal tenderness or masses (?cholecystitis, pancreatitis, perforated peptic ulcer, ruptured aortic aneurysm).
Investigations

- If the diagnosis of ST segment elevation MI within the first few hours is based upon history and ECG changes (serum cardiac markers may take several hours to rise—see below).

- Record an ECG as soon as possible, ideally within a few minutes of arrival at hospital. Sometimes pts arrive at hospital with ECGs of diagnostic quality already recorded by paramedics. If the initial ECG is normal, but symptoms are suspicious, repeat the ECG every 15min and re-evaluate,
Investigations

- Request old notes (these may contain previous ECGs for comparison).
- Ensure continuous cardiac monitoring and pulse oximetry.
- Monitor BP and respiratory rate.
- Obtain venous access and send blood for cardiac markers. U&E, glucose, FBC, lipids.
- Obtain a CXR if there is suspicion of LVF or aortic dissection.
Cardiac markers

Troponins are now universally used. Troponin T (cTnT) and Troponin I (cTnl) are proteins virtually exclusive to cardiac myocytes. They are highly specific and sensitive, but are only maximally accurate after 12hr. Troponin T and I cannot be used to rule out MI in the first few hours. In addition, cardiac cells may release troponin into the blood when cardiac muscle is damaged by pericarditis, pulmonary embolism with a large clot burden, or sepsis. Renal failure reduces excretion of troponin.
Hyperacute changes

Frequently ignored, although often subtle, some or all of the following may be observed within minutes of infarction:

• **↑ Ventricular activation time**, since the infarcting myocardium is slower to conduct electrical impulses. The interval between the start of the QRS and apex of the R wave may be prolonged >0.045 sec.

• **↑ Height of R wave** may be seen initially in inferior leads in inferior MI

• Upward-sloping ST segment—having lost normal upward concavity. II ST segment straightens, then slopes upwards, before becoming elevated.

• Tall, widened T waves.
Evolving acute changes

In isolation, none of these changes are specific to MI. In combination, with an appropriate history, they can diagnose MI:

- **ST elevation**: the most important ECG change. ST segments become concave down and are significant if elevated >1mm in 2 limb leads, or >2mm in 2 adjacent chest leads.

- **Reciprocal ST depression** may occur on the 'opposite side' of the heart.

- **Pathological Q waves** reflect electrically inert necrotic myocardium. ECG leads over a large transmural infarct, the deep QS waves. Leads directed towards the periphery of a large infarct or over a smaller infarct may show a QR complex or a loss of R amplitude.

- **T wave inversion**: typically deeply inverted, symmetrical and pointed.
Evolving acute changes

Conduction problems may develop
LBBB in a patient with acute cardiac chest pain makes
interpretation of the ECG very difficult. LBBB does not have to be
new to be significant. Do not delay intervention in pall with a good
clinical history of MI in order to obtain old ECGs.

Sgarbossa criteria for diagnosing ACS in the presence of LBBB
• ST segment elevation >1 mm in leads with positive QRS
  complexes.
• ST segment depression in leads V1, V2, or V3.
• ST segment elevation >5mm in lead with negative QRS complexes.
  If all 3 are present, MI is likely.
Chronic changes

In the months following an MI, ECG changes resolve to a variable extent. ST segments become isoelectric, unless a ventricular aneurysm develops, T waves gradually become positive again. Q waves usually remain, indicating MI at some time in the past.
Evolution of AMI

• Hyperacute
  - Early change suggestive of AMI
  - Tall & Peaked
  - May precede clinical symptoms
  - Only seen in leads looking at infarcting area
  - Not used as a diagnostic finding
Evolution of AMI

• **Acute**
  - ST segment elevation
  - Implies myocardial injury occurring
  - Elevated ST segment presumed acute rather than old
Evolution of AMI

• Acute
  - ST segment Elevated
  - Q wave at least 40 ms wide = pathologic
  - Q wave associated with some cellular necrosis
Evolution of AMI

• **Age Undetermined**
  - Wide (pathologic) Q wave
  - No ST segment elevation
  - Old or “age undetermined”
  
  MI
MI usually affects the left ventricle (LV), occasionally the right ventricle (RV), but virtually never the atria. The part of myocardium affected is implied by which leads show changes

<table>
<thead>
<tr>
<th>ECG leads</th>
<th>Location of MI</th>
</tr>
</thead>
<tbody>
<tr>
<td>V 1-3</td>
<td>Anteroseptal</td>
</tr>
<tr>
<td>V 5-6, AVL</td>
<td>Anterolateral</td>
</tr>
<tr>
<td>V2-4</td>
<td>Anterior</td>
</tr>
<tr>
<td>V1-6</td>
<td>Extensive anterior</td>
</tr>
<tr>
<td>I.II. aVL, V6</td>
<td>Lateral</td>
</tr>
<tr>
<td>II. III. aVF</td>
<td>Inferior</td>
</tr>
<tr>
<td>V1, V4R</td>
<td>Right ventricle</td>
</tr>
</tbody>
</table>
Localization

I: Inferior
II: Septal
III: Anterior
AVL: Left anterior descending artery
AVF: Left circumflex artery

- Inferior: II, III, AVF
- Septal: V1, V2
- Anterior: V3, V4
- Lateral: I, AVL, V5, V6
Localization

Which coronary arteries are most likely associated with each group of contiguous leads?

<table>
<thead>
<tr>
<th>I Lateral</th>
<th>aVR</th>
<th>V1 Septal</th>
<th>V4 Anterior</th>
</tr>
</thead>
<tbody>
<tr>
<td>II Inferior</td>
<td>aVL Lateral</td>
<td>V2 Septal</td>
<td>V5 Lateral</td>
</tr>
<tr>
<td>III Inferior</td>
<td>aVF Inferior</td>
<td>V3 Anterior</td>
<td>V6 Lateral</td>
</tr>
</tbody>
</table>
Localization: Left Coronary Artery

- Right Coronary Artery
- Left Main
- Left Circumflex
- Lateral Wall
- Anterior Wall of Left Ventricle
- Right Ventricle
- Septal Wall
- Anterior Descending Artery
Localization Practice ECG
Localization Practice ECG
Localization Practice ECG

HR: 107
PR Int: 132
P-QRS-T Axes: 75 0 71
QRS Dur: 88
QT/QTc: 336/399

8:45:08 PM 7/1/97
12-Lead #1

x1.0 0.05-150Hz 25mm/sec
11 11 2.2 LPK11000002671
Localization: Right Coronary Artery

- Left Coronary Artery
- Lateral Wall
- Left Ventricle
- Right Coronary Artery
- Posterior Descending Artery
- Posterior Wall
- Inferior Wall of left ventricle
Localization Practice ECG
Localization Practice ECG

Note: “R” designation manually placed on this ECG for teaching purposes
Localization Practice ECG
Localization Practice ECG
Localization Summary

• **Left Coronary Artery**
  - Septal
  - Anterior
  - Lateral
  - Possibly Inferior

• **Right Coronary Artery**
  - Inferior
  - Right Ventricular Infarct
  - Posterior
Proper Early Management

Does your institution achieve these goals?

Thrombolysis 30 minutes

Primary PCI 90 minutes

Time is the major predictor in patient outcomes
Type of reperfusion method (PCI vs. fibrinolysis) less imperative
Reperfusion Strategies

Several factors to consider:

- Time from the onset of symptoms
- Risk from the STEMI
- Risk of bleeding
- Time required for transport to cath lab
ED Evaluation of Patients With STEMI

Brief Physical Examination in the ED

1. Airway, Breathing, Circulation (ABC)
2. Vital signs, general observation
3. Presence/absence of jugular venous distension
4. Pulmonary auscultation for rales
5. Cardiac auscultation for murmurs and gallops
6. Presence or absence of stroke
7. Presence or absence of pulses (periferal- ao.-dissection??)
8. Presence or absence of systemic hypoperfusion (cool, clammy, pale, ashen)
9. Abdominal tenderness or masses (cholecystitis, pancreatitis, ulcer, perforation, ruptured ao.aneurysm)
Laboratory Examinations

Lab. examinations should be performed as part of the management of STEMI pts, **but should not delay the implementation of reperfusion therapy**.

- Serum biomarkers for cardiac damage
- Complete blood count (CBC) with platelets
- International normalized ratio (INR)
- Activated partial thromboplastin time (aPTT)
- Electrolytes and magnesium
- Blood urea nitrogen (BUN)
- Creatinine
- Glucose
- Complete lipid profile
Pts with STEMI should have a portable chest X-ray, but this should not delay implementation of reperfusion th. (unless a potential contraindication is suspected, such as aortic dissection). - Class I

**Imaging studies** such as a high quality portable chest X-ray, transthoracic and/or transesophageal echocardiography, and a contrast chest CT scan or an MRI scan should be used for differentiating STEMI from aortic dissection in pts for whom this distinction is initially unclear. - Class I
Reperfusion Options for STEMI Patients

**Step One: Assess Time and Risk.**

- **Time Since Symptom Onset**
- **Risk of STEMI**
- **Risk of Fibrinolysis**
- **Time Required for Transport to a Skilled PCI Lab**
If presentation is <3 hours and there is no delay to an invasive strategy, there is no preference for either strategy.

**Fibrinolysis generally preferred**
- Early presentation (<3 hours from symptom onset and delay to invasive strategy)
- Invasive strategy not an option
  - Cath lab occupied or not available
  - Vascular access difficulties
  - No access to skilled PCI lab
- Delay to invasive strategy
  - Prolonged transport
  - Door-to-balloon more than 90 minutes
  - >1 hour vs fibrinolysis (fibrin-specific agent) now
Reperfusion Options for STEMI Patients

Step 2: Select Reperfusion Treatment.

If presentation is < 3 hours and there is no delay to an invasive strategy, there is no preference for either strategy.

Invasive strategy generally preferred

- Skilled PCI lab available with surgical backup
  - Door-to-balloon < 90 minutes

- High Risk from STEMI
  - Cardiogenic shock, Killip class ≥ 3

- Contraindications to fibrinolysis, including increased risk of bleeding and ICH

- Late presentation
  - > 3 hours from symptom onset

- Diagnosis of STEMI is in doubt
Algorithm for management of pts with suspected AMI in the ED

Chest pain pattern suggesting acute coronary syndrome

Goal = 10 minutes
- Triage for rapid care
- Aspirin 160-325 mg chewed
- SL nitroglycerin 0.4 mg every 5 minutes for three doses
- Morphine sulfate 2 to 4 mg IV initially, then 2 to 8 mg IV every 5 to 15 minutes
- Establish intravenous access
- Obtain blood for initial laboratory work including serum biomarkers*,
- Institute continuous ECG monitoring
- Initiate supplemental oxygen therapy
- Obtain focused history and examination

Initial 12 lead ECG; if not diagnostic, repeat at 5 to 10 minute intervals

ST elevation or new or presumably new LBBB
- Beta blocker if not contraindicated
- IV nitroglycerin if persistent chest pain
- Intravenous heparin
- Most use GP IIb/IIIa inhibitor if primary PCI
- Clopidogrel

Primary PCI, if available, with goal less than 90 minutes
OR thrombolysis with goal of 30 minutes

Strong suspicion for ischemia, but no persistent ST elevation
- Enoxaparin or unfractionated heparin
- Beta blocker if not contraindicated
- IV nitroglycerin if persistent chest pain
- Clopidogrel but, if PCI is planned, some wait until coronary angiography to see if CABG is required

Normal or nondiagnostic ECG and normal cardiac enzymes
- Continue evaluation and treatment in ED or monitored bed
- Repeat ECG and cardiac enzymes at 6 to 12 hours

Evidence of ischemia/infarction
- Perform stress test
- Yes
- No
- Treat for non-ST elevation acute coronary syndrome

Look for high-risk features:
- ST depression
- Elevated cardiac enzymes
- Persistent chest pain
- Hemodynamic instability
- TIMI risk score ≥3

GP IIb/IIIa inhibitor if patient is to undergo PCI or has high-risk features

Catheterization if high-risk features; catheterization or medical management in low-risk patients
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    - Perform stress test
  - Yes
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Complications of Acute MI

- Extension / Ischemia
- Arrhythmia
- Pericarditis
- Expansion / Aneurysm
- Acute MI
- RV Infarct
- Mechanical
- Heart Failure
- Mural Thrombus
Emergency Management of Complicated STEMI

**Clinical signs:** Shock, hypoperfusion, congestive heart failure, acute pulmonary edema

**Most likely major underlying disturbance?**

- **Acute Pulmonary Edema**
  - Administer
    - Furosemide IV 0.5 to 1.0 mg/kg
    - Morphine IV 2 to 4 mg
    - Oxygen/intubation as needed
    - Nitroglycerin SL, then 10 to 20 mcg/min IV if SBP greater than 100 mm Hg
    - Dopamine 5 to 15 mcg/kg per minute IV if SBP 70 to 100 mm Hg and signs/symptoms of shock present
    - Dobutamine 2 to 20 mcg/kg per minute IV if SBP 70 to 100 mm Hg and no signs/symptoms of shock

- **Hypovolemia**
  - Administer
    - Fluids
    - Blood transfusions
    - Cause-specific interventions
    - Consider vasopressors

- **Low Output - Cardiogenic Shock**
  - Check Blood Pressure
  - Systolic BP Greater than 100 mm Hg
    - Nitroglycerin 10 to 20 mcg/min IV
  - Systolic BP 70 to 100 mm Hg
    - No signs/symptoms of shock
    - Dobutamine 2 to 20 mcg/kg per minute IV if SBP 70 to 100 mm Hg
  - Systolic BP 70 to 100 mm Hg
    - Signs/symptoms of shock
    - Dopamine 5 to 15 mcg/kg per minute IV
  - Systolic BP less than 70 mm Hg
    - Signs/symptoms of shock
    - Norepinephrine 0.5 to 30 mcg/min IV

- **Arrhythmia**
  - Bradycardia
  - Tachycardia
  - See Section 7.7 in the ACC/AHA Guidelines for Patients With ST-Elevation Myocardial Infarction

**First line of action**

- Check Blood Pressure
- Systolic BP Greater than 100 mm Hg and not less than 30 mm Hg below baseline
- ACE Inhibitors
  - Short-acting agent such as captopril (1 to 6.25 mg)

**Second line of action**

- Further diagnostic/therapeutic considerations (should be considered in nonhypovolemic shock)
  - **Diagnostic**
    - Pulmonary artery catheter
    - Echocardiography
    - Angiography for MI/ischemia
    - Additional diagnostic studies
  - **Therapeutic**
    - Intra-aortic balloon pump
    - Reperfusion/revascularization

**Circulation 2000;102(suppl I):I-172-I-216.**
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  - Check Blood Pressure

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  - Bradycardia
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- Systolic BP Greater than 100 mm Hg
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- Systolic BP 70 to 100 mm Hg
  - NO signs/symptoms of shock
  - Dobutamine 2 to 20 mcg/kg per minute IV

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- **Dobutamine** 2 to 20 mcg/kg per minute IV if SBP 70 to 100 mm Hg and no signs/symptoms of shock

Check Blood Pressure

Greater than 100 mm Hg and not less than 30 mm Hg below baseline

ACE Inhibitors
Short-acting agent such as captopril (1 to 6.25 mg)

To 100 mm Hg and no signs/symptoms of shock

Check Blood Pressure

Systolic BP
Greater than 100 mm Hg
and not less than 30 mm Hg
below baseline

ACE Inhibitors
Short-acting agent such as
captopril (1 to 6.25 mg)

Further diagnostic/therapeutic considerations (should be considered in nonhypovolemic shock)

Diagnostic
♥ Pulmonary artery catheter
♥ Echocardiography
♥ Angiography for MI/ischemia
♥ Additional diagnostic studies

Therapeutic
♥ Intra-aortic balloon pump
♥ Reperfusion/revascularization
Emergency Management of Complicated STEMI

- Fluids
- Blood transfusions
- Vasopressors
- Reperfusion/revascularization
- Oxygen
- ACE Inhibitors
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- Hypovolemia
- Administer
  - Fluids
  - Blood transfusions
  - Cause-specific interventions
  - Consider vasopressors

Most likely major underlying disturbance?

- Failure, acute pulmonary edema

Hypovolemia

Signs/symptoms of shock

- Cardiogenic Shock
- Low Output
- Arrhythmia

- Elevated Systolic BP greater than 100 mm Hg
- Low Systolic BP less than 70 mm Hg

See Section 7.7

Patients With ST Elevation Myocardial Infarction-216.

Check Blood Pressure

- Signs/symptoms of shock

- Tachycardia
- Shock, hypoperfusion, congestive heart failure, acute pulmonary edema
- Oxygen

- Dopamine
- Nitroglycerin
- Morphine
- Furosemide

- Norepinephrine
- Dobutamine

Bradycardia

Clinical signs:
Check Blood Pressure

Low Output - Cardiogenic Shock

Systolic BP
70 to 100 mm Hg
NO signs/symptoms of shock

Systolic BP
70 to 100 mm Hg
Signs/symptoms of shock

Systolic BP
Less than 70 mm Hg
Signs/symptoms of shock

Administer
• Fluids
• Blood transfusions
• Cause-specific interventions

Consider vasopressors

Arrhythmia
Bradycardia
Tachycardia

Systolic BP
Greater than 100 mm Hg

Systolic BP
70 to 100 mm Hg

NO signs/symptoms of shock

Systolic BP
Less than 70 mm Hg

Signs/symptoms of shock

Dobutamine
2 to 20 mcg/kg per minute IV

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Hypovolemia

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• Furosemide
• Morphine
• Oxygen/intubation as needed
• Nitroglycerin
• Dopamine
• Dobutamine

First line of action
Second line of action
Third line of action

See Section 7.7 in the ACC/AHA Guidelines for Patients With ST-Elevation Myocardial Infarction

Clinical signs:
Shock, hypoperfusion, congestive heart failure, acute pulmonary edema

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Arrhythmia
Bradycardia
Tachycardia

Systolic BP
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Systolic BP
70 to 100 mm Hg
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Third line of action

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Emergency Management of Complicated STEMI

Administer
• Fluids
• Blood transfusions
• Cause-specific interventions

Consider vasopressors

Arrhythmia

Bradycardia

Tachycardia

Systolic BP

Greater than 100 mm Hg

Systolic BP

70 to 100 mm Hg

NO signs/symptoms of shock

Systolic BP

Less than 70 mm Hg

Signs/symptoms of shock

Dobutamine

2 to 20 mcg/kg per minute IV

Dopamine

5 to 15 mcg/kg per minute IV

Norepinephrine

0.5 to 30 mcg/min IV

First line of action

Second line of action

Third line of action

See Section 7.7 in the ACC/AHA Guidelines for Patients With ST-Elevation Myocardial Infarction

Check Blood Pressure

Clinical signs:

Shock, hypoperfusion, congestive heart failure, acute pulmonary edema

Most likely major underlying disturbance?

Further diagnostic/therapeutic considerations (should be considered in nonhypovolemic shock)

Diagnostic

Therapeutic

♥ Pulmonary artery catheter

♥ Intra-aortic balloon pump

♥ Echocardiography

♥ Reperfusion/revascularization

♥ Angiography for MI/ischemia

♥ Additional diagnostic studies

Acute Pulmonary Edema

Check Blood Pressure

Systolic BP

Greater than 100 mm Hg and not less than 30 mm Hg below baseline

ACE Inhibitors

Short-acting agent such as captopril (1 to 6.25 mg)

Further diagnostic/therapeutic considerations (should be considered in nonhypovolemic shock)

**Diagnostic**
- Pulmonary artery catheter
- Echocardiography
- Angiography for MI/ischemia
- Additional diagnostic studies

**Therapeutic**
- Intra-aortic balloon pump
- Reperfusion/revascularization
Emergency Management of Complicated STEMI

Administer

- Fluids
- Blood transfusions
- Cause-specific interventions
  - Consider vasopressors

Arrhythmia

Bradycardia

Tachycardia

Systolic BP

- Greater than 100 mm Hg
- 70 to 100 mm Hg
- Less than 70 mm Hg

Signs/symptoms of shock

Dobutamine

2 to 20 mcg/kg per minute IV

Low Output - Cardiogenic Shock

Nitroglycerin

10 to 20 mcg/min IV

Dopamine

5 to 15 mcg/kg per minute IV

Norepinephrine

0.5 to 30 mcg/min IV

Hypovolemia

Administer

- Furosemide IV 0.5 to 1.0 mg/kg
- Morphine IV 2 to 4 mg
- Oxygen / intubation as needed
- Nitroglycerin SL, then 10 to 20 mcg/min IV if SBP greater than 100 mm Hg
- Dopamine 5 to 15 mcg/kg per minute IV if SBP 70 to 100 mm Hg and signs/symptoms of shock present
- Dobutamine 2 to 20 mcg/kg per minute IV if SBP 70 to 100 mm Hg and no signs/symptoms of shock

First line of action

Second line of action

Third line of action

See Section 7.7 in the ACC/AHA Guidelines for Patients With ST-Elevation Myocardial Infarction

Clinical signs:

- Shock, hypoperfusion, congestive heart failure, acute pulmonary edema

Most likely major underlying disturbance?

Further diagnostic/therapeutic considerations (should be considered in nonhypovolemic shock)

- Pulmonary artery catheter
- Intra-aortic balloon pump
- Echocardiography
- Reperfusion/revascularization
- Angiography for MI/ischemia
- Additional diagnostic studies

Acute Pulmonary Edema

Check Blood Pressure

Systolic BP

- Greater than 100 mm Hg and not less than 30 mm Hg below baseline

ACE Inhibitors

Short-acting agent such as captopril (1 to 6.25 mg)
