

## **Unstable Angina and Non-ST Elevation Myocardial Infarction:**

**Diagnostic and Therapeutic Management Based  
on Current Knowledge and Clinical Judgment**

**Konstantinos Dean Boudoulas, MD**  
Associate Professor of Medicine  
Section Head, Interventional Cardiology  
Director, Cardiac Catheterization Laboratory  
The Ohio State University Wexner Medical Center

## **Unstable Angina (UA) and Non-ST Elevation Myocardial Infarction (NSTEMI)**

- I. Pathophysiologic Mechanisms**
- II. Diagnosis**
- III. Management**
- IV. Prevention**

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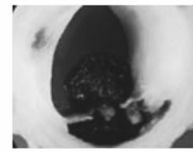
## **Common Pathophysiologic Mechanisms**

- UA and NSTEMI are acute coronary syndromes (ACS) characterized as a general rule by a significant decrease in blood supply to the myocardium.
- Most common cause for the decrease in myocardial perfusion is by a non-occlusive thrombus (with potential distal embolization) that has developed on a disrupted atherosclerotic plaque resulting in luminal narrowing.
- UA and NSTEMI pathogenesis and clinical presentations are similar differing in severity with NSTEMI resulting in myocardial damage releasing detectable quantities of a marker of myocardial injury.

## Less Common Causes of UA/NSTEMI

- Occlusive thrombus with collateral vessels
- Non-plaque thromboembolism (atrial fibrillation; LV thrombus)
- Dynamic obstruction (coronary spasm; vasoconstriction)
- Coronary arterial inflammation
- Coronary artery dissection
- Mechanical obstruction to coronary flow
- Hypotension, tachycardia, anemia, other

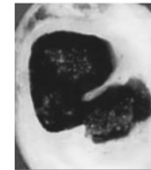
## Acute Coronary Syndromes (ACS)



ECG:

No ST Elevation

↓  
Unstable  
Angina



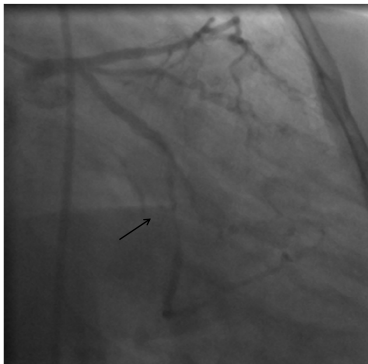
ST Elevation

↓  
NSTEMI  
(Non-Q wave MI)

↓  
STEMI  
(Q wave MI)

Modified from Anderson JL, et al. JACC. 2007;50:e1-e157.

## Non ST-Elevation Myocardial Infarction Left Circumflex Artery Occlusion



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## Clinical Presentation

- Chest pain or severe epigastric pain typical of myocardial ischemia or infarction:
  - Chest pressure, tightness, heaviness, cramping, burning, aching sensation
  - Unexplained indigestion, belching, epigastric pain
  - Radiating pain in neck, jaw, shoulders, back, or arm(s)
- Associated dyspnea, nausea/vomiting or diaphoresis

## Electrocardiogram

- ST segment depression
  - 1 mm  $\geq$  2 contiguous leads
- T-wave inversion

## Cardiac Biomarkers

- Troponin I or T (most sensitive/specific)
- CK, CK-MB
- Myoglobin
- Other

## Guidelines/Level of Evidence

Class I	Class IIa	Class IIb	Class III
<i>Benefit &gt;&gt;&gt; Risk</i>	<i>Benefit &gt;&gt; Risk</i>	<i>Benefit <math>\geq</math> Risk</i>	<i>Risk <math>\geq</math> Benefit</i>
<b>SHOULD</b> be performed	<b>REASONABLE</b> to perform	<b>MAY BE CONSIDERED</b>	<b>NOT</b> be performed <b>SINCE IT IS NOT HELPFUL AND MAY BE HARMFUL</b>

**Level A:** Recommendation based on multiple randomized trials or meta-analyses

**Level B:** Recommendation based on single randomized trial or non-randomized studies

**Level C:** Recommendation based on expert opinion, case studies, or standard-of-care

Modified from Wright RS, et al. JACC. 2011;57:1920-59.

## Electrocardiogram

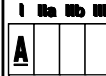


• A 12-lead ECG should be performed with a goal of within 10 min of arrival

• Initial ECG is not diagnostic, serial ECGs at 15- to 30-min intervals

2014 AHA/ACC NSTEMI Guideline. JACC. 2014;64:e139-228.  
2014 AHA/ACC NSTEMI Guideline. Circulation. 2014;130:2354-94.

## Cardiac Biomarkers



• Serial cardiac troponin I or T levels should be obtained at presentation and 3 to 6 hours after symptom onset

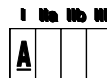
• Additional troponin levels should be obtained beyond 6 hours after symptom onset in patients with normal troponin levels on serial examination with suspicion for ACS

2014 AHA/ACC NSTEMI Guideline. JACC. 2014;64:e139-228.  
2014 AHA/ACC NSTEMI Guideline. Circulation. 2014;130:2354-94.

## Unstable Angina (UA) and Non-ST Elevation Myocardial Infarction (NSTEMI)

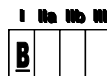
- I. Pathophysiologic Mechanisms
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## Initial Anti-Platelet Therapy



Aspirin 162 mg to 325 mg

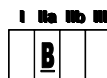
and



*Platelet P2Y12 Receptor Antagonists:*

Clopidogrel 300 or 600 mg or

Ticagrelor 180 mg



Ticagrelor in preference to Clopidogrel

2014 AHA/ACC NSTEMI Guideline. JACC. 2014;64:e139-228.  
2014 AHA/ACC NSTEMI Guideline. Circulation. 2014;130:2354-94.

## Initial Anti-Platelet Therapy



GP IIb/IIIa inhibitor in patients treated with dual anti-platelet therapy with intermediate/high-risk features (e.g., positive troponin); preferred options are eptifibatide or tirofiban

2014 AHA/ACC NSTEMI Guideline. JACC. 2014;64:e139-228.  
2014 AHA/ACC NSTEMI Guideline. Circulation. 2014;130:2354-94.

## GP IIb/IIIa Inhibitor Upstream vs. Time of Angiogram

- ACUITY Timing Trial<sup>1</sup> (n=9207)
  - No difference in ischemia end-points
  - 30-day  major bleeding in upstream (6.1%) vs. deferred (4.9%)
- EARLY ACS<sup>2</sup> (n=9492)
  - No difference in ischemia end-points
  - 5 day  non-life-threatening bleeding & transfusion with upstream

<sup>1</sup>Stone GW, et al. JAMA. 2007;297:591-602.  
<sup>2</sup>Giugliano RP, et al. NEJM. 2009; 360:2176-90.

## Anti-Coagulation



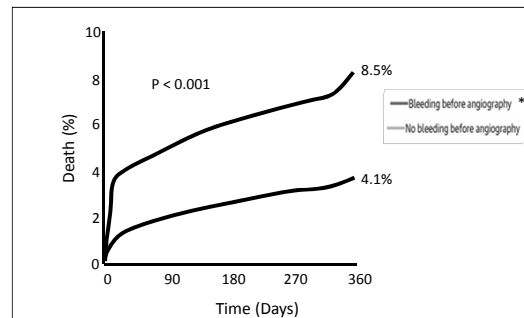
- Enoxaparin
  - continued for duration of hospitalization or until PCI performed



- Unfractionated heparin
  - continued for 48 hours or until PCI performed
- Bivalirudin
  - only with early invasive strategy

2014 AHA/ACC NSTEMI Guideline. JACC. 2014;64:e139-228.  
2014 AHA/ACC NSTEMI Guideline. Circulation. 2014;130:2354-94.

## Bleeding Event Before Coronary Angiography and Death In Patients with NSTEMI

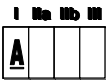


\*More likely to have received:

- low-molecular-weight heparin (less likely bivalirudin)
- upstream P2Y<sub>12</sub> or GPIIb/IIIa inhibitors

Redfors B, et al. J Am Coll Cardiol. 2016;68:2608-18.

## Beta-Blocker Therapy

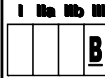


Oral beta-blocker therapy should be initiated within the first 24 h for patients who do not have 1 or more of the following:

1. signs of heart failure
2. evidence of a low-output state
3. increased risk for cardiogenic shock\*
4. other relative contraindications (PR interval >0.24 s, 2<sup>nd</sup> or 3<sup>rd</sup> degree AV block, active asthma/reactive airway disease)

\* > 70 years, SBP < 120 mmHg, heart rate >100 or < 60 bpm

## Beta-Blocker Therapy



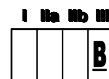
Administration of intravenous beta blockers is potentially harmful in patients with NSTEMI who have risk factors for shock

2014 AHA/ACC NSTEMI Guideline. JACC. 2014;64:e139-228.  
2014 AHA/ACC NSTEMI Guideline. Circulation. 2014;130:2354-94.

## Coronary Angiogram Management Options

- Medical therapy
- Coronary revascularization
  - Percutaneous coronary intervention (PCI)
  - Coronary artery bypass surgery
  - Hybrid procedure (LIMA to LAD and PCI to all other vessels)

## Anemia and Transfusion



A strategy of routine blood transfusion in hemodynamically stable patients with ACS and hemoglobin levels > 8 g/dL is not recommended

2014 AHA/ACC NSTEMI Guideline. JACC. 2014;64:e139-228.  
2014 AHA/ACC NSTEMI Guideline. Circulation. 2014;130:2354-94.

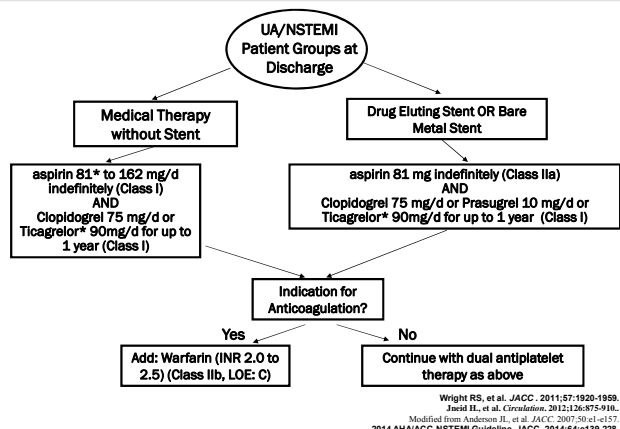
## Unstable Angina (UA) and Non-ST Elevation Myocardial Infarction (NSTEMI)

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## Prevention

- Medical therapy
  - Anti-platelet
  - Statin
  - Beta-blocker
  - ACE inhibitor
- Management of other diseases (HTN, DM, etc)
- Exercise and Diet
- Tobacco cessation
- Other

### Long-Term Anti-Platelet Therapy at Discharge



### Platelet P2Y12 Receptor Antagonists

	Plavix (Clopidogrel)	Effient (Prasugrel)	Brilinta (Ticagrelor)
Loading Dose	600 mg	60 mg (peak effect 2-4h)	180 mg (peak effect 2h)
Maintenance Dose	75 mg daily	10 mg daily	90 mg twice daily
Max % of Platelet Inhibition	30-50%	75-80%	75-80%
Time to 50% Inhibition	2-4 hours	Within 1 hour	Within 30-60 mins
Contraindications	•Active bleeding	•TIA or stroke •Intracranial hemorrhage •Active bleeding	•Intracranial hemorrhage •Severe hepatic impairment •Active bleeding

## Lipid Management

- 2013 ACC/AHA Guideline on Treatment of Blood Cholesterol
  - high intensity statin therapy (atorvastatin 40/80 mg or rosuvastatin 20 mg)

## Beta-Blocker Therapy

- Beta blockers are indicated for all patients recovering from UA/NSTEMI especially with LV systolic dysfunction unless contraindicated

2014 AHA/ACC NSTEMI Guideline. JACC. 2014;64:e139-228.  
 2012 ACCF/AHA UA/NSTEMI Guidelines. Circulation. 2012;126:875-910.  
 2011 ACCF/AHA UA/NSTEMI Guidelines. Circulation. 2011;123:e426-e579.

## ACE-Inhibitor

I	IIa	IIb	III
A			

ACE inhibitors should be given and continued indefinitely for patients with LVEF <40%, hypertension, diabetes mellitus, or stable chronic kidney disease

I	IIa	IIb	III
			B

ACE inhibitors may be reasonable in all other patients with cardiac or other vascular disease

2014 AHA/ACC NSTEMI Guideline. JACC. 2014;64:e139-228.  
 2014 AHA/ACC NSTEMI Guideline. Circulation. 2014;130:2354-94.

## Heart Outcomes Prevention Evaluation HOPE Trial

- Patients with CAD or high-risk of developing CAD (n=9,297)
  - 52% prior MI, 25% UA
- No LV dysfunction or heart failure
- Ramipril 10 mg/day vs placebo
- Primary end point (myocardial infarction, stroke, or CV death):
  - 14.0% ramipril vs 17.8% placebo ( p<0.001)
  - statistically lower for all individual endpoints

Yusuf S, et al. *N Engl J Med* 2000;342:145-53



## Aldosterone Blockade



Aldosterone blockade recommended in patients without significant renal dysfunction or hyperkalemia who are receiving therapeutic doses of ACE inhibitor and beta blocker, and have a LVEF  $\leq$  40%, diabetes mellitus, or heart failure

2014 AHA/ACC NSTEMI Guideline. JACC. 2014;64:e139-228.  
2014 AHA/ACC NSTEMI Guideline. Circulation. 2014;130:2354-94.

## Avoid NSAIDs and Estrogen/Progestin Replacement Therapy

- Increase risk of myocardial infarction and death

Hulley S, et al. JAMA 1998;280:605-13.  
Antman EM, et al. Circulation. 2007;115:1634-42.

## Unstable Angina (UA) and Non-ST Elevation Myocardial Infarction (NSTEMI) Conclusion

- Most commonly caused by a decrease in myocardial perfusion by a non-occlusive thrombus that has developed on a disrupted atherosclerotic plaque resulting in luminal narrowing.
- Coronary angiogram should be performed to define coronary anatomy and need for coronary artery revascularization.
- Medical therapy should include aspirin, P2Y<sub>12</sub> receptor antagonist,  $\beta$ -blocker, ACE inhibitor (especially with LVEF  $<$ 40%, hypertension, diabetes mellitus, or stable chronic kidney disease) and statin, regardless if revascularization performed.

## Unstable Angina (UA) and Non-ST Elevation Myocardial Infarction (NSTEMI) Conclusion

- Coronary artery disease is progressive requiring close follow-up with particular attention to modifying risk factors:
  - smoking cessation, obesity, hypertension, dyslipidemia, diabetes mellitus, avoidance of NSAID and hormone replacement therapy, other

# ST Elevation Myocardial Infarction

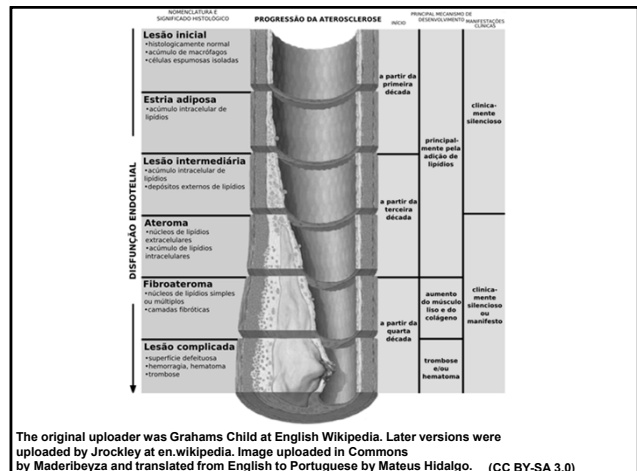
Cindy Baker, MD, FACC  
 Clinical Assistant Professor  
 Division of Cardiovascular Medicine  
 Director of Peripheral Vascular Intervention  
 The Ohio State University Wexner Medical Center

# Objectives

- Definition
- Statistics
- Reperfusion Strategies
- Drug Therapy
- Complications to Consider

# STEMI- Definition

- new ST elevation at the J point in at least 2 contiguous leads of 2 mm in men
- 1.5 mm in women in leads V2–V3 and/or of 1 mm in other contiguous chest leads or the limb leads
- New or presumably new LBBB maybe considered a STEMI equivalent.
- ST depression in 2 precordial leads (V1–V4) may indicate posterior STEMI



The original uploader was Grahams Child at English Wikipedia. Later versions were uploaded by Jrockley at en.wikipedia. Image uploaded in Commons by Maderibeyza and translated from English to Portuguese by Mateus Hidalgo. (CC BY-SA 3.0)

## Statistics

- STEMI comprises 25-40% of myocardial infarction presentations
- In-hospital mortality 5-6%
- One year mortality 7-18% has significantly decreased with appropriate care including primary PCI and GDMT

Benjamin EJ, et al. Heart Disease and Stroke Statistics 2017 Update, Report From The AHA

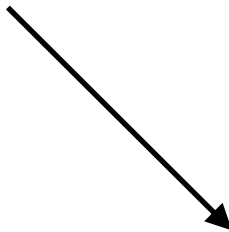
## Statistics

- Approximately 30% of patients with STEMI are women. Female sex is a strong independent predictor of failure to receive reperfusion therapy.
- Non-whites represent 13.3% of patients with STEMI. Disparities in care of racial and ethnic minorities appears to be improving over time.

Benjamin EJ, et al. Heart Disease and Stroke Statistics 2017 Update, Report From The AHA

## Incidence of STEMI

133 per 100 000  
person-years  
in 1999



50 per 100 000  
person years  
in 2008

Yeh RW et al. N Engl J Med 2010;362:2155-2165

## Case Presentation

55 year old male presents to an OSH with 6 hour history of chest pain. No significant past medical history. The hospital does not have PCI capability.

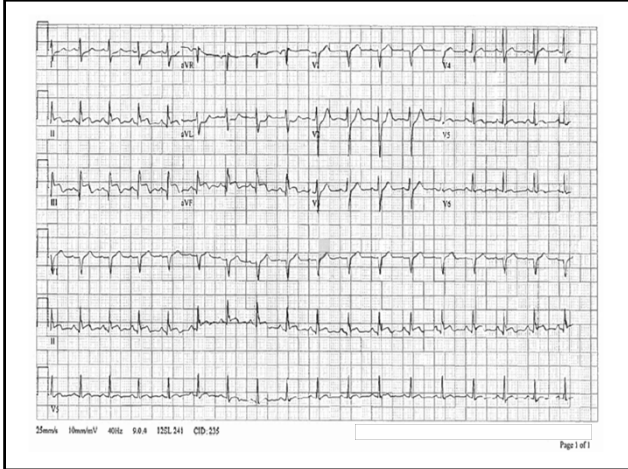
### Vital Signs:

HR 104 BP 95/60 RR 16

Cardiovascular: RRR no murmurs appreciated

Lungs: bibasilar crackles

Extremities: cool with equal pulses



## Questions to Consider??

- Reperfusion options
- Medical therapy
- Potential Post-MI complications

## Reperfusion Options

Patients with STEMI who are candidates for reperfusion therapy

Initially seen at a PCI capable hospital

Initially seen at a non-PCI capable hospital

## Reperfusion Options

Initially seen at a PCI capable hospital

Send to cath lab for primary PCI  
FMC-device time  $\leq$  90 mins

## Reperfusion Options

Initially seen at a non-PCI capable hospital  
(DIDO time  $\leq$  30 mins)

Transfer for primary PCI FMC-device time as soon as possible and  $\leq$ 120 mins

Administer fibrinolytic agent within 30 minutes of arrival when anticipated FMC-device  $>$ 120mins

## Primary PCI in STEMI

0	COR	LOE
Ischemic symptoms 12 h	I	A
Ischemic symptoms 12 h and contraindications to fibrinolytic therapy irrespective of time delay from FMC	I	B
Cardiogenic shock or acute severe HF irrespective of time delay from MI onset	I	B
Evidence of ongoing ischemia 12 to 24 h after symptom onset	IIa	B
PCI of a noninfarct artery at the time of primary PCI in patients without hemodynamic compromise	III: Harm	B

O'Gara PT, et al. Journal of the American College of Cardiology Jan 2013, 61 (4) e78-e140; DOI: 10.1016/j.jacc.2012.11.019

## Indications for Transfer for Angiography After Fibrinolytic Therapy

	COR	LOE
Immediate transfer for cardiogenic shock or severe acute HF irrespective of time delay from MI onset	I	B
Urgent transfer for failed reperfusion or reocclusion	IIa	B
As part of an invasive strategy in stable* patients with PCI between 3 and 24 h after successful fibrinolysis	IIa	B

\*Although individual circumstances will vary, clinical stability is defined by the absence of low output, hypotension, persistent tachycardia, apparent shock, high-grade ventricular or symptomatic supraventricular tachyarrhythmias, and spontaneous recurrent ischemia.

O'Gara PT, et al. Journal of the American College of Cardiology Jan 2013, 61 (4) e78-e140; DOI: 10.1016/j.jacc.2012.11.019

## Indications for Coronary Angiography in Patients Who Were Managed With Fibrinolytic Therapy or Who Did Not Receive Reperfusion Therapy

	COR	LOE
Cardiogenic shock or acute severe HF that develops after initial presentation	I	B
Intermediate- or high-risk findings on pre-discharge noninvasive ischemia testing	I	B
Spontaneous or easily provoked myocardial ischemia	I	C
Failed reperfusion or reocclusion after fibrinolytic therapy	IIa	B
Stable* patients after successful fibrinolysis, before discharge and ideally between 3 and 24 h	IIa	B

O'Gara PT, et al. Journal of the American College of Cardiology Jan 2013, 61 (4) e78-e140; DOI: 10.1016/j.jacc.2012.11.019

### Indications for PCI of an Infarct Artery in Patients Who Were Managed With Fibrinolytic Therapy or Who Did Not Receive Reperfusion Therapy

	COR	LOE
Cardiogenic shock or acute severe HF	I	B
Intermediate- or high-risk findings on predischarge noninvasive ischemia testing	I	C
Spontaneous or easily provoked myocardial ischemia	I	C
Patients with evidence of failed reperfusion or reocclusion after fibrinolytic therapy (as soon as possible)	IIa	B
Stable* patients after successful fibrinolysis, ideally between 3 and 24 h	IIa	B
Stable* patients 24 h after successful fibrinolysis	IIb	B
Delayed PCI of a totally occluded infarct artery 24 h after STEMI in stable patients	III: No Benefit	B

\*Although individual circumstances will vary, clinical stability is defined by the absence of low output, hypotension, persistent tachycardia, apparent shock, high-grade ventricular or symptomatic supraventricular tachyarrhythmias, and spontaneous recurrent ischemia. Journal of the American College of Cardiology Jan 2013, 61 (4) e78-e140; DOI: 10.1016/j.jacc.2012.11.019

### Adjunctive Antithrombotic Therapy to Support Reperfusion With Primary PCI

	COR	LOE
<b>Antiplatelet therapy</b>		
<b>Aspirin</b>		
• 162- to 325-mg load before procedure	I	B
• 81- to 325-mg daily maintenance dose (indefinite)*	I	A
• 81 mg daily is the preferred maintenance dose*	IIa	B
<b>P2Y12 inhibitors</b>		
<b>Loading doses</b>		
• Clopidogrel: 600 mg as early as possible or at time of PCI	I	B
• Prasugrel: 60 mg as early as possible or at time of PCI	I	B
• Ticagrelor: 180 mg as early as possible or at time of PCI	I	B

\*The recommended maintenance dose of aspirin to be used with ticagrelor is 81 mg daily.

O'Gara PT, et al. Journal of the American College of Cardiology Jan 2013, 61 (4) e78-e140; DOI: 10.1016/j.jacc.2012.11.019

### A Platelet Inhibition and Patient Outcomes (PLATO) Trial

- Randomized controlled trial comparing ticagrelor to clopidogrel
- STEMI substudy showed ticagrelor had significant reduction in myocardial infarction, total mortality and definite stent thrombosis over clopidogrel.
- There was a low stroke rate but it was significantly higher in the ticagrelor group (1.7% vs 1%) p0.02

Philippe Gabriel Steg et al. Circulation. 2010;122:2131-2141

### Adjunctive Antithrombotic Therapy to Support Reperfusion With Primary PCI (cont.)

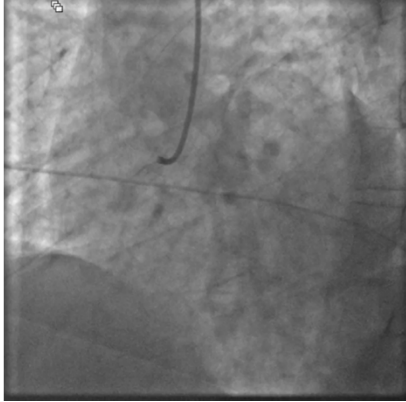
	COR	LOE
<b>Anticoagulant therapy</b>		
• UFH:	I	C
• With GP IIb/IIIa receptor antagonist planned: 50- to 70-U/kg IV bolus to achieve therapeutic ACT‡		
• With no GP IIb/IIIa receptor antagonist planned: 70- to 100-U/kg bolus to achieve therapeutic ACT§	I	C
• Bivalirudin: 0.75-mg/kg IV bolus, then 1.75-mg/kg/h infusion with or without prior treatment with UFH. An additional bolus of 0.3 mg/kg may be given if needed.	I	B
• Reduce infusion to 1 mg/kg/h with estimated CrCl 30 mL/min		
• Preferred over UFH with GP IIb/IIIa receptor antagonist in patients at high risk of bleeding	IIa	B
• Fondaparinux: not recommended as sole anticoagulant for primary PCI	III: Harm	B

‡The recommended ACT with planned GP IIb/IIIa receptor antagonist treatment is 200 to 250 s.

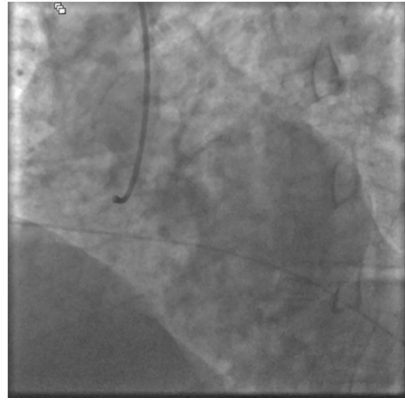
§The recommended ACT with no planned GP IIb/IIIa receptor antagonist treatment is 250 to 300 s (HemoTec device) or 300 to 350 s (Hemochron device).

O'Gara PT, et al. Journal of the American College of Cardiology Jan 2013, 61 (4) e78-e140; DOI: 10.1016/j.jacc.2012.11.019

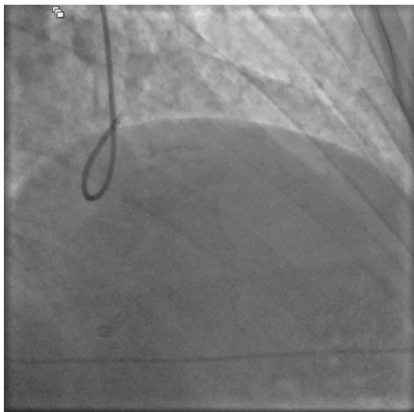
**STEMI RCA pre**



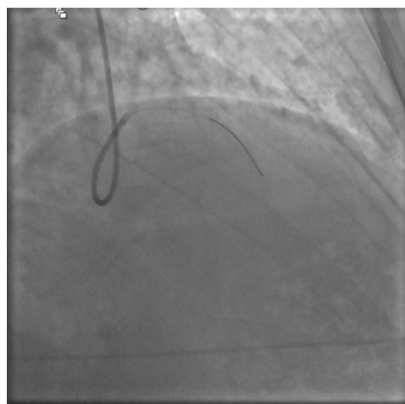
**STEMI RCA post**



**STEMI LAD pre**



**STEMI LAD post**



### Adjunctive Antithrombotic Therapy to Support Reperfusion With Primary PCI (cont.)

	COR	LOE
<b>P2Y12 inhibitors</b>		
<b>Maintenance doses and duration of therapy</b>		
<i>DES placed: Continue therapy for 1 y with:</i>		
• Clopidogrel: 75 mg daily	I	B
• Prasugrel: 10 mg daily	I	B
• Ticagrelor: 90 mg twice a day*	I	B
<i>BMS† placed: Continue therapy for 1 y with:</i>		
• Clopidogrel: 75 mg daily	I	B
• Prasugrel: 10 mg daily	I	B
• Ticagrelor: 90 mg twice a day*	I	B
<i>DES placed:</i>		
• Clopidogrel, prasugrel, or ticagrelor* continued beyond 1 y	IIb	C
• Patients with STEMI with prior stroke or TIA: prasugrel	III: Harm	B

\*The recommended maintenance dose of aspirin to be used with ticagrelor is 81 mg daily.  
†Balloon angioplasty without stent placement may be used in selected patients. It might be reasonable to provide P2Y<sub>12</sub> inhibitor therapy to patients with STEMI undergoing balloon angioplasty alone according to the recommendations listed for BMS. (LOE: C).

O'Gara PT, et al. Journal of the American College of Cardiology Jan 2013, 61 (4) e78-e140; DOI: 10.1016/j.jacc.2012.11.019

## Beta Blockers

I IIa IIb III

B			
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Oral beta blockers should be initiated in the first 24 hours in patients with STEMI who do not have any of the following: signs of HF, evidence of a low output state, increased risk for cardiogenic shock,\* or other contraindications to use of oral beta blockers (PR interval >0.24 seconds, second- or third-degree heart block, active asthma, or reactive airways disease).

I IIa IIb III

B			
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Beta blockers should be continued during and after hospitalization for all patients with STEMI and with no contraindications to their use.

**\*Risk factors for cardiogenic shock (the greater the number of risk factors present, the higher the risk of developing cardiogenic shock) are age >70 years, systolic BP <120 mm Hg, sinus tachycardia >110 bpm or heart rate <60 bpm, and increased time since onset of symptoms of STEMI.**

O'Gara PT, et al. Journal of the American College of Cardiology Jan 2013, 61 (4) e78-e140; DOI: 10.1016/j.jacc.2012.11.019

## Beta Blockers

I IIa IIb III

C			
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Patients with initial contraindications to the use of beta blockers in the first 24 hours after STEMI should be reevaluated to determine their subsequent eligibility.

I IIa IIb III

B			
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It is reasonable to administer intravenous beta blockers at the time of presentation to patients with STEMI and no contraindications to their use who are hypertensive or have ongoing ischemia.

O'Gara PT, et al. Journal of the American College of Cardiology Jan 2013, 61 (4) e78-e140; DOI: 10.1016/j.jacc.2012.11.019

## Renin-Angiotensin-Aldosterone System Inhibitors

I IIa IIb III

A			
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An ACE inhibitor should be administered within the first 24 hours to all patients with STEMI with anterior location, HF, or EF less than or equal to 0.40, unless contraindicated.

I IIa IIb III

B			
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An ARB should be given to patients with STEMI who have indications for but are intolerant of ACE inhibitors.

O'Gara PT, et al. Journal of the American College of Cardiology Jan 2013, 61 (4) e78-e140; DOI: 10.1016/j.jacc.2012.11.019



## Renin-Angiotensin-Aldosterone System Inhibitors



An aldosterone antagonist should be given to patients with STEMI and no contraindications who are already receiving an ACE inhibitor and beta blocker and who have an EF less than or equal to 0.40 and either symptomatic HF or diabetes mellitus.



ACE inhibitors are reasonable for all patients with STEMI and no contraindications to their use.

O'Gara PT, et al. Journal of the American College of Cardiology Jan 2013, 61 (4) e78-e140; DOI: 10.1016/j.jacc.2012.11.019

## Lipid Management



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.

O'Gara PT, et al. Journal of the American College of Cardiology Jan 2013, 61 (4) e78-e140; DOI: 10.1016/j.jacc.2012.11.019

## Post STEMI Complications

- Cardiogenic shock
- Congestive heart failure
- Infarct expansion/ recurrent ischemia
- Arrhythmias- tachy and brady
- Pericarditis (Dressler's syndrome)
- LV aneurysm
- Mechanical complications

## Mechanical Complications

- Rupture of LV free wall 0.5%
- Rupture of the interventricular septum 0.17%
- Acute mitral regurgitation 0.25%

\*Significantly fewer mechanical complications since Primary PCIs performed

## Left Ventricular Free Wall Rupture

- Most die suddenly.
- Those presenting with contained rupture or pseudoaneurysm are typically hypotensive with cardiogenic shock. Echo is the diagnostic tool of choice to assess pericardial effusion and site of rupture.
- Emergent surgical repair is recommended for those surviving initial rupture.
- Pericardiocentesis is controversial.

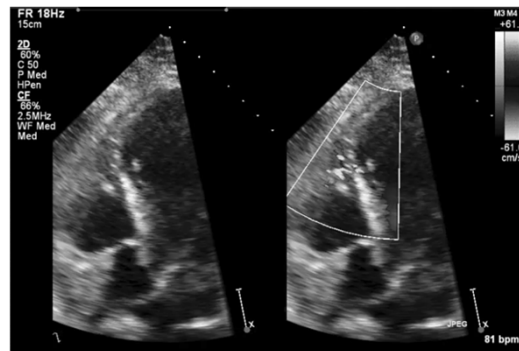


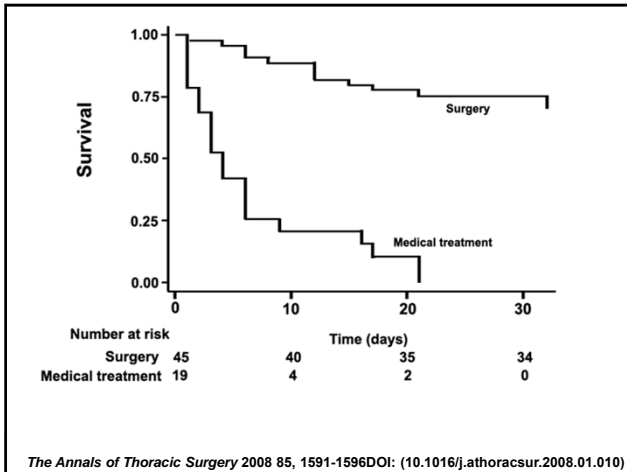
Patrick J. Lynch, medical illustrator - (CC BY 2.5)

## Rupture Of The Interventricular Septum

- Typically occurs in large anterior or inferior MIs.
- Physical exam: pulmonary edema, harsh pansystolic murmur at left sternal border or thrill palpated
- Echo is diagnostic tool of choice to assess
- Right heart cath maybe necessary if echo not adequate. Evaluate for O2 step-up in PA to signify left-right shunt.
- Prompt surgical patch repair recommended. Occasionally percutaneous closure device can bridge patient to surgery.

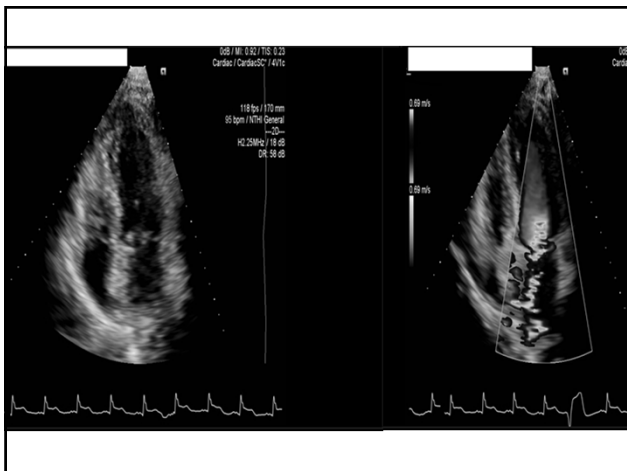
## Ventricular Septal Defect





## Acute Mitral Regurgitation

- Typically from papillary muscle rupture or severe ischemia to the posterior papillary muscle.
- Physical exam: patient's with severe pulmonary edema, pansystolic murmur at apex radiating to axilla.
- Echo is gold standard to diagnose but may need TEE to fully assess valve.
- Initial management is afterload reduction to stabilize patient followed by mitral valve repair and revascularization.



## Conclusions

- Incidence of STEMI is decreasing but mortality remains elevated.
- Early reperfusion is primary goal to treatment.
- Goal directed medical therapy important prior to discharge
- Monitor for post MI complications – rare but high mortality