Classification of Recommendations and Levels of Evidence

- **Class I**: Benefit >>> Risk
  - Procedure/Treatment SHOULD be performed/administered
  - Benefit should be indicated
  - Procedure/treatment is useful/effective/beneficial
  - Is recommended
  - Is indicated
  - Is probably recommended or indicated
- **Class IIa**: Benefit >> Risk
  - Procedure/Treatment MAY BE CONSIDERED
  - Is reasonable
  - Usefulness/effectiveness is unknown/unclear/uncertain or not well established
  - May/might be considered
- **Class IIb**: Benefit ≥ Risk
  - Additional studies with broad objectives needed; Additional registry data would be helpful
  - Procedure/Treatment MAY BE CONSIDERED
  - Is probably recommended or indicated
- **Class III**: Risk ≥ Benefit
  - Procedure/Treatment should NOT be performed/administered
  - Since it is not helpful and may be harmful
  - Should not be used
  - Is not useful/effective/beneficial
  - Is not recommended
  - Is not indicated
  - Should not be indicated

Guideline Documents

**What are they?**
- Collections of evidence packed in a format that reflects consensus on what likely will be optimal
- Documents designed to challenge us to better practice
- Living evolving collections of evidence

**What are they not?**
- Guidelines are NOT mandates, they are guides
- Documents which do not limit our abilities to tailor care to our patients
- Legal standards for litigation

Updates from ACC.17

Hospitalizations in the U.S. due to Acute Coronary Syndromes (ACS)

- **Acute Coronary Syndromes***
  - 1.57 Million Hospital Admissions - ACS
  - 1.24 million Admissions per year
  - .33 million Admissions per year

*Primary and secondary prevention. About 0.57 million NSTEMI and 0.67 million UA.

Approximately every 44 seconds, an American will have an MI.
Epidemiology

Incidence rates for STEMI have declined over the past decade, whereas those for non–ST-elevation ACS have increased.

At present, STEMI comprises approximately 25% to 40% of MI presentations.

Presentation
Working Dx
ECG
Cardiac Biomarker
Final Dx

Ischemic Discomfort
Acute Coronary Syndrome

No ST Elevation
ST Elevation

Non-ST ACS
NSTEMI
Myocardial Infarction
Qw MI

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Kristine A. Scordo, PhD, RN, ACNP-BC FAANP

**Low Grade Stenoses Cause Most Infarctions**

Coronary Stenosis Severity Prior to MI

- 50-70% Stenosis: 18%
- >70% Stenosis: 14%
- <50% Stenosis: 68%

- Mild stenoses are not inherently more ‘vulnerable’ than severe stenoses
- Both mild and severe can cause MI
- Minimal → mild stenoses more prevalent than severe stenoses

Lesions at greatest risk of causing MI

- Increased plaque burden
- Thin-cap fibroatheroma
- Minimal lumen area ≤4.0 mm

*No imaged coronary segment with <40% plaque burden resulted in a nonculprit event* Stone, PROSPECT Trial—NEJM 2011

**Spectrum of Chronic Coronary Syndrome**

- Risk Factors + Hypertension
- Endothelial Dysfunction
- Atherosclerosis
- HbD-Angina Pectoris
- Myocardial Ischemia
- Coronary Thrombosis
- Myocardial Infarction
- Arrhythmia & Loss of Muscle
- Remodeling
- Ventricular Dilation
- Congestive Heart Failure
- Endstage Heart Disease

Baroldi G. The Etiopathogenesis of Coronary Heart Disease. 2nd ed. 2004.

**ACC/AHA Guidelines: Key Points**

- Initial treatment of STEMI continues to be restoring blood flow to the heart ASAP
- STEMI patients should be treated within 90 minutes of first medical contact (door-to-balloon [D2B] time); if not possible, treat with fibrinolytic therapy within 30 minutes;
- Avoid fibrinolytic therapy in NSTE-ACS
- New medication guidelines that may improve long-term prognosis
- Modification of risk factors

From the beginning……
Acute Coronary Syndromes: Unstable Angina and Non-ST-Segment Elevation Myocardial Infarction
Kristine A. Scordo, PhD, RN, ACNP-BC FAANP

Options for Transport of Patients With STEMI and Initial Reperfusion Treatment

Little impact - ~ 114 minutes before patient reports symptoms

EMS on-scene
- Enrollees in non-STEMI or STEMI not PCI capable, EMS to reach within 30 min.
- STEMI patients within 90 min.
- STEMI transport within 90 min.
- PCI capable

EMS Transport
- Prehospital fibrinolysis
- STEMI patients within 30 min.
- STEMI transport within 90 min.
- PCI capable

Inter-Hospital Transfer
- PCI capable

PCI within 30 min.
- STEMI transport within 90 min.
- PCI capable

EMS Dispatch 1 min.

GOALS
- STEMI patients within 30 min.
- STEMI transport within 90 min.
- PCI capable

Golden Hour = first 60 min.
Total ischemic time: within 120 min.

Reperfusion Therapy for Patients with STEMI

Figure 1. Options for transportation of STEMI patients and initial reperfusion treatment.

D2B Evidence-based Strategies

1. ED physician activates the cath lab
2. One call activates the cath lab
3. Cath lab team ready in 20-30 minutes
4. Prompt data feedback
5. Senior management commitment
6. Team-based approach
* Pre-hospital ECG to activate the cath lab (optional)

Reality Check

- Door to balloon time
  - <90 min 4.2%
  - <2 h 16.2%
- European Trials (DANAMI-2 & Prague-2)
  - Transport times
  - Centralized network
  - Weather-simply better in Europe!
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Does it matter EMS vs. Self-Drive?

Is there any difference in care for EMS vs. Self-Drive patients with Chest Pain?

- 1 in 300 patients who self drive has a cardiac arrest enroute
- EMS has been shown to facilitate earlier hospital treatment
- Allows for pre-hospital ECG & fibrinolytic checklist – possible triage to interventional center


Relationship Between Mortality Reduction and Extent of Salvage

Early Risk Stratification

<table>
<thead>
<tr>
<th>Recommendation</th>
<th>COR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rapid assessment: 12-Lead EKG within 10 min</td>
<td>I</td>
</tr>
<tr>
<td>Serial EKG 15-30 min intervals during first hour in symptomatic pts with initial nondiagnostic EKG</td>
<td>I</td>
</tr>
<tr>
<td>Measure cardiac troponin</td>
<td>I</td>
</tr>
<tr>
<td>Measure serial cardiac troponin l or T 3-6 hrs after symptom onset</td>
<td>I</td>
</tr>
<tr>
<td>Use risk scores to assess prognosis</td>
<td>I</td>
</tr>
<tr>
<td>Obtain supplement EKG leads V1 - V6 in pts with initial nondiagnostic EKG at intermediate/high risk for ACS</td>
<td>Ila</td>
</tr>
<tr>
<td>Continuous 12-Lead EKG monitoring may be reasonable alternation with initial nondiagnostic EKG in patients at intermediate/high risk</td>
<td>Ila</td>
</tr>
<tr>
<td>BNP or NT-pro-BNP may be considered to assess risk in patients with suspected ACS</td>
<td>IIb</td>
</tr>
</tbody>
</table>

Reperfusion Time, Myocardial Salvage and Mortality

Evaluation and Management of Patients With STEMI and Out-of-Hospital Cardiac Arrest

Therapeutic hypothermia should be started as soon as possible in comatose patients with STEMI and out-of-hospital cardiac arrest caused by VF or pulseless VT, including patients who undergo primary PCI.

Immediate angiography and PCI when indicated should be performed in resuscitated out-of-hospital cardiac arrest patients whose initial ECG shows STEMI.
Acute Coronary Syndromes: Unstable Angina and Non-ST-Segment Elevation Myocardial Infarction
Kristine A. Scordo, PhD, RN, ACNP-BC FAANP

**DIAGNOSIS ACS: UNSTABLE ANGINA VS NSTEMI VS STEMI**

**Clinical classification of MI**

<table>
<thead>
<tr>
<th>Classification</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Spontaneous MI related to ischemia due to a primary coronary event, such as plaque erosion and/or rupture, fissuring, or dissection</td>
</tr>
<tr>
<td>2</td>
<td>MI secondary to ischemia due to an imbalance of O2 supply and demand, as from coronary spasm or embolism, anemia, arrhythmias, hypotension, or hypertension</td>
</tr>
<tr>
<td>3</td>
<td>Sudden unexpected cardiac death, including cardiac arrest, often with symptoms suggesting ischemia with new ST-segment elevation; new left bundle branch block; or pathologic or angiographic evidence of fresh coronary thrombus—in the absence of reliable biomarker findings</td>
</tr>
<tr>
<td>4a</td>
<td>MI associated with PCI</td>
</tr>
<tr>
<td>4b</td>
<td>MI associated with documented in-stent thrombosis</td>
</tr>
<tr>
<td>5</td>
<td>MI associated with CABG surgery</td>
</tr>
</tbody>
</table>


**Diagnosis of AMI (WHO Definition)**

- presence of at least two of the following criteria
  - clinical history of ischemic-type chest discomfort
  - changes on serially obtained EKG tracings
  - rise and fall in serum cardiac markers
- <25% patients admitted with ischemic-type pain are diagnosed with AMI
- ST segment elevation present in 50% of patients with MI
- lab plays essential role in diagnosing MI

**cCTA in acute chest pain**

- Allows for rapid noninvasive detection of CAD
- Widely available
- Sufficient accuracy and safety data available
- Biological information now possible (“high risk plaque”)
Acute Coronary Syndromes: Unstable Angina and Non-ST-Segment Elevation Myocardial Infarction
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**Coronary Vasomotor Disorders**
- Large Vessel Dysfunction
  - Vasospastic Angina

**Microvascular Dysfunction**
- Cardiac Syndrome X
- Microvascular Angina
- Microvascular Spasm
- Coronary Slow Flow

**EKG**
- STEMI:
  - Q waves, ST elevations, hyper acute T waves; followed by T wave inversions.
  - Clinically significant ST segment elevations:
    - > 1 mm (0.1 mV) in at least two anatomical contiguous leads
    - or 2 mm (0.2 mV) in two contiguous precordial leads (V2 and V3)

Note: LBBB and pacemakers can interfere with diagnosis of MI on EKG
Acute Coronary Syndromes:
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EKG

- **NSTEMI:**
  - ST depressions (0.5 mm at least) or T wave inversions (1.0 mm at least) without Q waves in 2 contiguous leads with prominent R wave or R/S ratio >1.
  - Isolated T wave inversions:
    - can correlate with increased risk for MI
    - may represent Wellen's syndrome:
      - critical LAD stenosis
      - >2mm inversions in anterior precordial leads

Wellen's syndrome: note the deeply negative T wave (>5 mm) in V2-V4; biphasic shape in lead V2. By definition, in Wellen's syndrome, no ST elevation >1mm and no Q waves (Wellen is not a STEMI)

Higher Risk ECG Features

- Major ST segment elevation in multiple leads
- ST segment elevation in leads V1 and aVR accompanied by ST depression in leads V3-V5 correlates with more frequent left main or extensive coronary disease
- Q waves in multiple vascular distributions accompanied by new ischemic ST segment changes

LAD Total Occlusion

ECG manifestations of acute myocardial ischemia occur within minutes; abnormal detectable biomarker release requires hours. Several ECG's should be acquired at presentation, particularly when the initial ECG is nondiagnostic. Lesion severity, location and extent, coronary dominance, collateral blood flow, vessel supply to conduction system, and prior myocardial necrosis impact morphology of ECG waveforms.

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Acute Coronary Syndromes:  
Unstable Angina and Non-ST-Segment Elevation Myocardial Infarction  
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LAD Occlusion: Post PCI

Left Ventricular Cineangiogram

Ejection Fraction (EF) = 26%

Remember the DDx for Chest Pain

- ACS
- Aortic Dissection
- Pulmonary Embolism
- Acute cholecystitis
- Pericarditis
- Costocondritis
- Esophageal spasm
- Many others

The Can’t Misses

ISCHEMIC HEART DISEASE EVALUATION

Table 3. Differential Diagnosis of STEMI

<table>
<thead>
<tr>
<th>Life-threatening</th>
<th>Other cardiovascular and nonischemic</th>
<th>Other noncardiac</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aortic dissection</td>
<td>Pericarditis</td>
<td>Gastroesophageal reflux (GERD) and spasm</td>
</tr>
<tr>
<td>Pulmonary embolus</td>
<td>Atypical angina</td>
<td>Chest-wall pain</td>
</tr>
<tr>
<td>Perforating ulcer</td>
<td>Early repolarization</td>
<td>Pleurisy</td>
</tr>
<tr>
<td>Tension pneumothorax</td>
<td>Wolff-Parkinson-White syndrome</td>
<td>Peptic ulcer disease</td>
</tr>
<tr>
<td>Bronchial syndrome</td>
<td>Deeply inverted T-waves suggestive of a central nervous system lesion or apical hypertrophic cardiomyopathy</td>
<td>Pancreatic attack</td>
</tr>
<tr>
<td>Echocardiographic rupture with mediastinitis</td>
<td>Hyperkalemia</td>
<td>Bilary or pancreatic pain</td>
</tr>
<tr>
<td>Bronchiolitis</td>
<td>Myocarditis</td>
<td>Cervical disc or neuropathic pain</td>
</tr>
<tr>
<td>Acute tension pneumothorax</td>
<td>Hypertension</td>
<td>Somatization and psychogenic pain disorder</td>
</tr>
<tr>
<td>Acute aortic dissection</td>
<td>Hypertrophic cardiomyopathy</td>
<td></td>
</tr>
</tbody>
</table>

What have I got?

- based on history, physical exam, EKG & cardiac markers
- categorized into one of four groups
  - non-cardiac chest pain
  - stable angina
  - unstable angina
  - MI
    - STEMI vs non-STEMI
**Acute Coronary Syndromes:**
Unstable Angina and Non-ST-Segment Elevation Myocardial Infarction
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**The History**
- most important factors that relate to the likelihood of ischemia due to CAD:
  - nature of the anginal symptoms
  - prior history of CAD
  - sex
  - age
  - number of traditional risk factors present

**Unstable Angina (UA)**
- acute coronary syndrome between stable angina and MI
- rapid increase in coronary stenosis, but incomplete occlusion
- chest pain evident:
  - at rest
  - on effort with change in previous pattern, variant
  - De novo becoming severe within 1-2 months of starting
  - previous stable angina becoming severe
- not associated with myocardial necrosis, but continuing risk of this

**Presentations of UA**
- rest angina
- new-onset severe angina
- increasing angina

**Precipitating Factors**
- second-wind phenomenon or walk-through angina
  - discomfort that develops during exertion but disappears while the activity is continued
- early-morning activity after a night’s sleep may precipitate angina that the same level of activity later in the day does not
- after heavy meal, smoking
- emotional stress
  - tends to last longer than that produced by physical stress
  - emotions not as easily controlled or abated as activity

**Precipitating factors**
- Inappropriate tachycardia
  - anemia, fever, hypoxia, tachyarrhythmias, thyrotoxicosis
- High afterload
  - aortic valve stenosis, LVH
- High preload
  - high cardiac output, chamber dilatation
- Inotropic state
  - sympathomimetic drugs, cocaine intoxication

**Cocaine and ACS**
- causes coronary vasospasm and thrombosis
- sinus tachycardia, increases BP, increases myocardial contractility—MVO₂
- important to inquire about use of cocaine, especially younger patients (<40 years)
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Clinical Characteristics of Angina

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<th>Characteristic</th>
<th>More likely to be angina</th>
<th>Less likely to be angina</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type of pain</td>
<td>Dull, pressure</td>
<td>Sharp, stabbing</td>
</tr>
<tr>
<td>Duration</td>
<td>2 to 5 min, always &lt;15–20 min</td>
<td>Seconds or hours</td>
</tr>
<tr>
<td>Onset</td>
<td>Gradual</td>
<td>Rapid</td>
</tr>
<tr>
<td>Location</td>
<td>Subternal</td>
<td>Lateral chest wall, back</td>
</tr>
<tr>
<td>Reproducible</td>
<td>With exertion</td>
<td>With inspiration</td>
</tr>
<tr>
<td>Associated symptoms</td>
<td>Present</td>
<td>Absent</td>
</tr>
<tr>
<td>Palpation of chest wall</td>
<td>Not painful</td>
<td>Painful, exactly reproduces pain complaint</td>
</tr>
</tbody>
</table>

Clinical Presentation: Gender Differences

Although at any age women have a lower incidence of CAD than men, they account for a considerable proportion of UA/NSTEMI. It is important to overcome long-held notions that severe coronary manifestations are uncommon in women. Women with CAD are, on average, older than men and are more likely to have comorbidities such as HTN, DM, and HF; to manifest angina rather than AMI; and have atypical symptoms.

Physical Examination

- Physical Exam
  - BP both arms
  - unequal pulses, AI with tearing-like back pain
  - auscultate for bruits and pulse deficits
    - carotid, aortic, peripheral
  - S4 – decreased LV compliance
  - rales, S3 gallop – LV failure
  - MR – ischemia of papillary muscle
  - pericardial friction rub with pulsus paradoxus
    - acute pericarditis

- What is the person’s risk?
  - RISK STRATIFICATION

Women do not snore, burp, sweat or pass gas. Therefore, they must "BITCH or they will BLOW UP!"
Which of the following features suggests high risk for obstructive coronary artery disease in a patient presenting with signs and/or symptoms of an acute coronary syndrome?

A) Diaphoresis  
B) Male gender  
C) Age >70 years  
D) History of diabetes  
E) Minor T wave changes  
F) History of extra-cardiac vascular disease

Which of the following features suggests high risk for obstructive coronary artery disease in a patient presenting with signs and/or symptoms of an acute coronary syndrome?

A) Diaphoresis  
B) Male gender  
C) Age >70 years  
D) History of diabetes  
E) Minor T wave changes  
F) History of extra-cardiac vascular disease

Early Risk Stratification

- Likelihood of CAD: high, intermediate or low risk
- Risk stratified by history, clinical findings, ECG and cardiac markers
- Obtain 12 Lead EKG within 10 minutes

<table>
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<tr>
<th>Risk Level</th>
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<th>Intermediate</th>
<th>Low</th>
</tr>
</thead>
<tbody>
<tr>
<td>History</td>
<td>Symptoms such as prior angina</td>
<td>Chest or left arm pain</td>
<td>Possibility ischaemic (symptoms) with no intermediate risk factors</td>
</tr>
<tr>
<td></td>
<td>Known CAD/MI</td>
<td>Male</td>
<td>Cocaine use</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Diabetes</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Age &gt;70</td>
<td></td>
</tr>
<tr>
<td>Exam</td>
<td>Transient MR</td>
<td>Hypotension</td>
<td>Reproduced by palpation</td>
</tr>
<tr>
<td></td>
<td>Diaphoresis</td>
<td>CHF</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Extra-cardiac vascular disease</td>
<td></td>
</tr>
<tr>
<td>ECG</td>
<td>New ST deviation (&gt;1 mm) or T wave inversion in multiple leads</td>
<td>Fixed ECG changes</td>
<td>Minor T wave changes or normal ECG</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Less marked ST, T wave changes</td>
<td></td>
</tr>
<tr>
<td>Cardiac Markers</td>
<td>Elevated troponin or CK-MB</td>
<td>Normal</td>
<td>Normal</td>
</tr>
</tbody>
</table>

Variables Used in the TIMI Risk Score

- Age ≥ 65 years
- At least 3 risk factors for CAD
- Prior coronary stenosis of ≥ 50%
- ST-segment deviation on ECG presentation
- At least 2 anginal events in prior 24 hours
- Use of aspirin in prior 7 days
- Elevated serum cardiac biomarkers
Acute Coronary Syndromes: Unstable Angina and Non-ST-Segment Elevation Myocardial Infarction
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**TIMI Risk Score**

<table>
<thead>
<tr>
<th>TIMI Risk Score</th>
<th>All-Cause Mortality, New or Recurrent MI, or Severe Recurrent Ischemia Requiring Urgent Revascularization Through 14 Days After Randomization %</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-1</td>
<td>4.7</td>
</tr>
<tr>
<td>2</td>
<td>8.3</td>
</tr>
<tr>
<td>3</td>
<td>13.2</td>
</tr>
<tr>
<td>4</td>
<td>19.9</td>
</tr>
<tr>
<td>5</td>
<td>26.2</td>
</tr>
<tr>
<td>6-7</td>
<td>40.9</td>
</tr>
</tbody>
</table>


The TIMI risk calculator is available at www.timi.org.


TIMI = Thrombolysis in Myocardial Infarction.

---

**GRACE Risk Score**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Odds ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Older age</td>
<td>1.7 per 10 y</td>
</tr>
<tr>
<td>Killip class</td>
<td>2.0 per class</td>
</tr>
<tr>
<td>Systolic BP</td>
<td>1.4 per 20 mm Hg ↑</td>
</tr>
<tr>
<td>ST-segment deviation</td>
<td>2.4</td>
</tr>
<tr>
<td>Cardiac arrest during presentation</td>
<td>4.3</td>
</tr>
<tr>
<td>Serum creatinine level</td>
<td>1.2 per 1 mg/dL ↑</td>
</tr>
<tr>
<td>Positive initial cardiac biomarkers</td>
<td>1.6</td>
</tr>
<tr>
<td>Heart rate</td>
<td>1.3 per 30-beat/min ↑</td>
</tr>
</tbody>
</table>

Eagle KA, et al. JAMA 2004;291:2727–33. The GRACE clinical application tool can be found at www.outcomesumassmed.org/grace. Also see Figure 4 in Anderson JL, et al. J Am Coll Cardiol 2007;50:e1–e157.

GRACE = Global Registry of Acute Coronary Events.

---

**High Risk for CAD**

- accelerating symptoms
- prolonged ongoing (>20 minutes) rest pain
- pulmonary edema, new or worsening MR Murmur, S3 gallop or new/worsening rales
- hypotension, bradycardia, tachycardia
- age > 75 years
- ST-segment changes >0.05mV, BBB, VT
- markedly elevated troponin

---

**Unstable Angina**

**Risk Stratification and Management**

**Unstable Angina Risk Stratification**

**Low Risk**

- new-onset exertional angina
- minor chest pain during exercise
- pain relieved promptly by nitroglycerine

**Management**

- can be managed safely as an outpatient (assuming close follow-up and rapid investigation)

**Intermediate Risk**

- prolonged chest pain
- diagnosis of rule-out MI

**Management**

- observe in the ER or Chest Pain Unit
- monitor clinical status and ECG
- obtain cardiac enzymes (troponin T or I) every 8 to 12 hours

---

**Unstable Angina**

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Unstable Angina
Risk Stratification

High Risk
- recurrent chest pain
- ST-segment change
- hemodynamic compromise
- elevation in cardiac enzymes

Management
- monitor in the Coronary Care Unit

Laboratory Examinations

- Serum biomarkers for myocardial damage
  - Measure serial cardiac troponin I or T at presentation and 3–6 h after symptom onset in all patients with symptoms consistent with ACS
- Complete blood count (CBC) with platelets
- Comprehensive metabolic panel (renal/glucose/liver)
- INR/aPTT
- HbA1c
- Complete lipid profile
- BNP or NT-pro-BNP may be considered to supplement assessment of global risk in patients with suspected ACS

Timing of Release of Various Biomarkers After Acute Myocardial Infarction

Troponin: Non-ACS increases+++  
- myocarditis
- cardiac contusion
- cardioversion, RF ablation
- CHF
- chemotherapy (adriamycin, 5-FU)
- septic shock
- PE
- extreme endurance athletes
- end-stage renal disease

Troponin
- check risk and fall
- not early release marker (similar to CPK)
- 10-14 days stays elevated
  - hard to detect reinfarction
- check 6-9 hours after symptom onset
  - 0.1-1.5 injury
  - >1.5 definite AMI

Cardiac troponin I (cTnI) levels in a healthy reference population and in an acute coronary syndrome (ACS) population.
Acute Coronary Syndromes: Unstable Angina and Non-ST-Segment Elevation Myocardial Infarction
Kristine A. Scordo, PhD, RN, ACNP-BC FAANP

Fibrinolysis

Fibrinolysis: Class I Ia/b

STEMI patients presenting to a facility without the capability for expert, prompt intervention with primary PCI within 90 minutes of first medical contact should undergo fibrinolysis unless contraindicated. (Level of Evidence: A)

Fibrinolysis: Class IIa

Fibrinolytic therapy should not be administered to asymptomatic patients whose initial symptoms of STEMI began more than 24 hours earlier. (Level of Evidence: C)

Fibrinolysis: Class IIb

Fibrinolytic therapy should not be administered to patients whose 12-lead ECG shows only ST-segment depression except if a true posterior MI is suspected. (Level of Evidence: A)

Absolute contraindications
- Any prior ICH
- Known structural coronary vascular lesion (e.g., AVM)
- Known malignant intracranial neoplasm (primary or metastatic)
- Ischemic stroke within 3 months EXCEPT acute ischemic stroke within 3 hours
- Suspected aortic dissection
- Active bleeding or bleeding diathesis (excluding menorrhagia)
- Significant closed head or facial trauma within 3 months

Relative contraindications
- History of chronic severe, poorly controlled hypertension
- Severe uncontrolled hypertension on presentation (SBP greater than 180 mm Hg or DBP greater than 110 mm Hg)
- History of prior ischemic stroke greater than 3 months, dementia, or known intracranial pathology not covered in contraindications
- Traumatic or prolonged (greater than 10 minutes) CPR or major surgery (less than 3 weeks)
- Recent (within 2 to 4 weeks) internal bleeding
- Noncompressible vascular punctures
- For streptokinase/nnanistreplase: prior exposure (more than 5 days ago) or prior allergic reaction to these agents
- Pregnancy
- Active peptic ulcer
- Current use of anticoagulants: the higher the INR, the higher the risk of bleeding

Fibrinolytic agent | dose | fibrin specificity | anticoagulant | fibrinolytic rate (50-70 kIU in 1 hour)
---|---|---|---|---
Tissue-type | --- | --- | no | 8% (22)
Recombinant (r) | 10-40 kIU intravenous in 30 min apart | no | 4% (21)
Hemochromatous | 50-100 kIU intravenous | yes | 7% to 6% (14,25,26)
Non-recombinant | --- | --- | no | 6% to 6% (24,25,26)

Bleeding Risk

Crusade bleeding score...determine baseline risk of in-hospital major bleeding
Acute Coronary Syndromes:
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Definitions

- **Primary PCI**
  - performed as the initial approach to reperfusion in the acute phase of STEMI with the goal of promptly restoring blood flow and function to the portion of the heart that is jeopardized by an acute coronary artery occlusion

- **Secondary PCI**
  - PCI after either successful or failed fibrinolysis

- **Rescue PCI**
  - PCI in a closed artery following fibrinolytic therapy

- **Adjunctive PCI**
  - PCI in an open artery following fibrinolytic therapy. Adjunctive PCI is defined as the intent to administer fibrinolytic agent in the setting of STEMI, and the performance of PCI for partial success of the fibrinolytic agent is unintended

---

Table 2. Primary PCI in STEMI

<table>
<thead>
<tr>
<th>Cor</th>
<th>LOE</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>A</td>
<td>17, 50, 51</td>
</tr>
<tr>
<td>I</td>
<td>B</td>
<td>52, 53</td>
</tr>
<tr>
<td>I</td>
<td>B</td>
<td>54–57</td>
</tr>
<tr>
<td>I</td>
<td>B</td>
<td>29, 30</td>
</tr>
<tr>
<td>I</td>
<td>B</td>
<td>58–60</td>
</tr>
</tbody>
</table>

COR indicates Class of Recommendation; FMC, first medical contact; HF, heart failure; LOE, Level of Evidence; MI, myocardial infarction; PCI, percutaneous coronary intervention; and STEMI, ST-elevation myocardial infarction.
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Evolution of PCI

Stents: Factoids

BMS (bare metal stent)
- Restenosis 20-30%
- At least one month APT
- Hazard higher if anticoagulant therapy discontinued for surgery; need surgery wait at least 6 weeks

DES (drug eluting stent)
- Superior to BMS
- Less restenosis
- Need dual antiplatelet therapy at least 3-6 months
- Avoid in pts with high risk bleeding; no insurance???
- 5-fold increase in bleeding if on coumadin
- Better for diabetics

TIMI Myocardial Perfusion (TMP) Grades

TIMI Flow Grades

Anticoagulant Therapy in Context

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Adjunctive Antithrombotic Therapy to Support Reperfusion With Primary PCI

A loading dose of a P2Y₁₂ receptor inhibitor should be given as early as possible or at time of primary PCI to patients with STEMI:
• Clopidogrel (Plavix) 600 mg; or
• Prasugrel (Effient) 60 mg*; or
• Ticagrelor (Brilinta) 180 mg

*Prasugrel should not be administered to patients with a history of prior stroke or transient ischemic attack.

Prasugrel and Ticagrelor
Pharmacodynamics of Platelet Inhibition vs Clopidogrel

Cangrelor
A Potent, Fast Onset - Fast Offset IV P2Y₁₂ Inhibitor for PCI

P2Y₁₂ inhibitor therapy should be given for 1 year to patients with STEMI who receive a stent (BMS or DES) during primary PCI using the following maintenance doses:
• Clopidogrel 75 mg daily; or
• Prasugrel 10 mg daily; or
• Ticagrelor 90 mg twice a day*

*The recommended maintenance dose of aspirin to be used with ticagrelor is 81 mg daily.
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Guideline Recommendation for Duration of DAPT after ACS

<table>
<thead>
<tr>
<th>Society</th>
<th>ACS</th>
<th>Management</th>
<th>Recommended Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>NSTEMI</td>
<td>Medical</td>
<td>Up to 12 mos</td>
<td></td>
</tr>
<tr>
<td>PCI</td>
<td>Lytic</td>
<td>At least 12 mos</td>
<td></td>
</tr>
<tr>
<td>STEMI</td>
<td>PCI</td>
<td>12 mos (may consider beyond 12 mos)</td>
<td></td>
</tr>
<tr>
<td>All</td>
<td>All</td>
<td>12 mos Strict min if 1 mo if BMS, 6 mos if DES</td>
<td></td>
</tr>
<tr>
<td>All</td>
<td>All</td>
<td>12 mos (After 12 mos, rec single antiplatelet Rx or continuing DAPT)</td>
<td></td>
</tr>
</tbody>
</table>

The bottom line….DAPT

- If on oral anticoagulants, short DAPT (3m).
- If bleeding in first year, short DAPT (3m if possible).
- If necessary to stop for surgery, restart soon after.
- If no major bleeding in the first year, continue DAPT beyond one year to prevent future MI.

Mauri, Kereiakes et al AHJ 2010; 160(6): 1035-1041

Vorapaxar (Zontivity)

- **Indication**
  - Reduction of thrombotic CV events in patients with a history of MI or with PAD, in combination with aspirin and/or clopidogrel
  - Contraindications:
    - History of stroke, TIA, ICH
    - Active Pathologic Bleeding

**Drug Facts**

- **Pharmacology:**
  - Reversible antagonist of protease-activated receptor-1 (PAR-1)
  - Long t½ makes it effective irreversible
  - Inhibits thrombin-induced and thrombin receptor agonist peptide (TRAP)-induced platelet aggregation

- **Dosing:** one 2.08 mg tablet by mouth once daily, with or without food

Table 4. Guideline-Recommended Antiplatelet and Anticoagulant Dosing for Initial Medical Treatment in NSTEMI(1)

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dosing Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Oral Antiplatelet Therapies</strong></td>
<td></td>
</tr>
<tr>
<td>Aspirin</td>
<td>162.325 mg enteric coated, orally or chewed</td>
</tr>
<tr>
<td>Clopidogrel</td>
<td>LOE of 300-600 mg/Day</td>
</tr>
<tr>
<td>Ticlopidine</td>
<td>LOE of 600 mg/Day</td>
</tr>
<tr>
<td>All</td>
<td>LOE of 30 mg/Day</td>
</tr>
<tr>
<td><strong>Anticoagulant Therapies</strong></td>
<td></td>
</tr>
<tr>
<td>Unfractionated Heparin</td>
<td>LOE of 60 U/kg (max 4000 U)</td>
</tr>
<tr>
<td>Enoxaparin</td>
<td>LOE of 30-40 U/kg every 12 hours</td>
</tr>
<tr>
<td>Fondaparinux</td>
<td>2.5 mg SC once daily</td>
</tr>
<tr>
<td><strong>Intravenous Antiplatelet Therapy</strong></td>
<td></td>
</tr>
<tr>
<td>Epifibatide</td>
<td>LOE of IV bolus 180 mcg/kg</td>
</tr>
<tr>
<td>Tirofiban</td>
<td>LOE of IV bolus 0.5 mcg/kg/min</td>
</tr>
</tbody>
</table>

MY PATIENT IS GOING FOR CABG, NOW WHAT??

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Antithrombotic Therapy

Aspirin should not be withheld before urgent CABG.

Clopidogrel or ticagrelor should be discontinued at least 24 hours before urgent on-pump CABG, if possible.

Short-acting intravenous GP IIb/IIIa receptor antagonists should be discontinued at least 2 to 4 hours before urgent CABG; Abciximab discontinue at least 12 hours before urgent CABG.

Anti-Ischemic Therapy

Aspirin should be chewed by patients who have not taken aspirin before presentation with STEMI. The initial dose should be 162 mg (Level of Evidence: A) to 325 mg (Level of Evidence: C)

Although some trials have used enteric-coated aspirin for initial dosing, more rapid buccal absorption occurs with non-enteric-coated formulations.

Triage of Patients

- ACS: Admit to CCU
- Transfer out of CCU within 24 to 36 hours in absence of:
  - hx of previous MI
  - persistent ischemic pain
  - CHF
  - hypotension
  - heart block
  - hemodynamically compromising ventricular arrhythmias

General Care

- continuous ECG monitoring
  - MCL
- bed rest
  - progress activity when symptom free
- oxygen
  - cyanosis, respiratory distress,
  - low arterial saturation (<90%) first 6 hours

Oxygen

- little justification to continue use beyond 2-3 hours
- excessive oxygen administration can lead to systemic vasoconstriction
- high flow rates can be harmful to patients with COLD
- oxygen and nitrates

  NTG dilates pulmonary vascular bed and increases ventilation-perfusion abnormalities—reasonable to provide supplemental oxygen
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Analgesia
- Morphine remains Class I for STEMI although may increase adverse events in UA/NSTEMI (2-4 mg IV with 2-8 MG repeated at 5-10 min intervals)
- NSAID medications increase mortality, reinfarction, and heart failure in proportion to degree of COX-2 selectivity
  - Discontinue on admission for STEMI
  - Do not initiate during acute phase of management

NSAIDS….FACTOIDS
- Avoid NSAIDS…because….
  - Block prostaglandins
  - Renal insufficiency-hyperkalemia
  - Indocin-thins infarct scar
  - Increased risks of mortality, reinfarction, HTH, HF and myocardial rupture

Diet
- NCEP Adult Treatment Panel III (ATP III) Therapeutic Lifestyle Changes (TLC) diet
  - Reduced intake of fats and cholesterol, less than 7% of total calories as saturated fats
  - less than 200 mg of cholesterol per day
  - increased consumption of omega-3 fatty acids
  - appropriate caloric intake for energy needs.
- Restrict sodium intake with HTN of HF

Caffeine
- no need to limit caffeine
  - BP changes related to caffeine consumption not significant until 400 mg of caffeine consumed
  - ~88 mg cup of brewed coffee
  - withdrawal causes headaches and increase in heart rate

“Coronary Precautions”
- despite the fact that research does not support their use, they are still some areas that practice CP
  - bed rest
  - restricted ice/hot fluids
  - assistance with eating
  - avoid caffeine
  - avoidance of Valsalva maneuver is the only coronary precaution of universal significance
    - causes sudden and intense changes in systolic BP and heart rate; may predispose patient to ventricular arrhythmias

Alleviate Pain and Anxiety
- essential element
- pain and anxiety contribute to excessive activity of autonomic nervous system and to restlessness
  - increased heart rate
  - increased afterload
  - augmentation of cardiac contractility
  - heightened tendency to occurrence of ventricular tachyarrhythmias

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Cardiac Imagery

- many patients surround themselves with negative thinking and negative images of their body, their heart, and their health
- pts describe images of their healing heart as a black hole inside the heart, a big, hot swollen inflamed bag
- we’ve done a good job in passing on images and words that describe malfunctions and pathophysiology

Guzzetta, C. Capsules and comments in critical care nursing, 1993;1:34.

Cardiac Imagery (con’t)

- we’ve not done as well in transmitting images patients need to conjure up healing and recovery
- what we think and feel change our physiology
- process imagery
  - strategy for guiding patients in a step-by-step biologic healing process
  - example: teach images of healthy, healing scar formation, images of a strong scar (superglue), new collaterals (beautiful lattice network) and strong pipes (coronary) through which blood flows without tension

Cardiac Imagery (con’t)

- End-stage imagery
  - strategy for guiding patients to rehearse being in a final, healed state
  - visions of a strong, healed heart
  - visions of returning to family activities, exercise, health diet, work and sexual activity
- combine strategies with relaxation, music and guided imagery sessions

Nitrates

Recommendations

| Patients with ACS with continuing ischemic pain should receive sublingual nitroglycerin (0.3 mg to 0.4 mg) every 5 minutes for up to 3 doses, after which an assessment should be made about the need for intravenous nitroglycerin if not contraindicated. | I | C |
| Intravenous nitroglycerin is indicated for patients with ACS for the treatment of persistent ischemia, HF, or hypertension. | I | B |
| Nitrates should not be administered to patients with NSTE-ACS who recently received a phosphodiesterase inhibitor, especially within 24 hours of sildenafil or vardenafil, or within 48 hours of tadalafil. | III: Harm | B |

Nitrates

- contraindications
  - hypotension, hypovolemia
  - increased ICP, constrictive pericarditis, pericardial tamponade
- tolerance can develop as soon as 12 hours after start of infusion
  - use lowest dose possible
  - alternate with other vasodilators
  - patch/oral nitrates – provide nitrate-free interval

IV NTG

- start at rate of 10 ug/min with nonabsorbing tubing
- increase by 10 ug/min q 3-5 min
- no response at 20 ug/min, increments of 10 and then 20 ug/min, ceiling of 200 ug/min
- Little benefit benefit beyond 48 hours
- pain free 12 hours, gradually reduce IV dose and change to oral or topical agents

- abrupt cessation of IV NTG associated with exacerbation of ischemic changes on EKG
- graded reduction in dose advised

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Beta-Blockers

**Effects of Metoprolol**

**COMMIT** (N = 45,852)

**Treaty of Evidence** (N = 52,411)

- **Increased early risk of shock**
- **Death 13%**
- **P=0.0006**
- **Re-MI 22%**
- **P=0.0002**
- **VF 15%**
- **P=0.002**

**Beta-Blockers**

- **Oral beta-blocker therapy should be administered promptly to those patients without a contraindication, irrespective of concomitant fibrinolytic therapy or performance of primary PCI.**

- **It is reasonable to administer IV beta-blockers promptly to STEMI patients without contraindications, especially if a tachyarrhythmia or hypertension is present.**

**Beta-Blockers**

- **IV beta-blockers should not be administered to STEMI patients who have any of the following: 1) signs of heart failure, 2) evidence of a low output state, 3) increased risk* for cardiogenic shock, or 4) other relative contraindications to beta blockade (PR interval > 0.24 sec, 2nd- or 3rd-degree heart block, active asthma, or reactive airway disease).**

---

**COMMIT: Study design**

**TREATMENT:** Metoprolol 15 mg iv over 15 mins, then 200 mg oral daily vs matching placebo

**INCLUSION:** Suspected acute MI (ST change or LBBB) within 24 h of symptom onset

**EXCLUSION:** Shock, systolic BP <100 mmHg, heart rate <50/min or II/III AV block

**1^ OUTCOMES:** Death & death, re-MI or VF/arrest up to 4 weeks in hospital (or prior discharge)

Mean treatment and follow-up: 16 days
Acute Coronary Syndromes: 
Unstable Angina and Non-ST-Segment Elevation Myocardial Infarction 
Kristine A. Scordo, PhD, RN, ACNP-BC FAANP

Factoids to consider.....

ACEI/ARB

ACEI/ARB

Ia

An ACE inhibitor should be administered orally within the first 24 h to UA/NSTEMI patients with pulmonary congestion or LV ejection fraction (LVEF) ≤ 40%, in the absence of hypotension (systolic blood pressure < 100 mm Hg or < 30 mm Hg below baseline) or known contraindications to that class of medications.

IIa

An angiotensin receptor blocker should be administered to UA/NSTEMI patients who are intolerant of ACE inhibitors and have either clinical or radiological signs of HF or LVEF ≤ 40%.

Ca++ Channel Blockers

used to control ongoing or recurring ischemia
• pts receiving adequate doses of nitrates and B-blockers
• pts unable to tolerate these agents
• pts with variant angina

nondihydropyridine (verapamil or diltiazem) in the absence of severe LV dysfunction or other contraindications

avoid rapid-release, short-acting nifedipine

Statin Therapy

High-intensity statin therapy should be initiated or continued in all patients with STEMI and no contraindications to its use.

It is reasonable to obtain a fasting lipid profile in patients with STEMI, preferably within 24 hours of presentation.

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Prior to Discharge:

**1st 24 hrs post MI**
- Total Chol drops
- LDL Drops
- TG Neutral

**HDL Increases 24 hrs post MI**

**Acute Phase Reactant**

Ryder et al Br Med J 1984

December 15; 299(6649): 1601–1603

---

**Exercise Testing: Class 1B**

Exercise testing should be performed either in the hospital or early after discharge in STEMI patients not selected for cardiac catheterization and without high risk features to assess the presence and extent of inducible ischemia. 2. In patients with baseline abnormalities that compromise

**Class III Exercise Testing**

Exercise testing should not be performed within 2 to 3 days of STEMI in patients who have not undergone successful reperfusion.

**Noninvasive Evaluation of Low-Risk Patients**
- graded exercise testing
  - assess functional capacity
  - evaluate efficacy of current medical regimen
  - risk-stratify post-MI patient
- low-level testing (done at 3 to 5 days post MI)
  - peak heart rate of 120 to 130 bpm
  - peak work level of 5 METs
  - clinical or EKG end points

**Risk Stratification after GXT**
- undergo coronary arteriography
  - signs of severe ischemia at low level of exercise
  - marked ST-segment change
  - inability to complete state I
  - failure to achieve 3 to 4 METs
  - paradoxical BP response
- achieve at least 5 METs: treat medically
Secondary Prevention

Smoking Cessation
- Smoking cessation reduces rates of reinfarction and death within a year of quitting
- 1/3 to 1/2 patients with AMI relapse within 6 to 12 months
- Nicotine gum and patches not recommended during hospitalization due to the sympathomimetic effects of nicotine

Long-Term Medications
- ASA 81 indefinitely
- Clopidogrel 75 mg qd / Prasugrel 10 mg qd / Ticagrelor 90 mg twice daily
- B-blockers
- Lipid-lowering agents
- AHA/ADA diet
- ACEIs/ARB for HF, LV dysfunction (EF <.40), HTN or DM
- Anticoagulants
  - For secondary prevention of MI in patients unable to take ASA (Plavix 75 mg qd)
  - Post-MI atrial fibrillation
  - Patients with LV thrombus

Long-Term Antithrombotic Therapy at Hospital Discharge after UA/NSTEMI

UA/NSTEMI Patient Groups at Discharge
- Medical Therapy without Stent
  - ASA 75 to 162 mg/d indefinitely (Class I, LOE: A)
  - Clopidogrel 75 mg/d at least 1 month (Class I, LOE: A)
- Bare-Metal Stent Group
  - ASA 162 to 325 mg/d for at least 1 month, then 75 to 162 mg/d indefinitely (Class I, LOE: A)
  - Clopidogrel 75 mg/d for at least 1 month and up to 1 year (Class I, LOE: B)
- Drug-Eluting Stent Group
  - ASA 162 to 325 mg/d for at least 3 to 6 months, then 75 to 162 mg/d indefinitely (Class I, LOE: A)
  - Clopidogrel 75 mg/d for at least 1 year (Class I, LOE: B)

Indication for Anticoagulation?
- Yes
- No
  - Continue with dual antiplatelet therapy as above
Influenza Vaccine

- Patients with cardiovascular disease should have an annual influenza vaccination (1-B)

Hormone Therapy

- Hormone therapy with estrogen plus progestin should not be given de novo to postmenopausal women after STEMI for secondary prevention of coronary events.

Hormone Therapy

- Postmenopausal women who are already taking estrogen plus progestin at the time of STEMI should not continue hormone therapy.

- However, women who are beyond 1 to 2 years after initiation of hormone therapy who wish to continue such therapy for another compelling indication should weigh the risks and benefits.

Antioxidants

- Antioxidant vitamins such as vitamin E and/or vitamin C supplements should not be prescribed to patients recovering from STEMI to prevent cardiovascular disease.

Psychosocial Impact of STEMI

- The psychosocial status of the patient should be evaluated, including inquiries regarding symptoms of depression, anxiety, or sleep disorders and the social support environment.

- Treatment with cognitive-behavioral therapy and selective serotonin reuptake inhibitors can be useful for STEMI patients with depression that occurs in the year after hospital discharge.

Cardiac Rehabilitation: Benefits

- Improve functional capacity
- Promote adherence to medical regimens
- Decrease emotional distress
- Improve quality of life
- Reduce cardiovascular mortality
- Mitigate ischemic symptoms
- Promote reversal of atherosclerosis
- Reduce risk of subsequent coronary events
- Decrease denial
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Return to Prior Levels of Activity
- uncomplicated MI return to work within 2 weeks
- driving can begin a week after discharge; must meet DMV standards
- air travel should be undertaken only by stable patients without a fear of flying within the first 2 weeks and then only as long as they travel with companions, carry NIT, and request airport transportation to avoid rushing

Sexual Counseling Post MI
- coital death is a nonverbalized fear of both patient and spouse; low incidence (0.6% of 559,000)
- increased ventricular arrhythmia when sexual activity is with an unfamiliar partner
- sexual intercourse is comparable in physiologic response to stair climbing
- no significant differences between positions using sustained isometric arm & shoulder muscle contraction (pt on top) & positions that do not use isometric exercise

Resumption of Sexual Activity
- can perform low-level exercise test
- climb two flights of stairs at a brisk rate
- pass the Master's test without undue increase in blood pressure, heart rate, or EKG changes
- uncomplicated MI, resume sexual activity with usual partner within a week to 10 days

Treatment
A = Aspirin and Antianginal therapy
B = Beta-blocker and BP
C = Cigarette smoking and Cholesterol
D = Diet and Diabetes
E = Education and Exercise
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Medical Record

- should indicate the discharge regime
- major instructions about post-discharge activities & rehabilitation
- patient's understanding and plan for adherence to the recommendations

Follow-up

- low-risk medically treated and revascularized pts should return in 2-6 weeks
- higher risk pts should return in 1-2 wks

Questions for follow-up

- Has the pt decreased level of physical activity since last ov?
- Have anginal symptoms increased in frequency?
- How well is pt tolerating therapy?
- How successful has pt been in modifying risk factors and improving knowledge about ischemic heart disease?
- Has pt developed any new comorbid illnesses or has severity or known comorbid illnesses worsened patient's angina?

General Teaching

- Avoiding factors that precipitate angina, i.e., cold weather, activities, stress
- Refill medications; don't abruptly stop medications
- Importance of follow-up visits
- Cardiac rehabilitation programs

References


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ACS Complications

Postinfarction Angina and Reinfarction
- chest pain that is frequently similar to the original discomfort, with or without transient ST-T changes and occurring 24 hours or more after the onset of AMI
- patients at high risk of postinfarction angina and reinfarction
  - non-Q-wave infarction
  - pts who received thrombolysis
  - pts with multiple risk factors

Postinfarction Angina
- ominous clinical development that must be aggressively treated
- higher mortality rates with EKG changes
- decrease oxygen demand
  - beta-blockers
  - nitrates
  - calcium-channel blockers
- pts usually undergo cardiac catheterization and PCI or CABG

Recurrent Chest Pain
- ischemia
  - more common
  - if within 12 hours after MI, usually related to viable tissue within infarct site
- pericarditis
  - MILIS trial: incidence 20%
  - associated with larger infarcts, lower EF & CHF

Pericarditis: Presentation
- positional discomfort
- pain radiates to left shoulder
- pericardial rub
- J-point elevation with concave upward ST-segment elevation and PR depression
- pericardial effusion on 2D in 40% cases- rarely of hemodynamic consequence
- not associated with re-elevation of CK-MB

Acute Pericarditis
Acute Coronary Syndromes:
Unstable Angina and Non-ST-Segment Elevation Myocardial Infarction
Kristine A. Scordo, PhD, RN, ACNP-BC FAANP

Pericarditis: Treatment
- ASA
- Class Ila Colchicine 0.6 mg q12 h or Acetaminophen 500 q6 h if not controlled on ASA
- Indomethacin
  - may cause increased coronary vascular resistance
  - experimentally causes thinning of developing scar
- Ibuprofen and corticosteroids
  - tendency for thinning of scar and myocardial rupture
- Evidence of impending cardiac tamponade is an indication for prompt termination of antithrombotic therapy

Right Ventricular AMI

Right Ventricular Infarction
- encompasses a spectrum of disease states ranging from asymptomatic mild right ventricular dysfunction through cardiogenic shock
- RV ischemia found in up to ½ of all inferior MIs; 10% to 15% patients show classical hemodynamic abnormalities
- associated with higher mortality than isolated IWMI

Clinical Diagnosis RV Infarction
- hypotension
- clear lung fields
- elevated jugular venous pressure
- Kussmaul's sign
- 1 mm ST-segment elevation in right precordial lead V4R is single most predictive EKG finding
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RVI: Physical Findings
- Elevated jugular venous pressure with Kussmaul’s
- Clear lung fields: decreased BP
- RV S₃ and/or S₄; pulsus paradoxus
- Murmur of tricuspid regurg from rt sided papillary
- Muscle dysfunction and/or chamber dilatation

RVI: Right Heart Cath
- Elevated right atrial mean pressure
- Elevated RVEDP
- Little if any elevation of RVSP
- PCWP usually normal or slightly elevated

RVI: Treatment
- Volume administration
- + inotropic agent
- Treat arrhythmias
- Use preload reducing agents cautiously
- Afterload reducing agents

Treatment of RV Infarction
- maintain RV preload
- reduce RV afterload
- inotropic support
- early reperfusion
- caution with nitrates and diuretics; may cause profound hypotension; can be reversed with volume loading with normal saline
- initiate inotropic support (dobutamine) if CO fails to improve after 1 to 2 L of fluid
- AV sequential pacing
- prompt cardioversion from atrial fibrillation

Arrhythmias
Acute Coronary Syndromes:  
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Atrial Fibrillation

- usually occurs within the first 24 hours and is usually transient, but may recur (10% to 16%)
- incidence higher with larger infarcts, CHF, complex ventricular arrhythmias, advanced AV block, atrial infarction, or pericarditis
- systemic embolization more frequent in patients with paroxysmal AF; most events occur by 4th day
- PR-segment displacement may predict risk of developing AF--AF associated with pericarditis

Ventricular Tachycardia/VF

- primary VF
  - incidence highest in the first 4 hours after AMI
  - associated with higher in-hospital mortality
  - pts who survive to hospital discharge have same long-term prognosis as pts who do not experience primary VF
- secondary VF
  - develops more than 48 hours after onset of MI
  - occurs in presence of severe CHF or cardiogenic shock

Arrhythmias During Acute Phase of STEMI: Electrical Instability

<table>
<thead>
<tr>
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<th>Treatment</th>
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<td>K⁺, Mg++, beta blocker</td>
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<tr>
<td>VT</td>
<td>Antiarrhythmics, DC shock</td>
</tr>
<tr>
<td>AIVR</td>
<td>Observe unless hemodynamic compromise</td>
</tr>
<tr>
<td>NPJT</td>
<td>Search for cause (e.g., dig toxicity)</td>
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VT/VF Treatment

- practice of prophylactic lidocaine abandoned
- beta-blocks shown to reduce incidence of early VF
- monitor electrolytes
- VF
  - unsynchronized shock of 200 J
  - ACLS protocol

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“Warning Arrhythmias”

Antman and Rutherford. Coronary Care Medicine.  
Acute Coronary Syndromes: Unstable Angina and Non-ST-Segment Elevation Myocardial Infarction
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Bradyarrhythmias and Heart Block
- sinus bradycardia 30% to 40% of patients - especially with reperfusion of RCA (increased parasympathetic activity-Bezold-Jarish reflex)
- heart block 6% to 14% of patients
  - increased risk of in-hospital mortality
  - poor predictor of long-term mortality
- new onset BBB 4%
  - associated with increased in-hospital mortality

Bradyarrhythmias: Treatment
- atropine
- transcutaneous pacing
  - painful temporary use
- transvenous pacing
  - asystolic
  - symptomatic bradycardia; type I second-degree AV block
  - bilateral BBB (alternating BBB or RBBB with alternating LAFB/LPFB)
  - new or indeterminate age bifascicular block with first-degree
  - Mobitz type II second-degree AV block

Atropine
- Class I
  - symptomatic bradycardia
- 0.5 to 1 mg IV, repeated q3 to 5 minutes to total dose of 2.5 mgm
- side effects
  - paradoxic effect
  - urinary retention
  - hallucinations

ICD Implantation After STEMI
One Month After STEMI; No Spontaneous VT or VF 48 hours post-STEMI
- EF < 0.30
- EF 0.31 - 0.40
- EF > 0.40

Additional Marker of Electrical Instability?
- Yes
- No

No ICD. Medical Rx

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Mechanical Defects

Ventricular Septal Rupture
- 1% to 3% AMI
- equal frequency between anterior and inferior infarctions
- higher prevalence in first infarctions
- majority occur within first week; rare after 2 weeks
- new harsh, holosystolic murmur LSB, often associated with a thrill
- sudden clinical deterioration with hypotension and pulmonary congestion

Free Wall Rupture

Ventricular Septal Rupture
- 1-2% 1-6%
- 3-5 d MI 3-5 d MI
- murm. 90% murm. 50%
- JVD, EMD JVD, EMD
- Common No
- Shunt Peric. Effusion
- Diast Press Equal. Regurg Jet
- c-v wave in PCW

Ventricular Septal Rupture
- step-up in oxygenation
- increase in oxygen saturation of >5% from right atrium to right ventricle
- 2D echocardiogram
- emergency surgical repair indicated when pulmonary edema or cardiogenic shock is present
- Amplatzer® Occluder

Acute Mitral Valve Regurgitation
- rupture of papillary muscle
  - ~1% MI
  - more frequent involvement of posteromedial papillary muscle with either RCY or less common CFX (reason uncommon with AMI)
  - sudden onset of pulmonary edema usually 2-7 days (medium rupture time 13 hours)
  - mid- or holosystolic murmur; thrill rare
- non-surgical treatment - 70% mortality
- surgical treatment associated with 27% - 55% mortality
- Support with IABP; nitrates; afterload reduction

Images: Courtesy of W.D. Edwards (Mayo Foundation)

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Images: Courtesy of W.D. Edwards (Mayo Foundation)
Cardiac Rupture

- Free wall of LV most common site of rupture
- Herald by chest pain and ST-T wave changes with rapid progression to hemodynamic collapse and electromechanical dissociation
- Frequency
  - Early peak within 24 hours
  - Late peak from 4-7 days after AMI (infarct expansion)
- Incidence higher with first MI, anterior MI, elderly and women
- Risk factors: hypertension during AMI, NSAIDS, steroids, Q-waves on EKG

Cardiogenic Shock

Other Complications

- Pulmonary embolism
  - 12% to 38%
  - Higher incidence with large infarctions in any location, anterior infarctions, CHF, reduced EF, immobilization
- Systemic emboli
  - Dislodgment of LV thrombi result in renal, mesenteric, cerebrovascular or other arterial systems
- Ventricular aneurysm

Shock

- Shock is a symptom of its cause
- Characterized by organ blood flow that is inadequate to meet tissue oxygen demands

Table 7. Clinical Profile of Mechanical Complications of Myocardial Infarction

<table>
<thead>
<tr>
<th>Variable</th>
<th>VSD Rupture</th>
<th>Free Wall Rupture</th>
<th>Papillary Muscle Rupture</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (mean, yr)</td>
<td>65</td>
<td>61</td>
<td>61</td>
</tr>
<tr>
<td>Days post MI</td>
<td>3.5</td>
<td>3-6</td>
<td>1.3</td>
</tr>
<tr>
<td>Anterior MI</td>
<td>16%</td>
<td>50%</td>
<td>25%</td>
</tr>
<tr>
<td>New reoccur</td>
<td>30%</td>
<td>25%</td>
<td>25%</td>
</tr>
<tr>
<td>Papillary Bulb</td>
<td>Yes</td>
<td>No</td>
<td>Rare</td>
</tr>
<tr>
<td>Previous MI</td>
<td>25%</td>
<td>21%</td>
<td>30%</td>
</tr>
</tbody>
</table>

Echocardiographic findings

<table>
<thead>
<tr>
<th>Two-dimensional</th>
<th>VSD</th>
<th>Free Wall</th>
<th>Papillary Muscle</th>
</tr>
</thead>
<tbody>
<tr>
<td>Doppler</td>
<td>VSD defect</td>
<td>Direct short</td>
<td>May have pericardial effusion</td>
</tr>
<tr>
<td>PA catheterization</td>
<td>Oxygen step-up in 16 RV</td>
<td>Equalization of diastolic pressure</td>
<td>Prominent V wave in PCW tracing</td>
</tr>
</tbody>
</table>

- Mortality
  - Medical: 90%
  - Surgical: 50%

Acute Coronary Syndromes:  
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Cardiogenic Shock  
- Systemic clinical syndrome characterized by hypotension and hypoperfusion  
- 5-9% STEMI; ~2.1% NSTEMI  
- Hypoperfusion at the tissue level causes cellular dysfunction  
- End result -- cellular injury and necrosis  

Cardiogenic Shock: Diagnostic Criteria  
- Clinical  
  - Hypotension with evidence of inadequate end-organ tissue perfusion due to cardiac pump failure  
- Hemodynamic  
  - Persistent hypotension (systolic blood pressure <90 mm Hg)  
  - Reduced cardiac index (<1.8 L/min/m²)  
  - Elevated LVEDP (>18 mm Hg)  

Usual Onset  
- Median time frame for development of cardiogenic shock is 12 hours into AMI  
- ~40% patients develop cardiogenic shock within 6 hours  
- ~63% develop cardiogenic shock within 24 hours  

Risk Factors  
- Four risk factors account for 85% of the predictive information needed to determine if a pt is at high risk for shock  
  - Age  
    - Single greatest risk factor; for every 10 year increase in age, the risk of developing shock increases by 47%  
  - Systolic BP  
  - HR  
  - Killip Class
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Killip classification.
- Stage I — No heart failure. No clinical signs of cardiac decompensation;
- Stage II — Heart failure. Diagnostic criteria include rales, S3 gallop and pulmonary venous hypertension. Pulmonary congestion with wet rales in the lower half of the lung fields;
- Stage III — Severe heart failure. Frank pulmonary oedema with rales throughout the lung fields;
- Stage IV — Cardiogenic shock. Signs include hypotension (SBP 90mmHg), and evidence of peripheral vasoconstriction such as oliguria, cyanosis and diaphoresis.

Cardiogenic Shock: Epidemiology
- develops in 5% to 15% of patients with AMI
- in-hospital mortality remains in excess of 80%
- mortality directly related to the duration and severity of shock
- 5-year mortality rate for those who survive the initial insult is 60%

Cardiogenic Shock: Causes
- Most frequent: LV failure following AMI
- Mechanical causes:
  - Acute severe mitral regurgitation
  - Ventricular septal rupture
  - Ventricular free-wall rupture
  - Cardiac tamponade
  - Acute stent thrombosis

Positive “Bag” Sign

Classification of Shock by Hemodynamic Profiles

<table>
<thead>
<tr>
<th>Type of Shock</th>
<th>Pulmonary Artery Occlusion Pressure</th>
<th>Cardiac Output</th>
<th>Systemic Vascular Resistance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiogenic Shock</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Hypovolemic Shock</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Distributive Shock</td>
<td>1 or normal</td>
<td>1 or normal of 1</td>
<td>1</td>
</tr>
<tr>
<td>Obstructive Shock</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>• Cardiac Tamponade</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Pulmonary embolus</td>
<td></td>
<td></td>
</tr>
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Low SVR = higher mortality rate
Acute Coronary Syndromes:
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Clinical Presentation
- most patients present with AMI and therefore present with constellation of s/s
- chest pain
- dyspnea
- diaphoresis
- nausea and vomiting
- pulmonary edema
- presyncopal or syncopal symptoms

Clinical Presentation
- altered mental status
- restlessness, agitation
- ashen, cool and clammy skin
- elevated JVD; crackles
- hypotension; narrow pulse pressure; tachycardia
- reduce urine output (<20ml/hr)
- reduced CI (<1.8L/min/m²) [2.5-3.5L/min/m²]
- PCWP >18mmHg [5-12mm Hg]

Clinical Presentation
- S3 or S4 gallop
- systolic murmur -- acute mitral regurgitation or ventricular septal rupture
- murmur + parasternal thrill -- VSD
- systolic murmur becomes louder on Valsalva --- hypertrophic obstructive cardiomyopathy (idiopathic hypertropic subaortic stenosis)
- muffled heart tones with JVD and pulsus paradoxus
  - suggest tamponade

Hypoperfusion: Clinical Presentation
- altered mental status
- reduced urine output (<20 ml/hr)
- cool clammy skin
  - absence of these clinical markers despite a systolic BP of 90 mm Hg or less, or a mean BP 30 mm Hg below baseline = hypotension but not overt shock
  - tissue hypoperfusion is key element in diagnosis of shock

Clues
- tachycardia or evidence of peripheral vasoconstriction early clinical signs of preshock state
- data from SPRINT study presence of
  - DM, hx angina, peripheral vascular or cerebrovascular disease, prior MI, female gender increases risk
  - 35% probability if all factors present

LCA with AMI in cardiogenic shock
LAD totally occluded; lesions at distal LM-proximal circumflex bifurcation; Note slow washout of contrast and sluggish flow in the aorta
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Clinical
- Cardiogenic shock is a medical emergency
- Perform complete clinical assessment
  - Critical to understand cause of shock and target of therapy to correct the cause
- Shock post AMI generally develops after admission to the hospital
  - Clinical evidence of low cardiac output
    - Sinus tachycardia, low urine output, cool extremities
    - Systemic hypotension
- Airway usually patent
- Hypoxemia - early ET
- Breathing may be labored
- May have audible coarse crackles or wheezing
- Poor peripheral pulses
- Initial VS: BP on both arms
  - No aortic dissection

Invasive Hemodynamic Monitoring
- PCWP >15 mm Hg
- CI <2.2 L/min/m²
- Large V waves
- Step-up in oxygen saturation between RA and RV diagnostic of ventricular septal rupture
- High right-sided filling pressures in absence of elevated PCWP with EKG criteria = RV infarctions

Cardiogenic Shock

Principles of Management
Emergency revascularization with either PCI or CABG is recommended in suitable patients with cardiogenic shock due to pump failure after STEMI irrespective of the time delay from MI onset.

In the absence of contraindications, fibrinolytic therapy should be administered to patients with STEMI and cardiogenic shock who are unsuitable candidates for either PCI or CABG.

PCI preferred over thrombolysis??

Hypotension limits penetration of lytics

Acidosis inhibits conversion of plasminogen to plasmin

Reasonable to give lytic if delay for PCI; combine with IABP

Devices (stay tuned)

Early Shock, Diagnosed on Hospital Presentation

Delayed Onset Shock Echocardiogram to Rule Out Mechanical Defects

Cardiac Catheterization and Coronary Angiography

IABP

PCI for Cardiogenic Shock

Primary PCI for STEMI: Specific Considerations

Primary PCI should be performed in patients less than 75 years old with ST elevation or LBBB who develop shock within 36 hours of MI and are suitable for revascularization that can be performed within 18 hours of shock.

Goal: Maximize Oxygenation

Increase O2 Delivery to Tissues

- improve myocardial function
- increase cardiac output and blood pressure
  - adjust preload
  - increase cardiac contractility
  - optimize SVR
  - assure adequate oxygenation of sufficient Hb
  - treat arrhythmias

Figure 2A: Kaplan-Meier survival of cardiogenic shock after early revascularization—cure/1-year postrandomization. Survival estimates for early revascularization (>102) and initial medical stabilization (>104) groups. Log-rank test P = .026. Reprinted with permission from Hochman et al. JAMA 2001;285:190-2. Copyright © 2001, American Medical Association. All rights reserved (104).
Improving Oxygen Delivery

- increase cardiac output
- increase hemoglobin concentration
  - increase Hb from 9 to 11.5 g/dL increases O₂ delivery by ~30%
- increase oxyhemoglobin saturation
  - increase PaO₂ from 60 to 90 torr increases SaO₂ from only 88% to 95%
  - once PaO₂ raised to the 60-70 torr range, little additional benefit of raising PaO₂ further

IV Fluids

- establish IV access ASAP
- relative hypovolemia is present in up to 20% of patients presenting with cardiogenic shock
- minimal signs of pulmonary congestion
  - administration of multiple small fluid boluses (50-100 cc) may be helpful
  - 100-200cc/hr fluid as initial treatment
- severe RV infarction
  - may require large volumes of multiple liters
  - Caution: septal shift into LV impairs LV filling; don’t flood pt
- assess lungs and PaSo2 during IV admin

Intraaortic Balloon Counterpulsation

- introduced in the late 1960s
- effective treatment for patients with unstable ischemic syndromes and cardiogenic shock
- NOT A DEFINITIVE THERAPY
- usually used as stabilizing device or bridge to facilitate diagnostic angiography & revascularization or repair
- reduce LV afterload
  - deflation of balloon in end diastole
- inflate in diastole
  - increase diastolic coronary artery & systemic perfusion

Treatment of Cardiogenic Shock

- fluid resuscitation to correct hypovolemia and hypotension (unless pulmonary edema present)
- treat underlying arrhythmias
- large-gauge venous access, central lines, PA line & oximetry
- correction of electrolyte and acid-base abnormalities
  - hypokalemia
  - hypomagnesemia
  - acidosis

The use of intra-aortic balloon pump counterpulsation can be useful for patients with cardiogenic shock after STEMI who do not quickly stabilize with pharmacological. Alternative LV assist devices for circulatory support may be considered in patients with refractory cardiogenic shock.
Indications

- Failure to wean from cardiopulmonary bypass.
- Cardiogenic shock.
- Heart failure.
- Acute heart attack.
- Support during high-risk percutaneous transluminal coronary (balloon) angioplasty, rotoblator procedures, and coronary stent placement.

Complications in up to 30% of patients: local vascular problems, emboli, infection, hemolysis.

Impact on long-term survival controversial.

Early insertion of IABP may result in clinical benefit rather than waiting until full-blown cardiogenic shock developed.

IABP Contraindications

- Aortic insufficiency
- Aortic aneurysm
- Severe peripheral vascular occlusive disease

TandemHeart

- Percutaneous ventricular assist device
- Comprised of a centrifugal blood pump
- System designed to provide both ventricular unloading and increased systemic perfusion
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**ECMO Complications**
- Bleeding
- Intracranial
- Cannulation and other sites
- Lower extremity ischemia
- Infection/sepsis
- Renal failure
- Hemolysis
- Coagulopathy

**Conclusions**
- Early institution of ECMO in the cath lab in patients with MI, cardiogenic shock, repeated cardiac arrest and severe hemodynamic instability refractory to inotropic agents and IABP may provide prompt circulatory support.
- ECMO may serve as a bridge to heart transplantation to salvage those patients who otherwise have a high mortality rate.

**Ventricular Assist Devices**
- devices function as prosthetic ventricles but most require sternotomy for insertion
- support left ventricular performance, RV performance or a combination
- indications for insertion controversial

**Cardiogenic Shock**
**Pharmacologic Therapy**

- LVAD used as bridge to cardiac transplantation
  - allows survival to transplant in 75% or 29 patients
- Hemopump
  - transvalvular LV assist device that provides larger increase in CO than IABP
  - circumvents problem associated with median sternotomy and allows percutaneous placement of canula across aortic valve, which is coupled to an extracorporeal power source
  - major complications ventricular arrhythmias & embolic phenomenon
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Analgesics
- Morphine sulfate
  - 0.1 mg/kg/IV
  - maintenance dose
    - 5-20 mg/70kg IV q4h
    - relatively hypovolemic patients start with 2 mgm IV, reassess hemodynamic effects of the dose

Dopamine (Intropin)
- Most frequently used inotropic/vasopressor agent—US wedded to Dopamine—Europe to norepinephrine
- Dopaminergic response: 2-4 mcg/kg/min
  - modest inotropic and chronotropic effects
  - acts on dopaminergic receptors in kidney
    - increase renal blood flow
- Beta-adrenergic response: 5-10 mcg/kg/min
  - primarily inotropic effect; loses kidney effect
- Adrenergic response: 10-20 mcg/kg/min
  - alpha-agonist effects
  - dose related vasoconstriction

Dobutamine (Dobutrex)
- β-adrenergic agonist
- doses of 5-20 μg/kg/min
- associated with increased in cardiac output
- arterial BP may remain unchanged or slightly increase
- need adequate circulating blood volume
- less arrhythmogenic than dopamine, however arrhythmias still occur

Phosphodiesterase enzyme inhibitors
- Milrinone (Primacor)
  - positive inotropic & vasodilator with little chronotropic activity
  - 50 mcg/kg IV loading dose over 10 min followed by 0.375-0.75 mcg/kg/min continuous IV infusion
- Amrinone (Inocor)
  - positive inotropic and vasodilator activity; more likely to cause tachycardia than dobutamine
  - 0.75 mg/kg IV bolus over 2-3 minutes
  - 5-10 mcg/kg/min

Diuretics
- immediate impact of IV diuretics (eg. furosemide) is vasodilation and reduction in preload
  - 20-80 mg IV
  - preload reduction can benefit pulmonary congestion
  - may lead to decrease in LV stroke volume & CO
  - patients with diastolic dysfunction (hypertrophic cardiomyopathy) and certain valvular outflow obstruction (aortic or mitral) are particularly sensitive to decreases in preload

Vasodilators
- in setting of depressed ventricular function, afterload is major determinant of CO
- afterload reduction: increase SV and CO, reduce MVO₂
- nitroprusside and IV nitroglycerine
- caution: excessive hypotension
- usually require hemodynamic monitoring
Oliguria

- Important marker of hypoperfusion
- Defined as urine output <0.5 mL/kg/hr for ≥ 2hrs
- May also be due to inherent renal injury or postrenal causes
  - Prevents use of urine output as a target of adequate resuscitation of shock
- Urine output should be maintained between 0.5 and 1 mL/kg/hr
- Cardiogenic shock (hypotension) most common cause of pre-renal failure

Prerenal vs. Acute Tubular Necrosis

<table>
<thead>
<tr>
<th>Indicator</th>
<th>Prerenal</th>
<th>ATN</th>
</tr>
</thead>
<tbody>
<tr>
<td>BUN/creatinine ratio</td>
<td>&gt;20</td>
<td>10-20</td>
</tr>
<tr>
<td>Urine-SP</td>
<td>&gt;1.020</td>
<td>&lt;1.010</td>
</tr>
<tr>
<td>Urine osmolality (mOsm/L)</td>
<td>&gt;500</td>
<td>&lt;350</td>
</tr>
<tr>
<td>Urinary sodium (mEq/L (mmol/L))</td>
<td>&lt;20 [&lt;20]</td>
<td>&gt;40 [&gt;40]</td>
</tr>
<tr>
<td>Fractional excretion of sodium (%)</td>
<td>&lt;1</td>
<td>&gt;2</td>
</tr>
</tbody>
</table>

Prevention is the Key to Survival

- Identify patients at high risk for development of cardiogenic shock
  - #1 predictor is age >75 years
- Problem: 70% of pts who developed cardiogenic shock after admission were Killip class I on admission
- Need other clinical clues