

# Coronary Artery Disease

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# Overview

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- Stable CAD
- Unstable CAD
  - Angina
  - MI
- Pathophysiology
- Clinical Features
  - Hx/PE
- Differential Dx
- Diagnostic Testing
- Basic Rx

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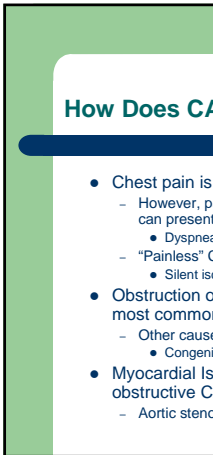
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# How Does CAD Present?

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- Chest pain is primary Sx
  - However, pain not always prominent in pts with CAD; pts can present with "anginal equivalents"
    - Dyspnea, faintness, fatigue, exercise intolerance
  - "Painless" CAD can also present as:
    - Silent ischemia; CHF; Arrhythmias; Sudden death
- Obstruction of CA's by atheromatous plaque is most common cause
  - Other causes of nonatherosclerotic obstruction include
    - Congenital abnormalities, arteritis, "bridging"
- Myocardial Ischemia can occur in absence of obstructive CAD
  - Aortic stenosis and hypertrophic cardiomyopathy

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## How Does CAD present?



- **Retrosternal** – viselike, constricting, heavy, squeezing, pressure-like, numb or burning
  - **Common** = Radiation to ulnar surface of left arm common, epigastric discomfort with or w/o chest pressure
  - **Uncommon** = Radiation above jaw or below epigastric area
- **Chronology** –
  - **Common** = gradual onset with activity, max intensity in minutes, then dissipates after rest or nitro (< 5 minutes)
  - **Uncommon** = max intensity in seconds
- **Non anginal pain**
  - Pleuritic pain, pain localized with one finger, pain reproduced with palpation of chest wall, pain lasting seconds or pain lasting hours

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## What about physical examination items in CAD?



- **Physical Examination**
  - Corneal arcus – “ring” around the periphery of cornea
  - Xanthelasma – intracellular lipid deposits near lower lids
  - Blood pressure
  - Arterial – decrease ankle/brachial index (<.9); decreased peripheral pulses
  - Cardiac examination
    - Signs of possible CAD = paradoxical splitting of S2; S3 or S4; systolic murmur due to MR (papillary muscle dysfunction)

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## Xanthelasma



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## The Pathophysiology of Stable CAD



- **Imbalance between myocardial O<sub>2</sub> requirements and supply**
  - Increased requirements in tachycardia, increased wall stress, increased contractility
    - Physical exertion; heavy meal; fever; thyrotoxicosis; emotional stress (increased catecholamines)
  - Supply determined by coronary blood flow and coronary arterial O<sub>2</sub> content
    - "Fixed" CAD
    - Transient vasoconstriction - Coronary "tone" affected by a variety of stimuli

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## New York Heart Association Classification



I Uncompromised	II Slight Compromise	III Moderate Compromise	IV Severe Compromise
Patients with cardiac disease but without resulting limitations of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, dyspnea, or anginal pain.	Patients with cardiac disease resulting in slight limitation of physical activity. They are comfortable at rest. Ordinary physical activity results in fatigue, palpitation, dyspnea, or anginal pain.	Patients with cardiac disease resulting in marked limitation of physical activity. They are comfortable at rest. Less than ordinary physical activity causes fatigue, palpitation, dyspnea, or anginal pain.	Patients with cardiac disease resulting in inability to carry on any physical activity without discomfort. Symptoms of cardiac insufficiency or of the anginal syndrome may be present even at rest. If any physical activity is undertaken, discomfort is increased.

*Circulation 64:1227, 1981.*

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## What else should I consider in the differential diagnosis?



- More likely to be confused:**
- **Esophageal problems** – reflux; motility disorders; spasm;
    - 10% of healthy population in US
    - Chest pain with normal coronaries commonly due to esophageal abnormalities
    - Key elements like CAD = characteristics of pain; relieved with nitro
    - Key elements unlike CAD = pain changes with posture or meals; relieved with antacids (GI cocktail);
  - **Pericarditis**
    - Chest pain not relieved with rest or nitro; pericardial friction rub and diffuse ST elevations on ECG
    - Key elements like CAD = retrosternal location, abnormal ECG
    - Key elements unlike CAD = pain is positional
  - **Costosternal syndromes**
    - Local pain and tenderness limited to anterior chest wall
    - Presence does not exclude CAD

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## What else should I consider in the differential diagnosis?



### Less likely to be confused:

- **Aortic dissection**
  - Severe, sharp, radiates to back, with or without aneurysm
  - Dx with chest CT or TEE
- **Severe pulmonary hypertension**
  - From right ventricular ischemia
- **Pulmonary emboli**
  - Dyspnea is cardinal sx; pleuritic chest pain with infarction; pleural friction rub
- **Biliary Colic** – consider in those with “atypical” pain, esp diabetics
  - Pain is steady, lasts 2-4 hours, no sx’s between attacks
  - RUQ abd pain is usual; radiation to scapula common, can radiate to epigastric area and chest

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## What about non-invasive tests?



### • Metabolic abnormalities

- **Lipids**
  - LDL < 100 is optimal
    - Esp for pts with multiple risks factors, DM and established CAD
  - HDL < 40 is an independent risk factor
  - TG's > 200 should be treated (lifestyle)
- **Glucose**
  - Impaired fasting glucose = 110-126
  - DM = FBS > 126
- **Others** – presence increase the risk of future CV events – no consensus on routine measurement
  - C-reactive protein; homocysteine; lipoprotein Lp(a)
- **ECG**
  - resting ECG is normal in 50% pts with stable angina
  - Most common abnormality with chronic CAD is non-specific ST-T wave changes
    - Non specific changes also seen in electrolyte abnormalities, LVH, antiarrhythmic drugs

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## Lipids



- LDL = primary target of therapy

### • Risk categories

- > 20% risk of major coronary event in 10 years = high risk
  - Known CAD
  - Diabetes
  - Known atherosclerotic disease (PVD, carotid disease and abdominal aortic aneurysm)
  - > 2 risk factors
- Consider causes of secondary hyperlipidemia
  - Diabetes
  - Hypothyroidism
  - Obstructive liver disease
  - Chronic renal failure
  - Drugs such as progestins, anabolic steroids, corticosteroids

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## Major Risk Factors

- Smoking
- Hypertension ( $\geq 140/90$  or on antihypertensive Rx)
- HDL  $< 40^*$
- Family Hx premature CAD ( $< 55$  males,  $< 65$  females)
- Age (men  $> 45$ ; women  $> 55$ )

\* HDL  $> 60$  is a negative risk factor, its presence removes one risk factor from total count

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## LDL Cholesterol goals based upon risk categories

	LDL goal	LDL level to consider drug Rx	LDL level to consider lifestyle changes
CHD or CHD Risk Equiv	$<100$	$>130$	$> 100$
2+ Risk factors	$<130$	$>130$ (10 yr risk 10-20%) $>160$ (10 yr risk $<10\%$ )	$>130$
0-1 risk factor	$<160$	$>190$	$>160$

JAMA 2001;285:2486

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## Stable CAD: Non invasive tests

- **Exercise ECG**
  - Looking for ST segment depression and symptoms
    - 1 mm (+) typical sx's = 90% positive predictive value
    - 2 mm (+) typical sx's = diagnostic
    - 1 mm (-) typical sx's = 70% PV
    - 2 mm (-) typical sx's = 90% PV
    - Overall sensitivity = 70%; Specificity = 80%
  - Patients need to get to  $>85\%$  predicted maximal heart rate
    - Predicted max HR =  $(220 - \text{age})$

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## Stable CAD Non invasive tests



- **Noninvasive tests – Radionuclide Perfusion imaging** (“Cardiolite”)
  - **Exercise ECG** with images of myocardial blood flow
    - Compare images at maximal exercise with images at rest
      - “Defects” with exercise and not with rest = ischemia
      - “Defects” with both exercise and rest = MI
    - Sensitivity = 90%; Specificity = 80%
  - **Pharmacological** nuclear stress tests in pts unable to exercise
    - Adenosine or dipyridamole (persantine)
      - Vasodilators – unmask coronary stenosis by causing relative increases in flow in non-diseased CA’s
    - Dobutamine – inotropic agent, increases myocardial work

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## Radionuclide imaging vs traditional exercise stress test



- **Radionuclide imaging**
  - ECG abnormalities that make interpretation hard
    - LBBB
    - Paced Rhythm
    - >1 mm ST depression at rest
  - Inability to exercise
  - Angina and h/o revascularization
    - Here the point of EST is not to detect CAD which is already established but to identify the amount of viable myocardium

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## Stress ECHO



- **ECHO**
  - Directly visualizes ventricles
  - Exercise ECHO look for “regional wall motion abnormalities”
    - Sensitivity and Specificity = 85% for multivessel disease, lower sensitivity for single vessel disease (60%)
  - Pharmacological (dobutamine) stress ECHO for those who can’t exercise

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## Pretest likelihood of CAD in symptomatic patients according to age and sex



Age	Nonanginal chest pain		Atypical angina		Typical angina	
	Men	Women	Men	Women	Men	Women
30-9	4	2	34	12	76	26
40-9	13	3	51	22	87	55
50-9	20	7	65	31	93	73
60-9	27	14	72	51	94	86

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## CAD in Diabetics



- CAD –
  - 7 year incidence of MI or death 20%; equal to that of non-diabetics with a history of MI
  - Therefore DM requires aggressive antiatherosclerotic Rx
  - Mortality reduction 15-20% higher in diabetic hyperlipidemic patients treated with a statin than in non-diabetic hyperlipidemia patients.
  - Treating hypertension more important than tight glycemic control in limiting CV events in diabetics
  - B-blockers reduce the risk of MI by 23% in patients with DM without increasing diabetes related complications

JAMA 2002;287:2570-2581

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## Acute Coronary Syndromes



- ST segment elevation MI (STEMI)
  - Q Wave MI
- Unstable angina (UA)
- Non ST segment elevation MI (NSTEMI)
  - Non Q wave MI

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## Acute Coronary Syndromes



- Patients presenting to ED with acute chest pain
  - 15% will be found to have an MI
  - 30% found to have unstable angina
- 50% of deaths from AMI occur within one hour of sx onset – usually due to arrhythmia (V. fib)
- MI's due to coronary atherosclerosis with superimposed coronary thrombosis – brought on by plaque rupture
  - This is the cause of almost all acute coronary syndromes leading to either complete occlusion of coronaries leading to Q wave infarction or partial occlusion leading to acute coronary syndrome of unstable angina or non-Q MI

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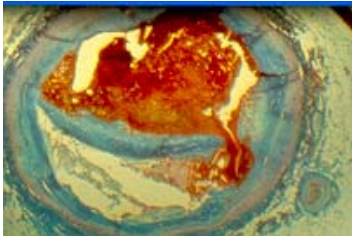
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## Plaque Disruption: The site of intracoronary thrombosis



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## Myocardial Infarction



- >75% of patients with MI have > 1 coronary artery diseased;
  - However about 6% of patients with AMI will have angiographically normal coronary arteries
- Biochemical markers of necrosis are CK-MB or troponin.

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## Cardiac Biomarkers “enzymes”



- Enzymes (cardiac biomarkers) diffuse into the cardiac interstitium after MI and become detectable in the blood within hours.
- **CK-MB** detectable 4 hours after MI and up to about 2 days
  - Some CK-MB detectable in healthy patients
- **Troponin I** detectable 4 hours after an MI and up to a week afterwards
  - Therefore, difficult to use to Dx re-infarction
  - Troponin I not detectable in healthy patients
  - “Microinfarctions” can increase troponin I and not increase CK-MB (30% of pts with UA)

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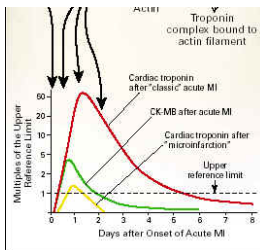
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## Time Course of Cardiac Biomarkers after M.I.




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## Unstable Angina and Non-ST segment elevation myocardial infarction (NSTEMI)



- Caused by *non occlusive* thrombus
- Risk of death and non-fatal cardiac ischemic events can be determined
- “High risk” history:
  - Nature of Sx
    - Accelerating ischemic in past 48 hours and prolonged ongoing (>20 minutes) rest pain
  - Prior Hx MI
  - Age
  - Sex
  - # of traditional risk factors present

Circulation 2000;102:1193

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## Unstable Angina and Non-ST segment elevation myocardial infarction (NSTEMI)



- "High Risk" PE Findings
  - Pulmonary edema
  - S3 gallop
  - New or worse MR murmur
  - Hypotension, bradycardia, tachycardia
- ECG findings
  - ST-segment changes of  $\geq 1$  mm
  - Sustained V Tachy
  - New or presumed new BBB

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## Chest Pain Assessment



- ECG most important single source of data in the evaluation of patients with chest pain
- ECG findings in patients with acute chest pain
  - New ST-segment elevation of  $\geq 1$  mm
    - Probability of MI = 80%
  - ST-segment depression or T-wave inversion no know to be old
    - Probability of MI 20%

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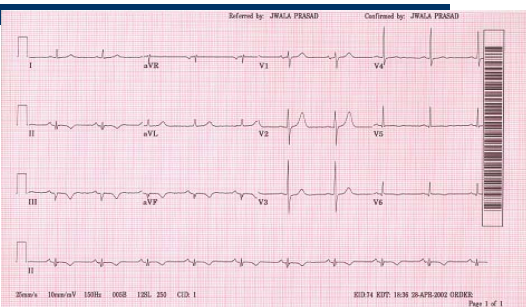
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## Interpret the ECG



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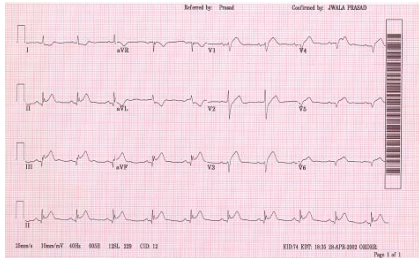
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## Interpret the ECG



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## Therapeutics Patients with Acute Coronary Syndromes



### Anti-ischemic therapy

- Nitrates
  - relieve pain and ischemia, given sublingually or IV acutely
- Morphine sulfate
  - Relieve pain, decreases agitation and decreases preload (decreased venous congestion)
- B- Blockers
  - Decreases myocardial oxygen demand, decreases heart rate, stabilizes membranes thereby decreasing arrhythmia risk
- Danger – do not use short acting Calcium channel blockers (nifedipine)

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## Therapeutics Patients with Acute Coronary Syndromes



### Antiplatelet and anticoagulant Rx

- Antiplatelets
  - ASA
    - Give promptly
  - Clopidogrel
    - Use if unable to take ASA
- Anticoagulants
  - Heparin – LMWH or unfractionated
- RE-vascularization (thrombolytics or PTCA with stent) in patients with STEMI
- Danger – thrombolytics in patients with NSTEMI or unstable angina

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**Therapeutics**  
Acute Coronary Syndromes- Post discharge



- ASA
- B-blockers
- Statins
- NTG
- ACE-I for patients with LV dysfunction

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