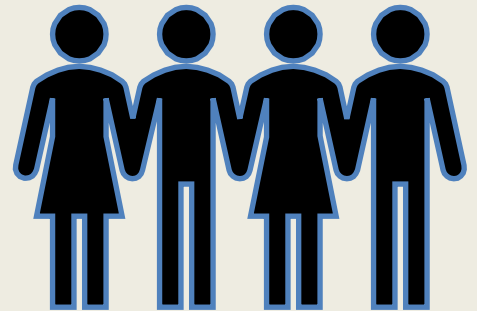# CORONARY HEART DISEASE

Shalon R. Buchs, MHS, PA-C

- Outline the diagnostic criteria and management for stable angina

- Discuss clinical features and diagnostic approach for each of the acute coronary syndromes: unstable angina, STEMI and NSTEMI

- Recognize causes of MI –
  - *Type 1 (blocked coronary due to atherosclerosis)*
  - *Type 2- (ischemia from a non coronary artery disease cause)*

- Develop an understanding of the medical management for each of the acute coronary syndromes

- Discuss the indications for percutaneous coronary intervention vs. thrombolytics vs. surgical intervention for coronary artery disease
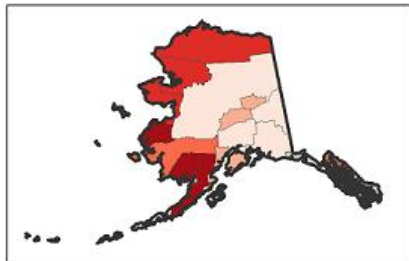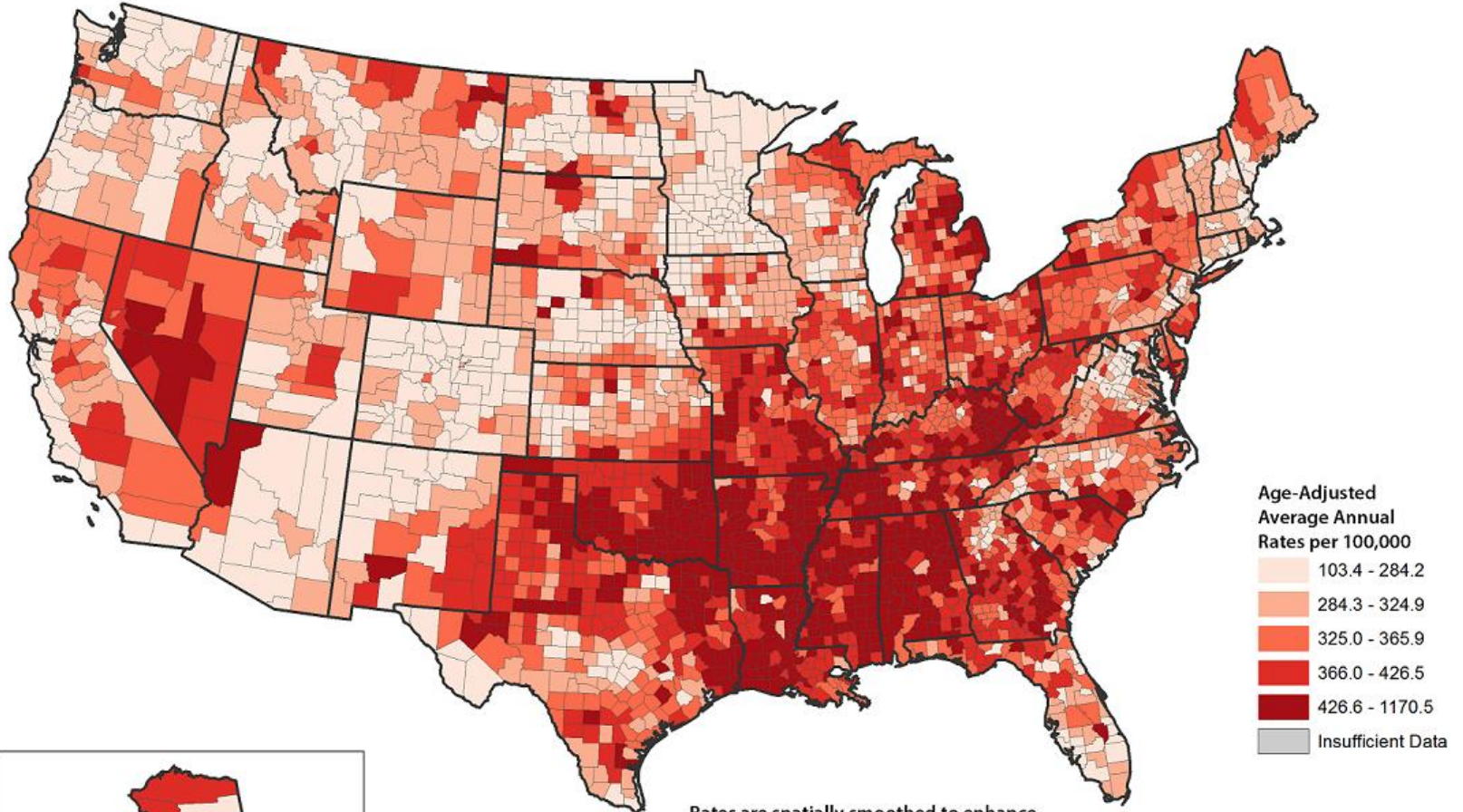
Objectives

Epidemiology of CHD

- Heart disease mortality has been declining in the US and areas where economies and health care systems are advanced

- BUT from a global perspective it is the number one cause of death and disability in the developed world

Heart Disease Death Rates, 2014-2016
Adults, Ages 35 +, by County

Age-Adjusted Average Annual Rates per 100,000

- 103.4 - 284.2
- 284.3 - 324.9
- 325.0 - 365.9
- 366.0 - 426.5
- 426.6 - 1170.5
- Insufficient Data

Rates are spatially smoothed to enhance the stability of rates in counties with small populations.

Data Source:
National Vital Statistics System
National Center for Health Statistics

www.cdc.gov/dhdsp/maps

Epidemiology of CAD

■ While recent numbers show an overall decline in mortality; prediction models estimate that mortality from CAD will grow from ~9 million in 1990 to ~19 million in 2020.

– *Increased life expectancy*

– *Diet and obesity*

– *Sedentary lifestyles*

– *Increased cigarette smoking*

Epidemiology

CAD is the leading cause of death in adults in the US

Approximately one third of all deaths in persons over age 35 can be attributed to CAD

18% increase for both sexes by 2030

Incidence

Lifetime risk of development of CAD is 49% for men age 40

Lifetime risk of development of CAD is 32 % for women age 40

Prevalence and burden
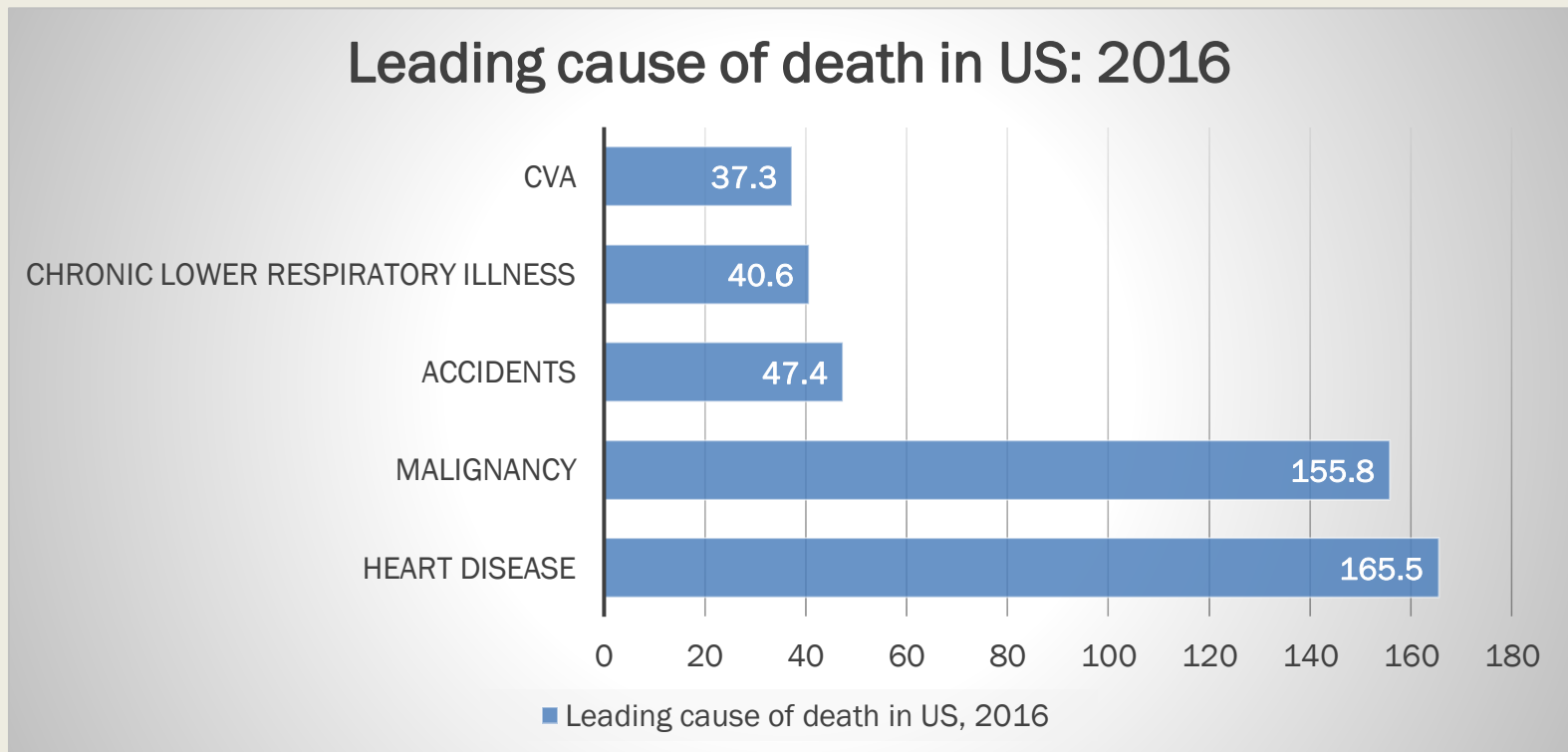
~18.2 million adults in the US have CAD (CDC)

More than 1 million individuals were expected to have a coronary event in 2019. with nearly 750,000 being new and just over 300,000 being recurrent events (AHA)
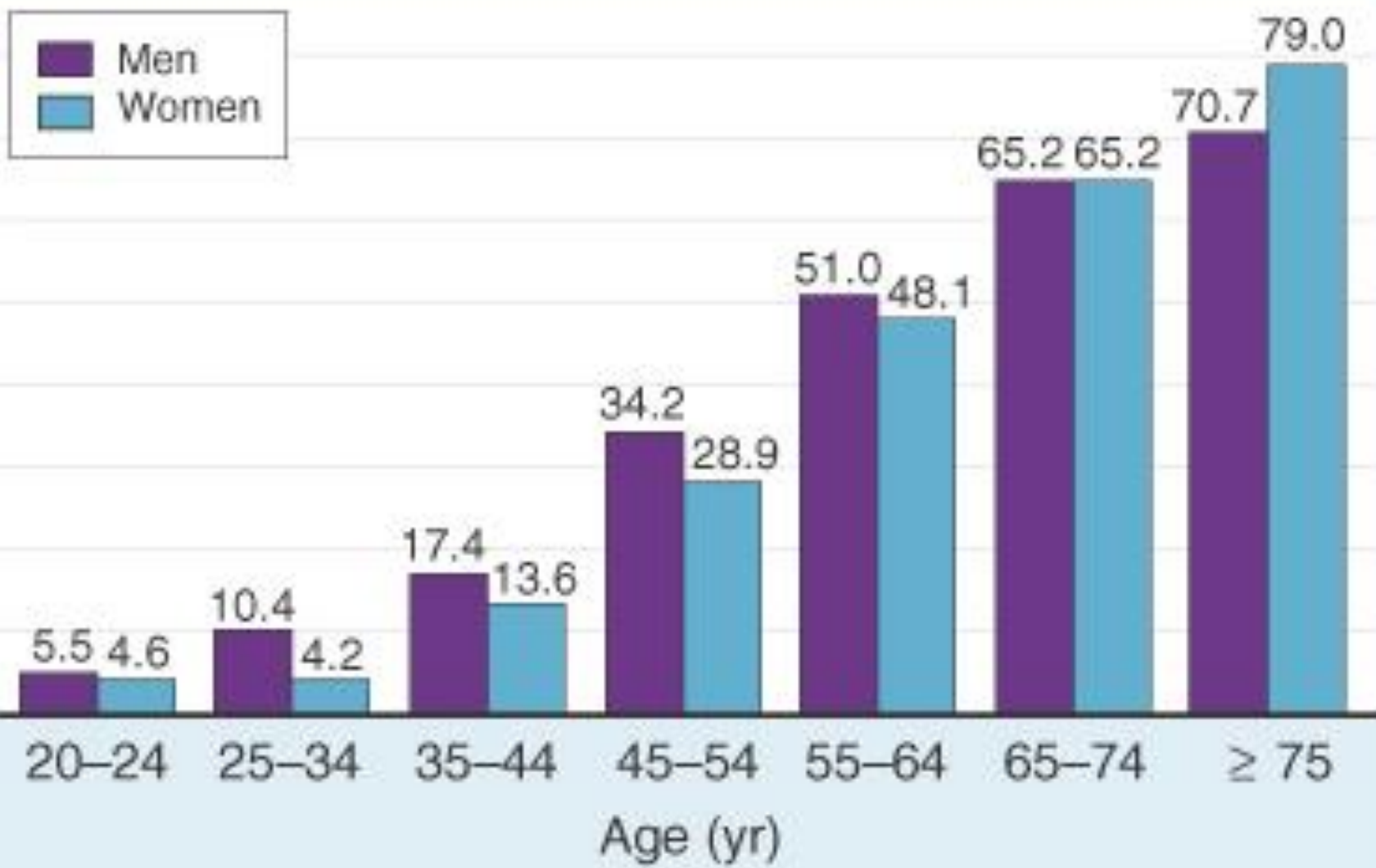
Total cost was ~$351.2 billion in 2014-2015 for the care of CVD, direct cost was $213.8 billion (AHA)

Leading cause of death in US: 2016

Leading cause of death in US: 2016

| Cause	Value
CVA	37.3
CHRONIC LOWER RESPIRATORY ILLNESS	40.6
ACCIDENTS	47.4
MALIGNANCY	155.8
HEART DISEASE	165.5

■ Leading cause of death in US, 2016

Source: CDC and AHA

- Diminished coronary perfusion; insufficient to meet myocardial oxygen demand

- Pathophysiology may be:

1. *fixed atherosclerotic narrowing of the coronary arteries*

2. *intra-coronary thrombosis overlying a disrupted atherosclerotic plaque*

3. *platelet aggregation*

4. *vasospasm of the artery*

Ischemia

Clinical Diagnoses in Ischemic Heart Disease

Chronic Stable Angina

Unstable Angina

Myocardial Infarction
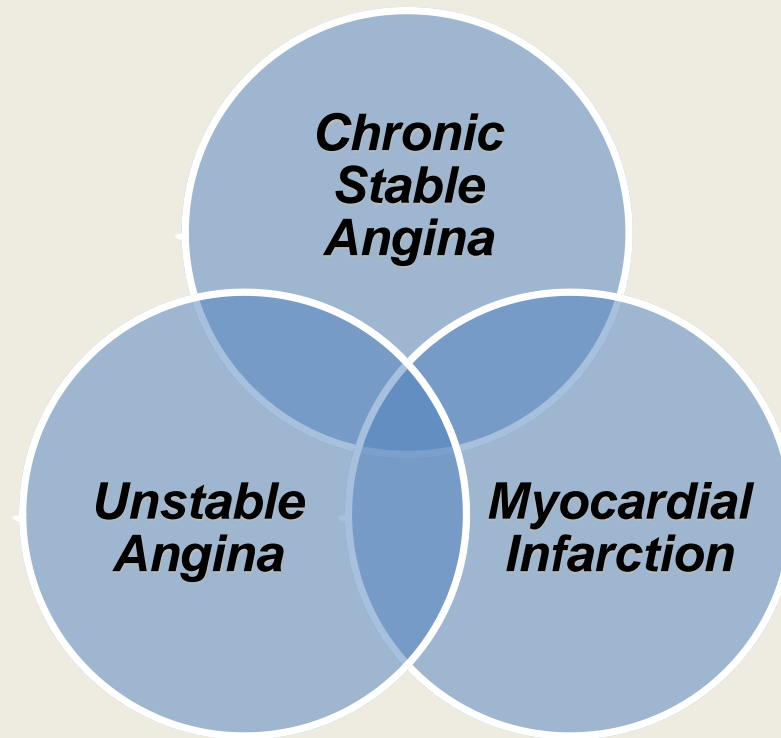
Angina

- Stable angina
  - *Occurs in a stable pattern, predictable, relieved by rest or nitroglycerin*

- Unstable angina
  - *Any change in the stable angina pattern, brand new angina or angina occurring at rest, may or may not respond to nitro*

- Variant angina (Prinzmetal's angina)
  - *Non specific pattern, often secondary to vasospasm rather than atherosclerotic narrowing of the vessels*

Extent of ischemia depends on intra-luminal pathophysiology
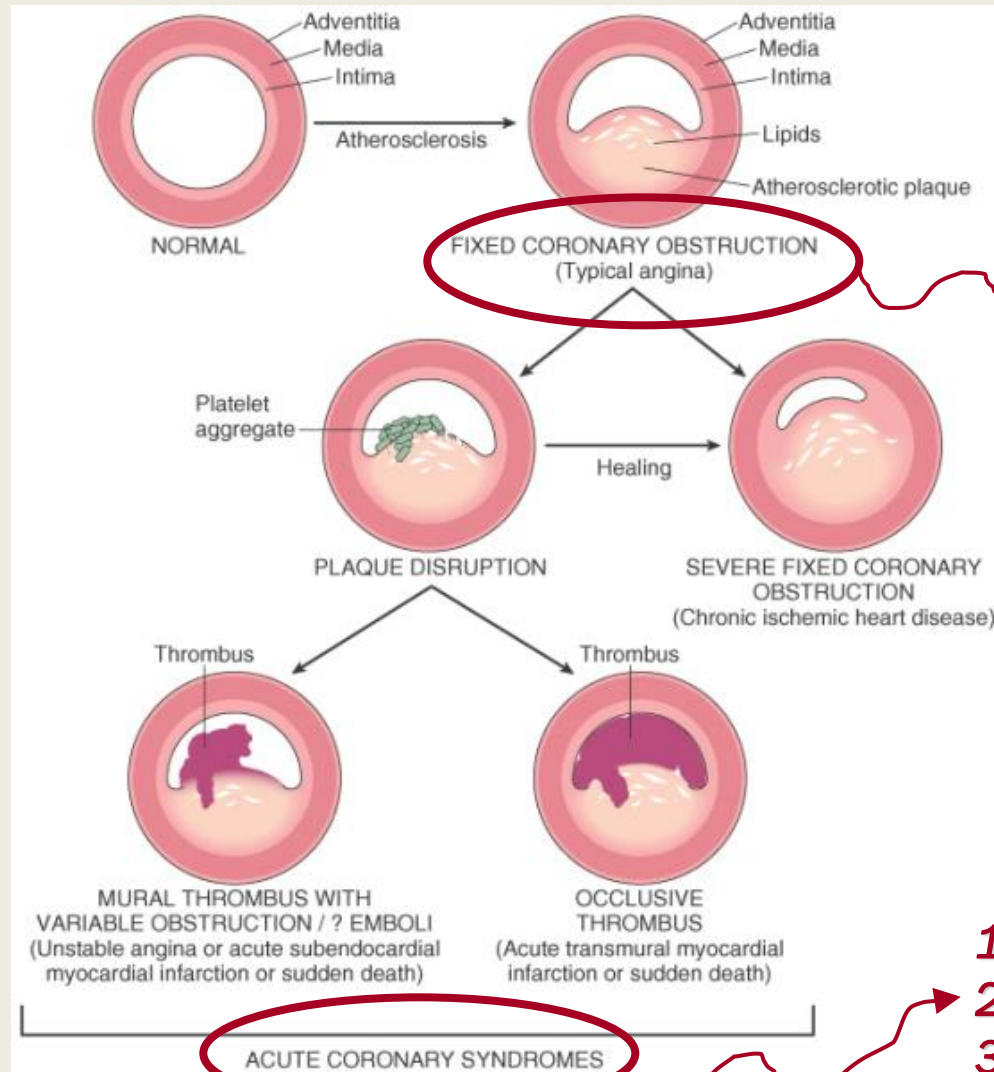
Chronic Stable Angina

1. *Unstable Angina*
2. *NSTEMI*
3. *STEMI*

- *Heart rate*

- *Ventricular wall stress*
  - *Non-compliance or "stiffness" of the ventricle*
  - *Increased afterload (e.g. systemic hypertension)*

- *Contractility of the heart*

In response to these factors, the _normal_ body response is for small arteries, or arterioles, to dilate, thereby increasing coronary blood flow to meet the increased demand.

With a narrowed artery, there is less dilation possible and therefore increased oxygen supply-demand mismatch in the setting of stress on the heart.

Things that stress the heart

Risk Factors

Non – Modifiable

- Age
- Gender
- Personal hx of CAD or stroke
- Family hx
- Ethnicity

Modifiable

- HTN
- Hyperlipidemia
- Diabetes Mellitus
- Smoking
- Metabolic syndrome
- Weight
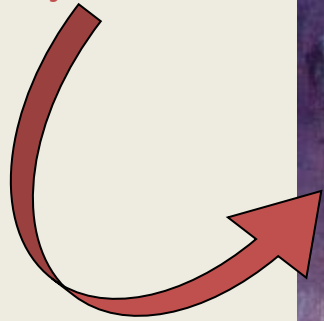- Sedentary lifestyle

Presentation

Chest discomfort

Dyspnea
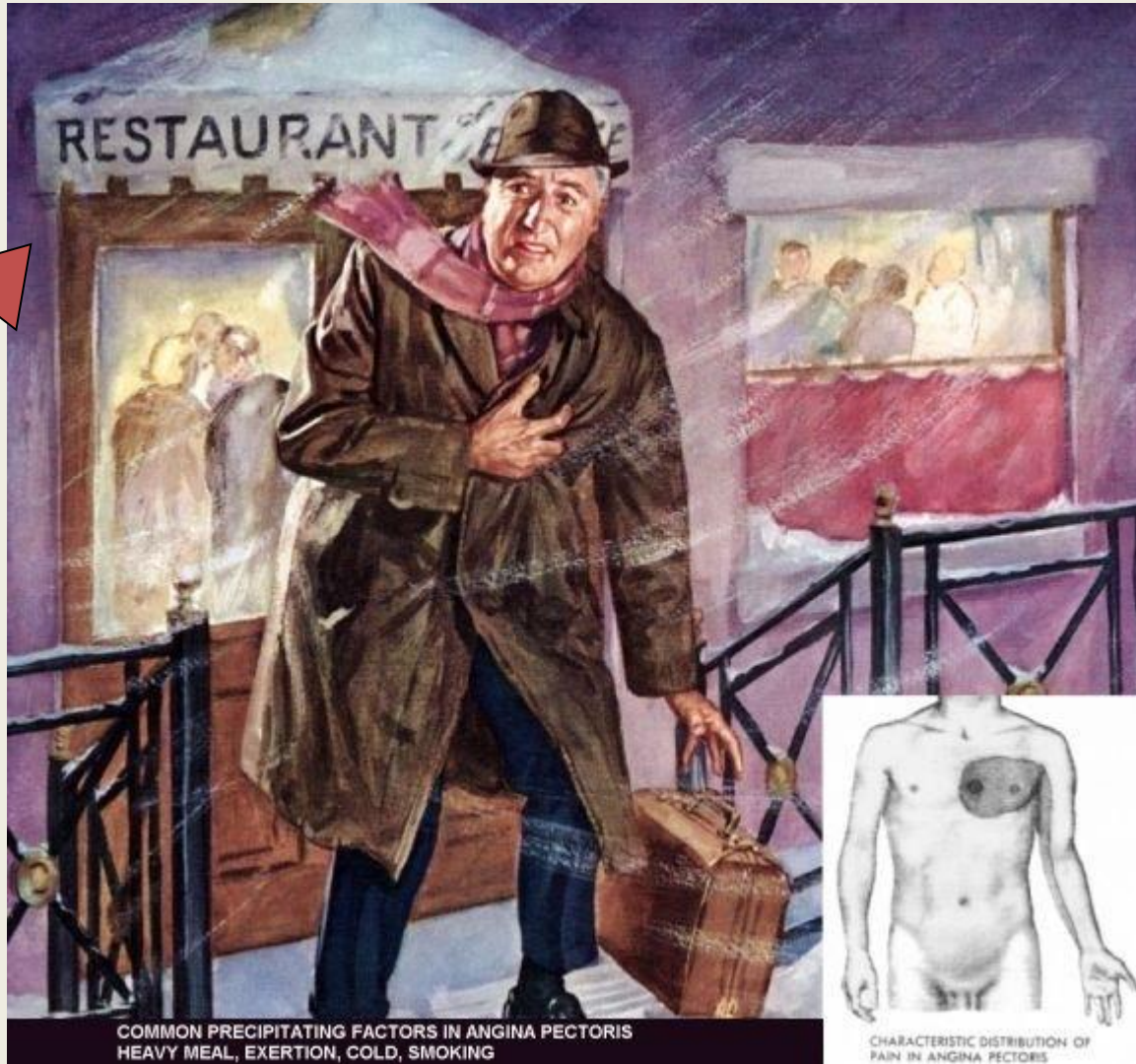
Diaphoresis

Nausea

Silent ischemia

Levine's Sign

Precipiated by food

cold

exertion

smoking

COMMON PRECIPITATING FACTORS IN ANGINA PECTORIS
HEAVY MEAL, EXERTION, COLD, SMOKING

CHARACTERISTIC DISTRIBUTION OF
PAIN IN ANGINA PECTORIS

Differential Diagnosis

- Angina
  - *Stable*
  - *Unstable*
  - *Vasospastic*

- Myocardial infarction
  - *STEMI*
  - *NSTEMI*

Differential Diagnosis

- Pericardial disease
  - *Pericarditis*
  - *Tamponade*

- Aortic Dissection

Differential Diagnosis

- Pulmonary Embolism

- Pleuritic pain
    - *Pneumonia*
    - *Pleurisy*

- Pneumothorax

Differential Diagnosis

- Gastrointestinal disease
  - *GERD*
  - *Esophageal disease*
  - *PUD*
  - *Gallbladder*
- Chostochondritis

- Anxiety
  - *Panic*

- ECG!! - should be obtained ASAP in a patient presenting with symptoms or signs of coronary artery disease

- A baseline ECG is helpful in those that have significant risk factors even if they have no symptoms

Evaluation

Evaluation

- History
  - *Identify presence/absence of risk factors*
  - *Determine temporal sequence and activities that exacerbate or relieve symptoms*
  - *Discover associated symptoms*

  - *Family Hx of CAD*
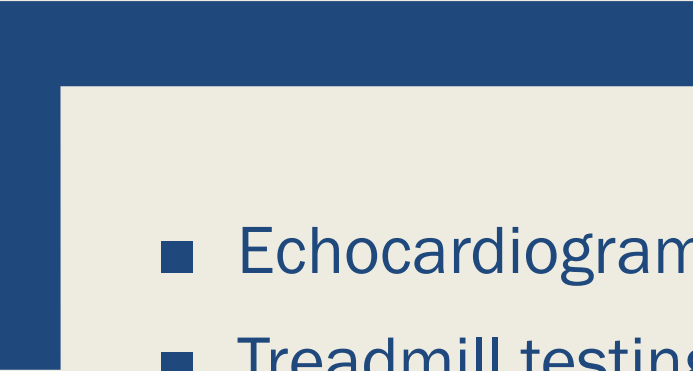
Evaluation

- Physical exam

  - *General survey*
  - *Vital signs*
  - *Pulmonary exam*
  - *Cardiac exam*
  - *Abdominal exam*
  - *Peripheral vascular exam*

Evaluation

- 12 – lead ECG
  - *ST-T wave changes*
  - *Rhythm disturbances*
  - *Evolution of ST-T changes*

- Cardiac biomarkers
  - **CK/CK-MB*
  - *Troponin*
  - **Myoglobin*

Evaluation - Radiographs

- Chest X-ray

    - *Evaluate for evidence of cardiomegaly, CHF, widened mediastinum suggestive of dissection, pneumothorax, pneumonia, free air under diaphragm, etc.*

- Echocardiogram

- Treadmill testing or pharmacologic stress
  - *Dobutimine or adenosine*

  Myocardial perfusion study

- CT angiogram

- Coronary angiogram

Additional work up

- Chronic, characteristic **unchanged pattern** of angina, precipitating factors and relief

- EKG may be "normal" *or* may show non-specific ST segment or T-wave abnormalities

- Requires ≥ 70% stenosis of a vessel

- Atherosclerotic plaque in culprit coronary artery has not ruptured or fissured (*hence no thrombosis*)

- Symptoms are purely based on supply/demand mismatch of oxygen to the affected myocardium

- Patients take sublingual nitroglycerin PRN for successful relief of anginal symptoms

Chronic Stable Angina

Chronic stable angina is.....

- REPRODUCIBLE

- PREDICTABLE

Chronic Stable Angina

TREAT ALL MODIFIABLE RISK FACTORS!!

Medical therapies *often* include:
- *Aspirin**
  - Antiplatelet action
- *PRN rapid-acting nitroglycerin*
  - vasodilator
- *β-blockers*
  - decrease HR, BP, contractility
- *Calcium channel blockers*
 - decreases HR, BP
- *Ranolazine*
  - decreases HR, BP

Chronic Stable Angina

Prognosis in Chronic Stable Angina based on:

- *Left ventricular dysfunction*
- *Extent of myocardium at risk*
  - *Areas of myocardium determined to have ischemia based on chemical stress testing*

  - *Revascularization should be considered for certain patients*
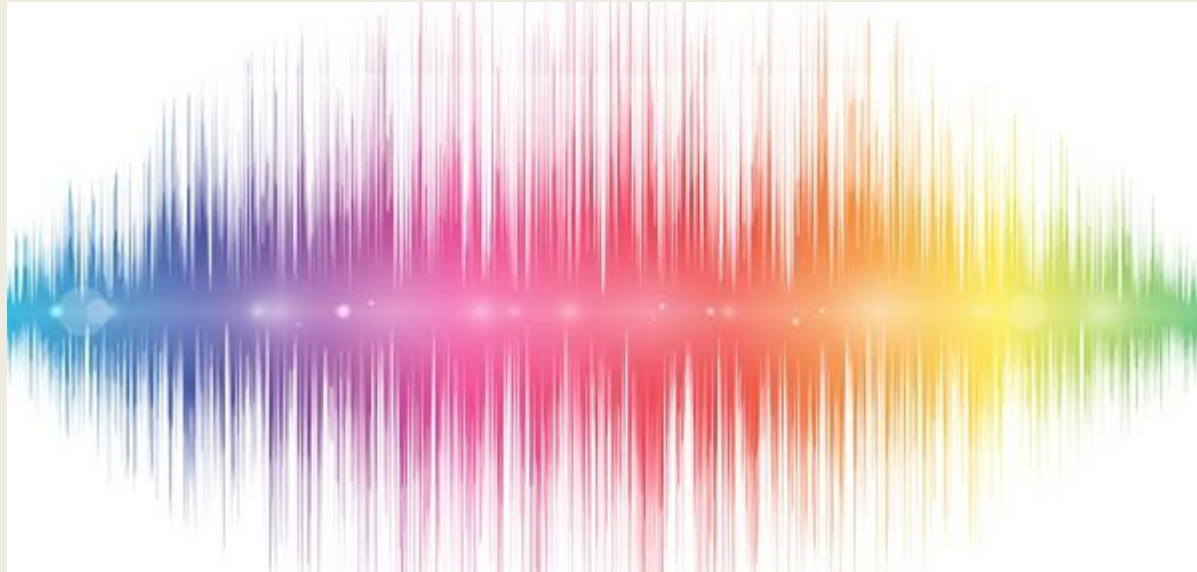
Clinical Diagnoses in Ischemic Heart Disease
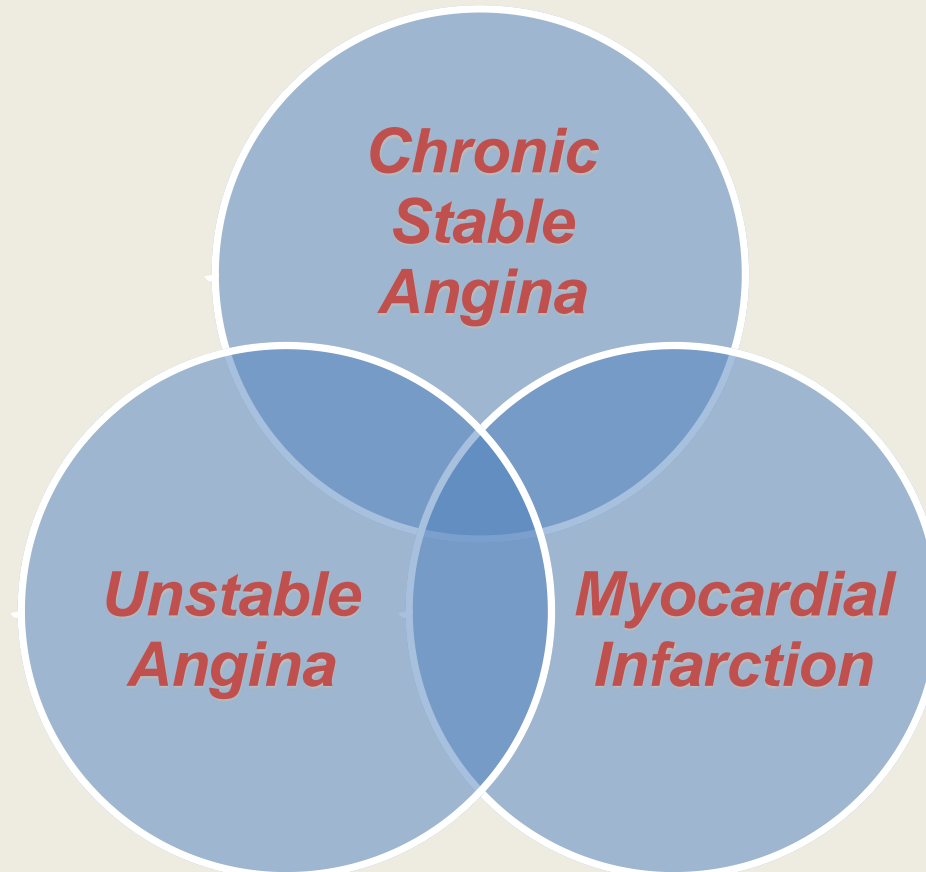
The *Acute Coronary Syndromes* (ACS) span the diagnoses of *Unstable angina (UA)*, *Non-ST segment elevation MI (NSTEMI)*, and *ST segment elevation MI (STEMI)*

Acute Coronary Syndrome

UA   NSTEMI   STEMI

Epidemiology

- Approximately 6 million patients present to hospital ERs each year with CP

- ~ 20-25% are diagnosed with ACS

Etiology, Type 1

- Fatty streak → foam cells → fibrous cap with lipid rich core → soft plaque

- Rupture of an atherosclerotic soft plaque within the coronary artery

- Platelet aggregation, thrombosis formation and vasospasm

Extent of ischemia depends on intra-luminal pathophysiology

Chronic Stable Angina

1. *Unstable Angina*
2. *NSTEMI*
3. *STEMI*
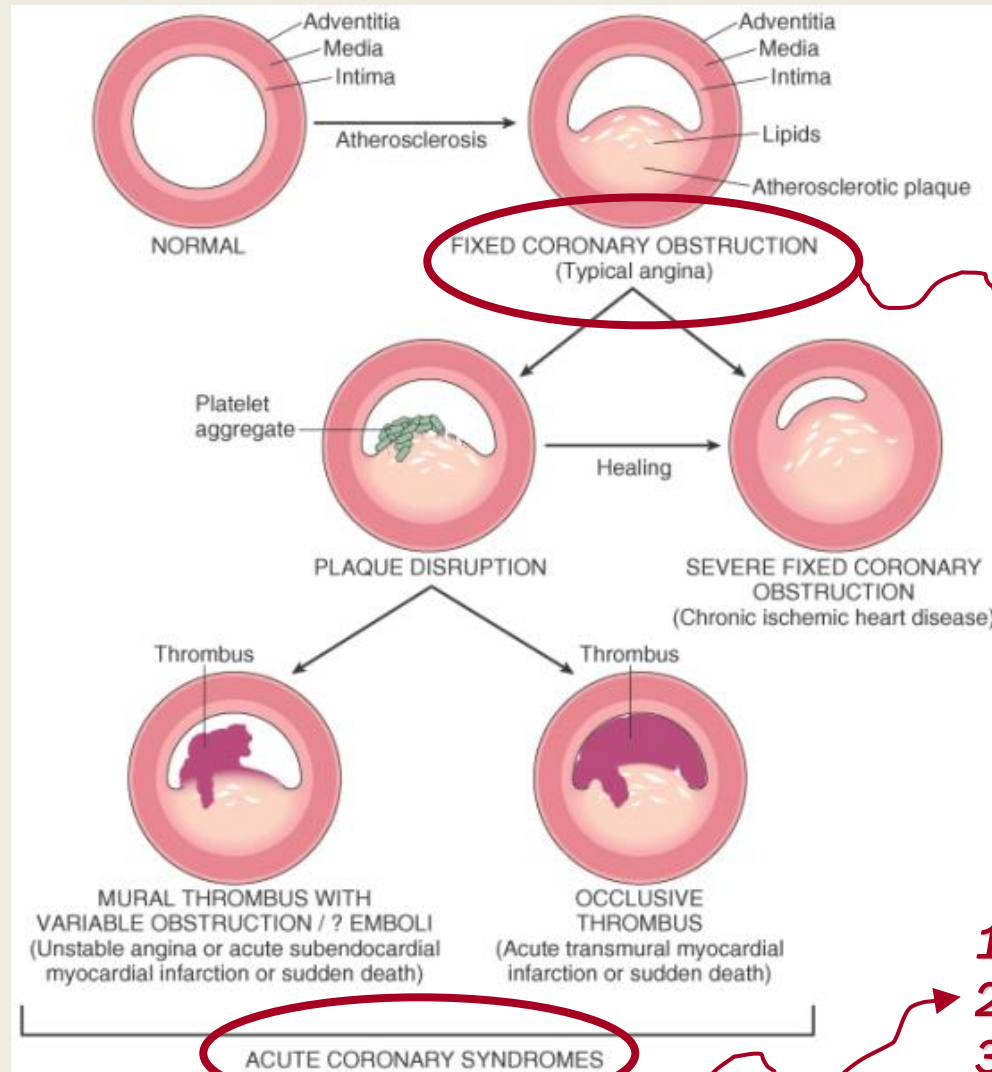
Type-2 myocardial infarction

- MI secondary to ischemia due to increased oxygen demand or decreased supply
  - *Not really defined by what is, rather what is not (no plaque rupture or erosion)*

Etiology, Type 2

- Supply demand mismatch outside of typical atherosclerotic pathophysiologic process (increased demand or decreased supply)
  - *Vasospasm*
  - *Poor perfusion of the coronary due to high or low BP*
  - *Arrhythmia*
  - *Anemia*
  - *Embolism*

Type-2 myocardial infarction

Assess the probability of underlying CAD

Informal vs. formal classification of high, medium or low probability

Consider initial troponin and possibly renal function; other tests guided by clinical presentation

Type-2 myocardial infarction

Look	Look holistically at the patient
Monitor	Monitor serial cardiac enzymes • Stable or downward trending
Treat	Treat the underlying condition to remove cardiac insult
Use	Use of evidence based, traditional ACS treatments may actually be harmful in Type 2 MI patients

UNSTABLE ANGINA	NSTEMI	STEMI
Non occlusive thrombus	Partially occluding thrombus Myocardial necrosis	Total coronary thrombosis Transmural myocardial necrosis

All tables will address type 1 only

Presentation

Chest discomfort · Dyspnea · Diaphoresis · Nausea · Silent ischemia
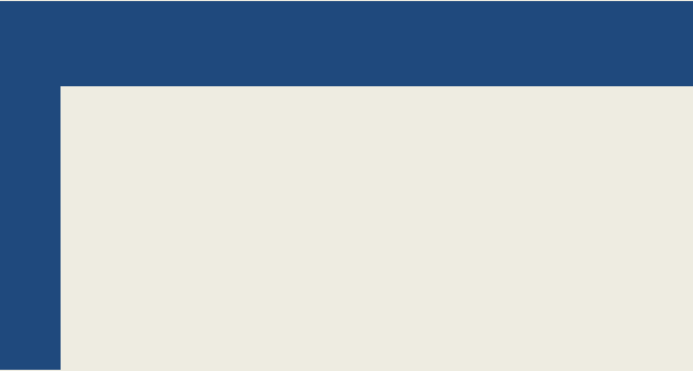
UNSTABLE ANGINA	NSTEMI	STEMI
Non occlusive thrombus	Partially occluding thrombus Myocardial necrosis	Total coronary thrombosis Transmural myocardial necrosis
New-onset, increasing or at rest	→	

- ECG!! - should be obtained ASAP in a patient presenting with potential ACS

Evaluation

ECG findings

UA
- *Normal*
- *Non-specific ST-T wave changes*

NSTEMI
- *Normal*
- *ST segment depression, T wave inversion*

STEMI
- *ST segment elevation in a regional pattern*
  - *1mm or greater in precordial leads or 2mm or greater in limb leads*
- *New LBBB, sgarbossa criteria*

UNSTABLE ANGINA	NSTEMI	STEMI
Non occlusive thrombus	Partially occluding thrombus Myocardial necrosis	Total coronary thrombosis Transmural myocardial necrosis
New-onset, increasing or at rest	⟶	
+/- ST-T wave changes or non specific ECG findings	ST depression or T wave inversion on ECG	ST elevation or equivalent

■ History

– *Identify presence/absence of risk factors*

– *Determine temporal sequence and activities that exacerbate or relieve symptoms*

– *Discover associated symptoms*

Evaluation

Evaluation

- 12 – lead ECG
  - *ST-T wave changes*
  - *Rhythm disturbances*
  - *Evolution of ST-T changes*
- Cardiac biomarkers
  - **CK-MB*
  - *Troponin*

Cardiac Biomarkers

- CKMB
  - *Found in cardiac and skeletal muscle*
  - *MB type more specific to cardiac muscle*
  - *Particularly useful in reinfarction and chronic kidney disease patients*
    - No longer utilized at some institutions
- Troponin
  - **Most specific for cardiac injury*

- Pt. evaluation will be different
  - *Other DDx possibilities will remain high on list in type 2 patients*
- ECG will not be as helpful
- Troponin will be 'positive' for both!
  - *Important to trend troponin for type 2 patients*

Type 1 vs. Type 2

UNSTABLE ANGINA	NSTEMI	STEMI
Non occlusive thrombus	Partially occluding thrombus Myocardial necrosis	Total coronary thrombosis Transmural myocardial necrosis
New-onset, increasing or at rest	⟶	
+/- ST-T wave changes or non specific ECG findings	ST depression or T wave inversion on ECG	ST elevation or equivalent
Negative cardiac biomarkers	Positive cardiac biomarkers	Positive cardiac biomarkers

Used to assess likelihood of adverse event

in given time frame

- HEART (6 week major cardiac event)
  - *Best for undifferentiated chest pain*
  - *Helps determine if pt. can be safely discharged*
  - *History, ECG, Age, Risk factors, initial troponin*
- VANCOUVER (4 week MI or ACS event)
  - *Best for undifferentiated chest pain*
  - *Helps determine if pt. can be safely discharged*
  - *ECG, Troponin, Hx of ACS or nitrate use, Pain reproducible, Age, Radiation of the pain*

Evaluation – Risk Stratification

- **TIMI (Mortality)**
  - *Useful for pts. with UA/NSTEMI*
  - *Age, Risk factors, Known CAD, ASA use, Angina, ECG, Enzymes*

- **GRACE (6 month mortality)**
  - *Useful for pts. with UA/NSTEMI*
  - *Age, HR, SBP, Creatinine, Cardiac arrest, ECG, Enzymes, PE findings*

Management – UA and NSTEMI

■ Anti – platelet therapy (DAPT)

 – *ASA*

 – *P2Y$_{12}$ agents: Clopidogrel; ticagrelor; prasugrel*

 – *+/-Glycoprotein IIb/IIIa inhibitors: Abciximab; Eptifbatide*

■ Nitroglycerin

■ Beta – blockers

■ Anti – coagulation therapy

 – *LMWH, synthetic factor Xa inhibitor or UFH*

 – *Direct thrombin inhibitors*

■ Oxygen

■ Morphine

Management – UA and NSTEMI

■ Cardiac cath and revascularization

–   *Persistent symptoms despite optimal medical therapy*

–   *Persistent elevations in troponin*

–   *Persistent ST segment depression*

–   *CHF*

–   *Hemodynamic instability*

–   *Dysrhythmia*

–   *Recent coronary intervention (6 months)*

–   *TIMI risk score (5-7)*

Management STEMI

Revascularization ASAP

Medical Management STEMI

- Anti – platelet therapy (DAPT)
  - *ASA*
  - *P2Y$_{12}$ agents: Clopidogrel; ticagrelor; prasugrel*
  - *+/- Glycoprotein IIb/IIIa inhibitors: Abciximab; Eptifbatide*

- Nitroglycerin

- Beta – blockers

- Anti – coagulation therapy
  - *LMWH or UFH*
  - *Direct thrombin inhibitor*

- Oxygen

- Morphine

Medical Management STEMI

Fibrinolysis

- When cath not available within 90 minutes

Contraindications

- History of cerebrovascular hemorrahage
- Cerebrovascular event within the last year
- SHTN >180 and or DHTN >110
- Internal bleeding

Management of patients with Type 2 ischemic disease should be directed at the underlying cause

Acute management

UNSTABLE ANGINA	NSTEMI	STEMI
Aspirin P2Y12 inhibitor +/- GP IIb/IIIa inhibitor	Aspirin P2Y12 inhibitor +/- GP IIb/IIIa inhibitor	PCI or Thrombolysis Aspirin P2Y12 inhibitor +/- GP IIb/IIIa inhibitor
LMWH or UFH Direct Thrombin inhibitor	LMWH or UFH Direct Thrombin inhibitor	LMWH or UFH Direct Thrombin inhibitor
Nitro	Nitro	Nitro
Beta Blockers	Beta Blockers	Beta Blockers

Long term management

- ASA; possibly P2Y12 inhibitor

- Statin

- PRN nitro

- Beta blocker

- ACE Inhibitor

- Secondary prevention

Take home points

- ECG immediately

- Cardiac biomarkers and ECG changes are the means by which we differentiate the ACS diagnoses

- Medical therapy for ACS patients is essentially the same

- STEMI patients go to intervention or receive thrombolytics

- Reduce other modifiable risk factors

Thank you!