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Introducing

CXR Interpretation

Objectives:

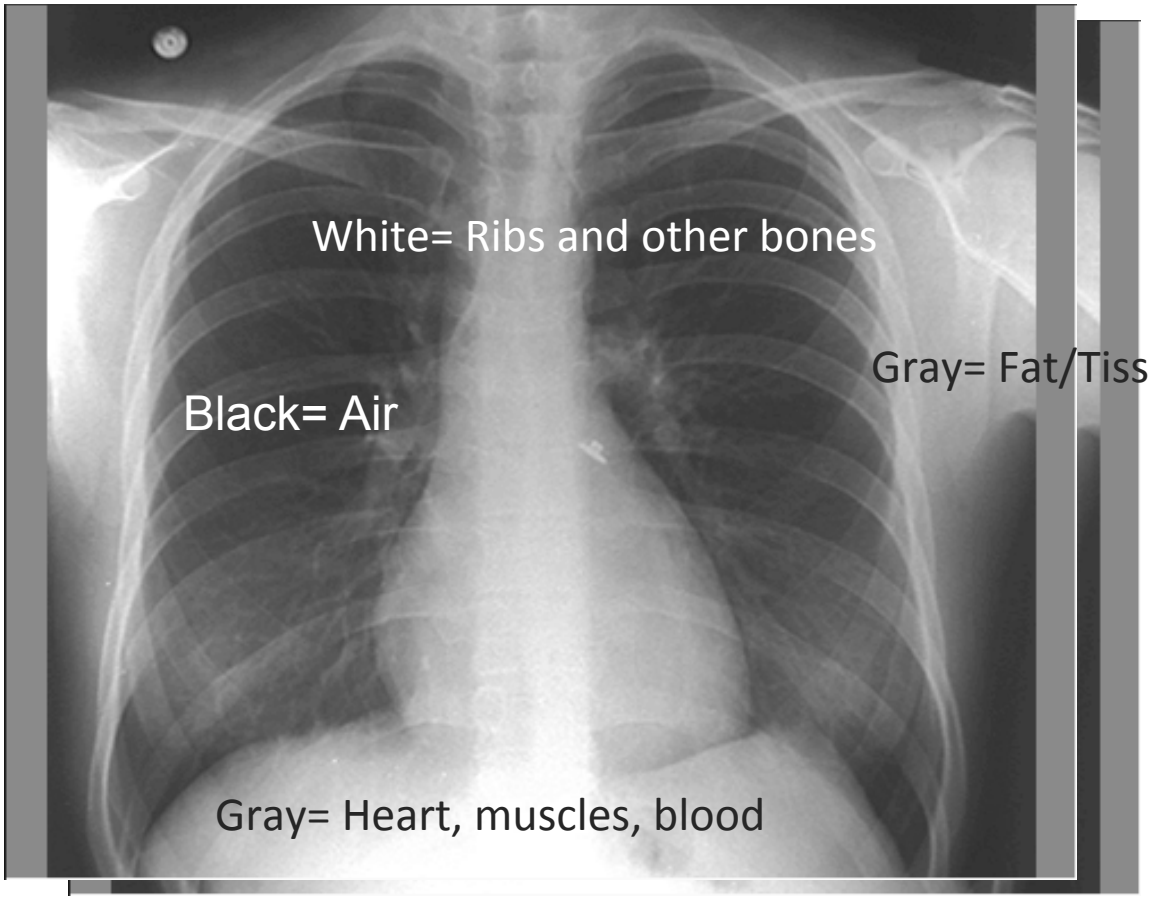
1. Describe the basic physics of the CXR
2. Describe the standard views of the CXR
3. Interpret in a step-wise approach

Test your skills with Case Studies

Densities & Lucency

- **Air < fat < liver < blood < muscle < bone < barium < lead**
- **Air** — least dense; most transparent or radiolucent; unobstructed beam or air-filled densities appear **black**
- **Fat** — breasts appear **gray (less black)**
- **Fluid** — vessels, heart, diaphragm, soft tissues, effusions appear **light gray**
- **Mineral** — most dense (or radiolucent) :calcifications such as the aortic knob, old granulomas; bullets, rods appear **white**

1



Density Summary

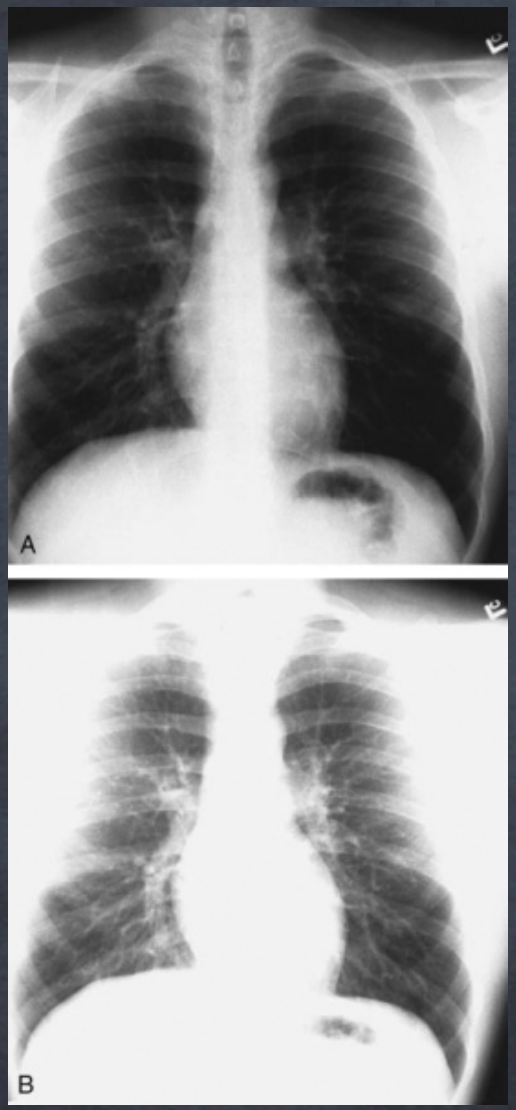
Exposure

Over-Exposed XR

- **makes it very easy to see behind the heart and the regions of the clavicles and thoracic spine, but the pulmonary vessels peripherally are impossible to see.**

Under-Exposed XR

- **accentuates the pulmonary vascularity, but you cannot see behind the heart or behind the hemi-diaphragms.**



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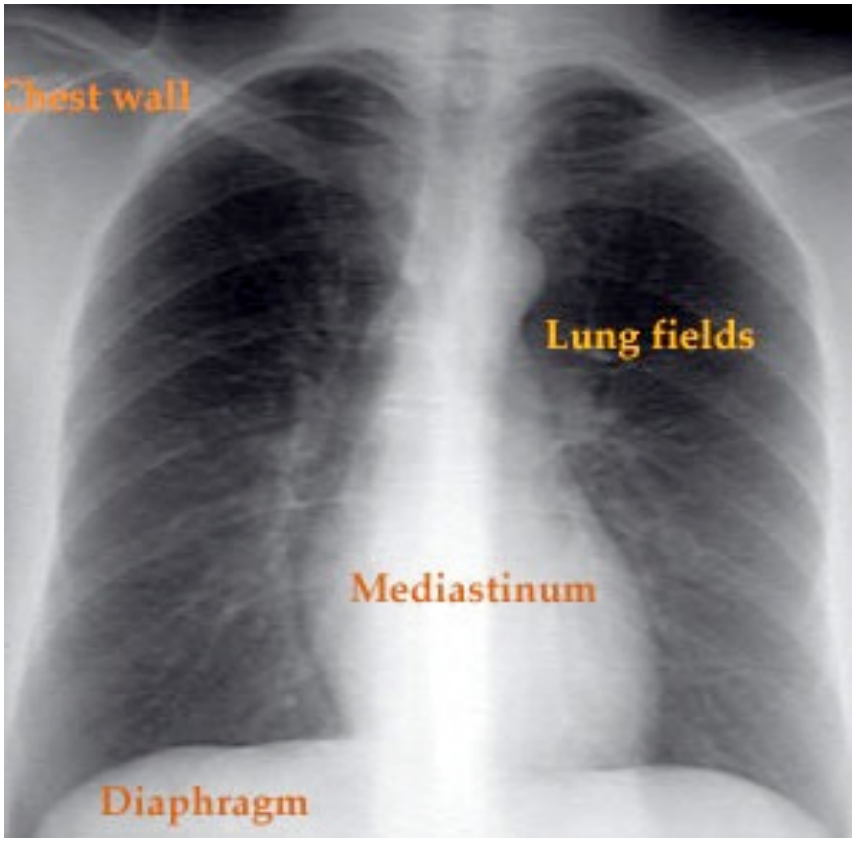
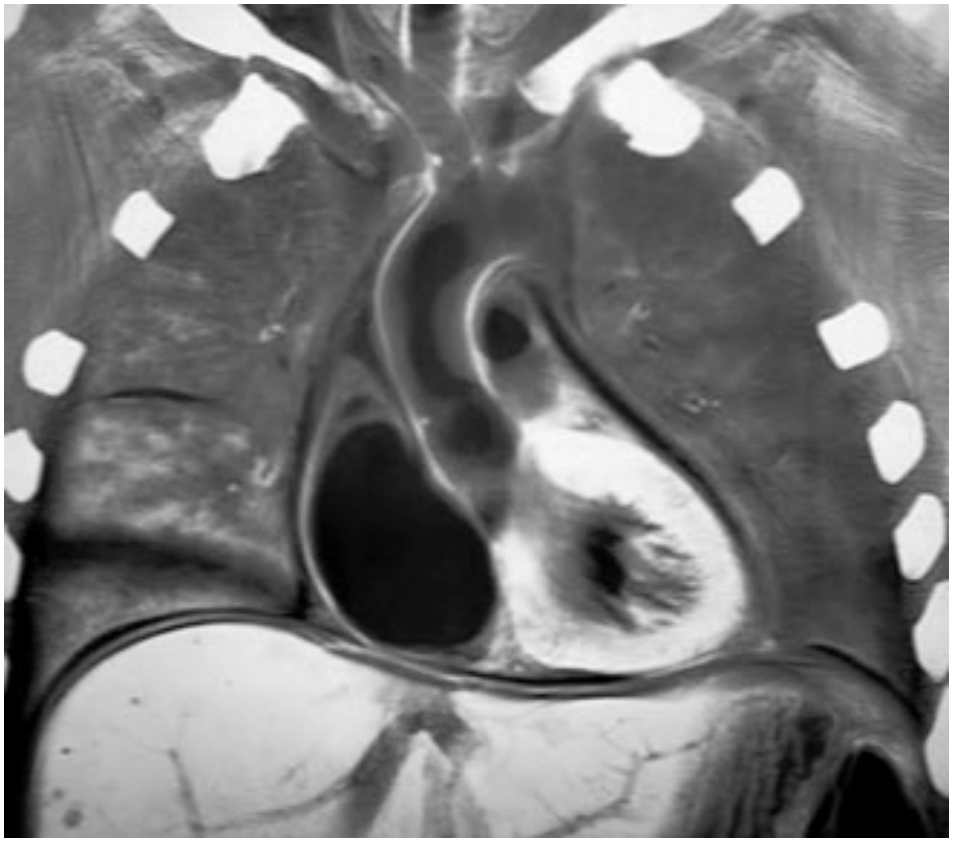
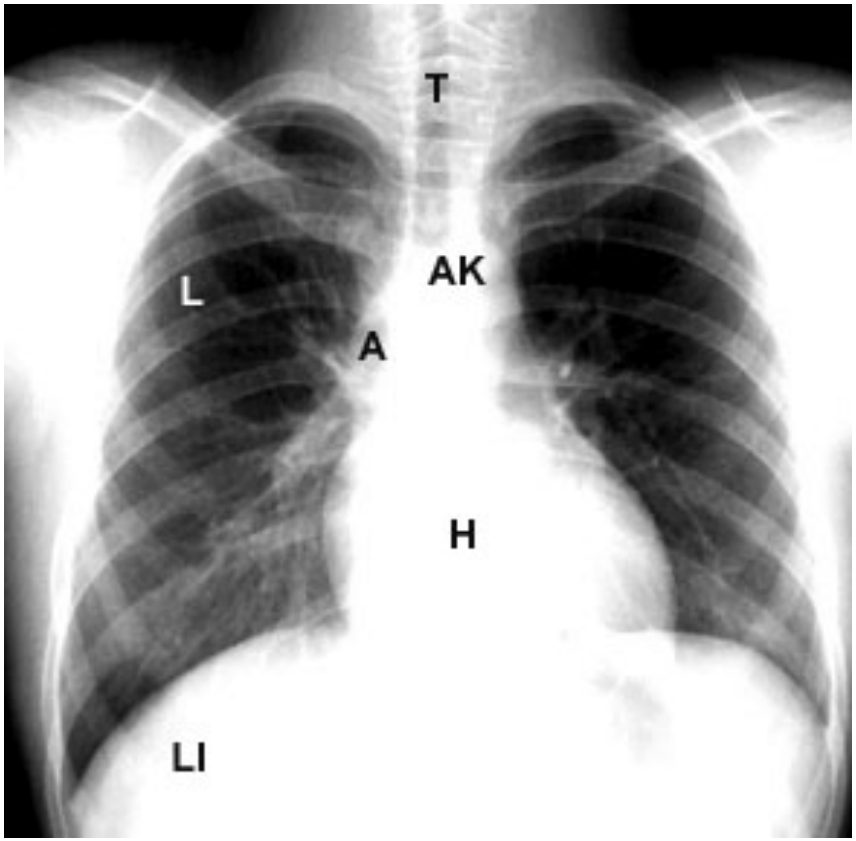
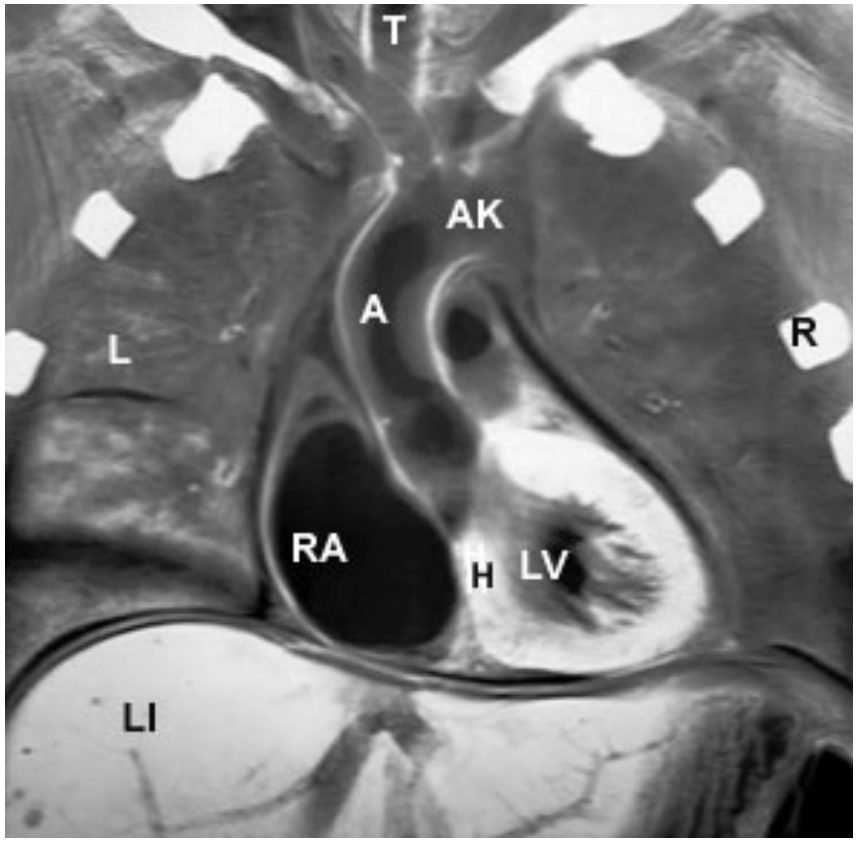
Anatomical Structures



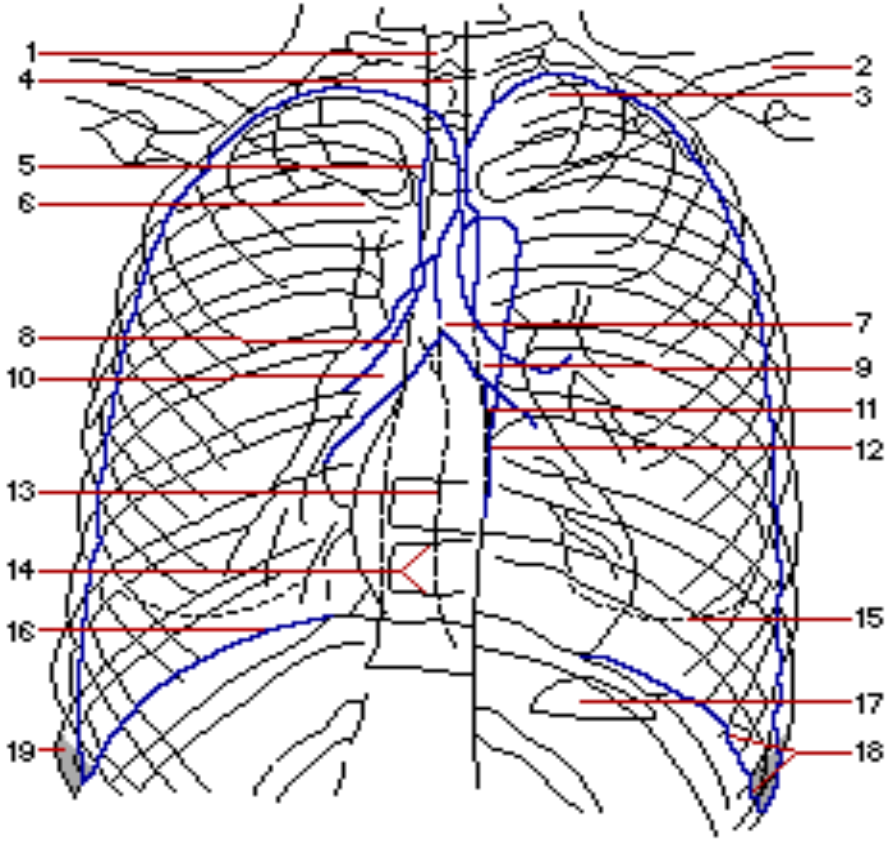


Anatomical Structures in Detail



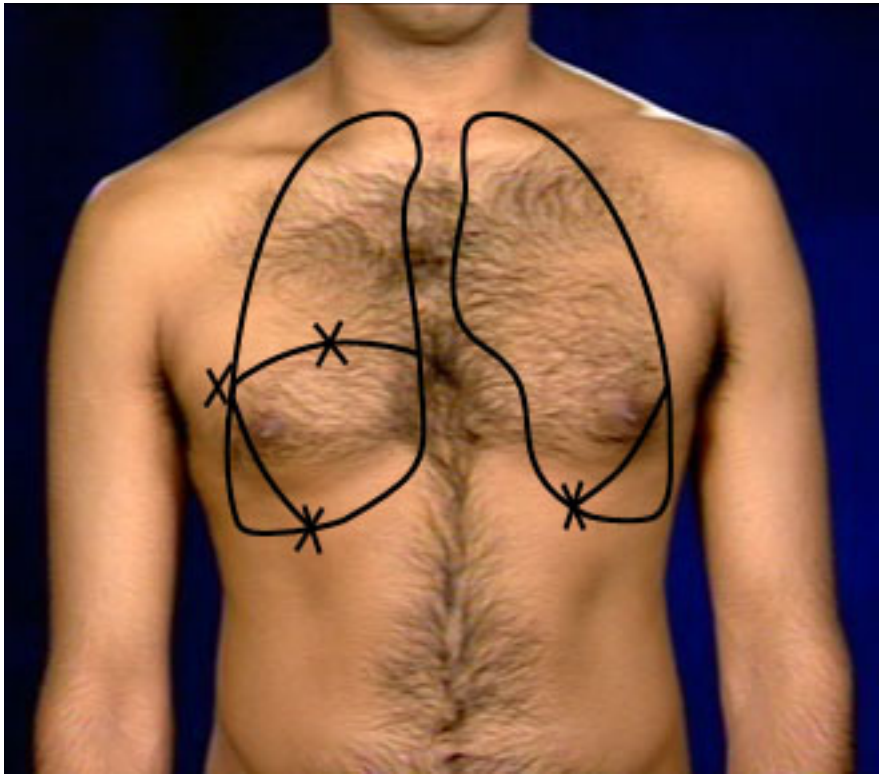


Chest, Frontal View

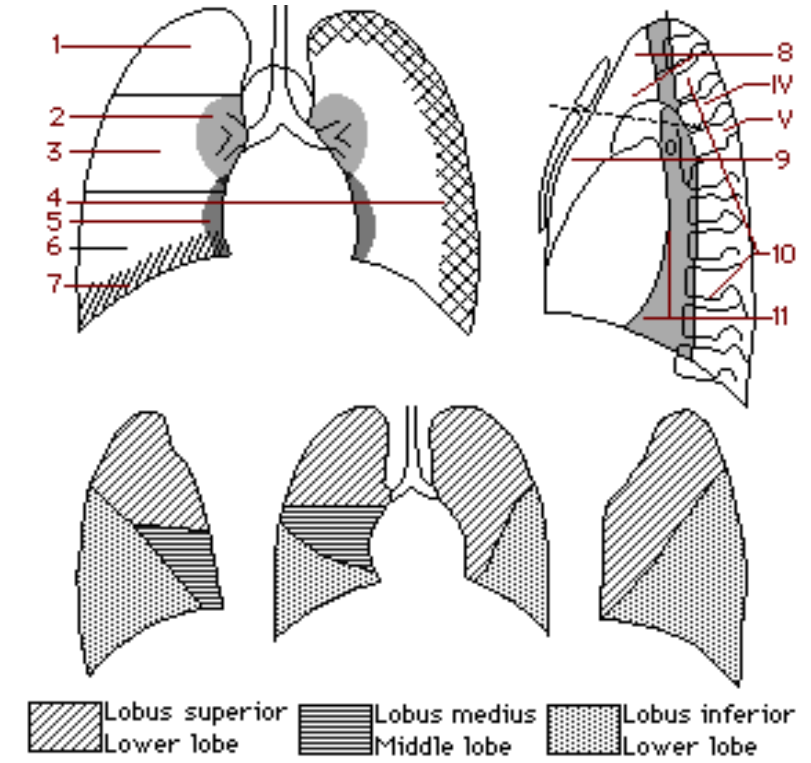


1 Trachea
2 Clavicle
3 Lung apex
4, 5 Posterior superior junction-line complex
6 Right paratracheal stripe
7 Tracheal carina
8 Right main stem bronchus
9 Left main stem bronchus
10 Bronchus intermedius
11 Preaortic stripe
12 Descending aorta
13 Azgoesophageal stripe
14 Paraspinal stripe
15 Breast contour
16 Dome of the diaphragm
17 Stomach bubble
18 Diaphragmatic muscle slips
19 Costophrenic sulcus

Surface Anatomy of lobes

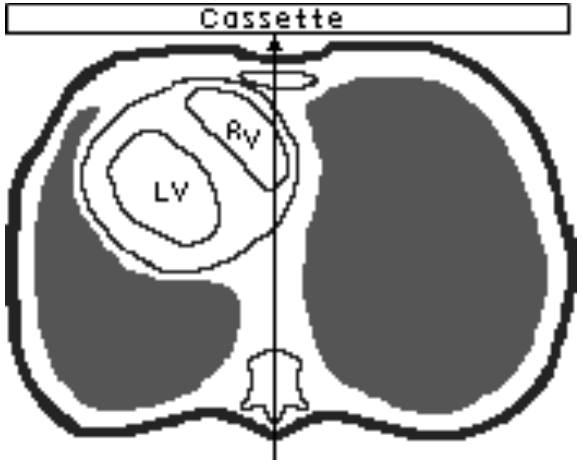


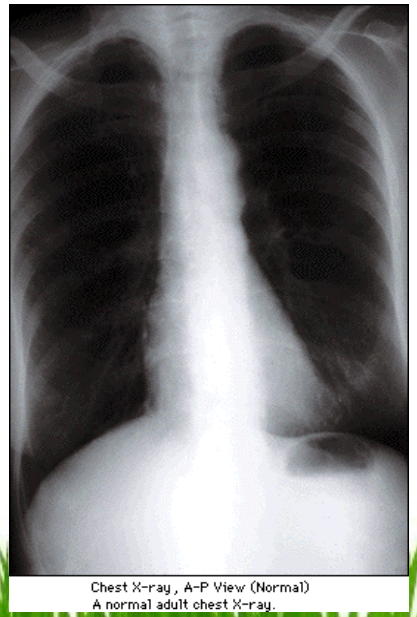
Pulmonary Disease



1 Upper lung zone
2 Perihilar region
3 Mid-lung zone
4 Chest wall
5 Pericardiac region
6 Lower lung zone
7 Supradiaphragmatic region
8 Superior anterior mediastinum
9 Inferior anterior mediastinum
10 Posterior mediastinum
11 Middle mediastinum
IV, V Thoracic vertebrae

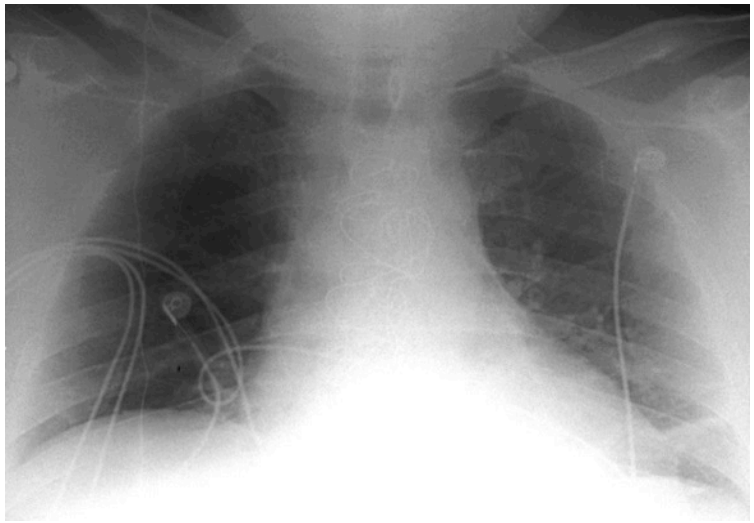
STANDARD UPRIGHT CXR

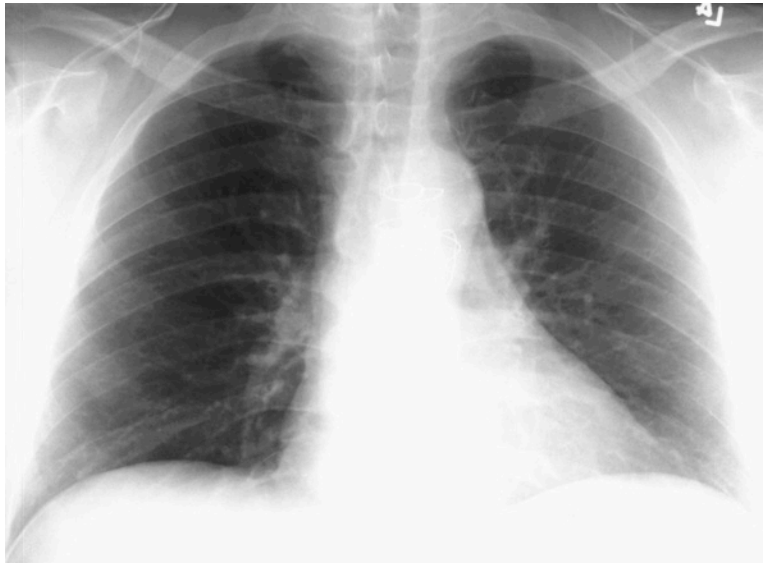




- PA or film in *front* of patient, beam behind
- AP film, taken from the same distance (6') enlarges the shadow of the heart which is far anterior in the chest and makes the posterior ribs appear more horizontal.
- In a supine film, the diaphragm will be higher and the lung volumes less than in a standing patient.

Same Patient but Different Views : Which is Which?





One is a PA film while the other is an AP chest radiograph?

Tell me why?

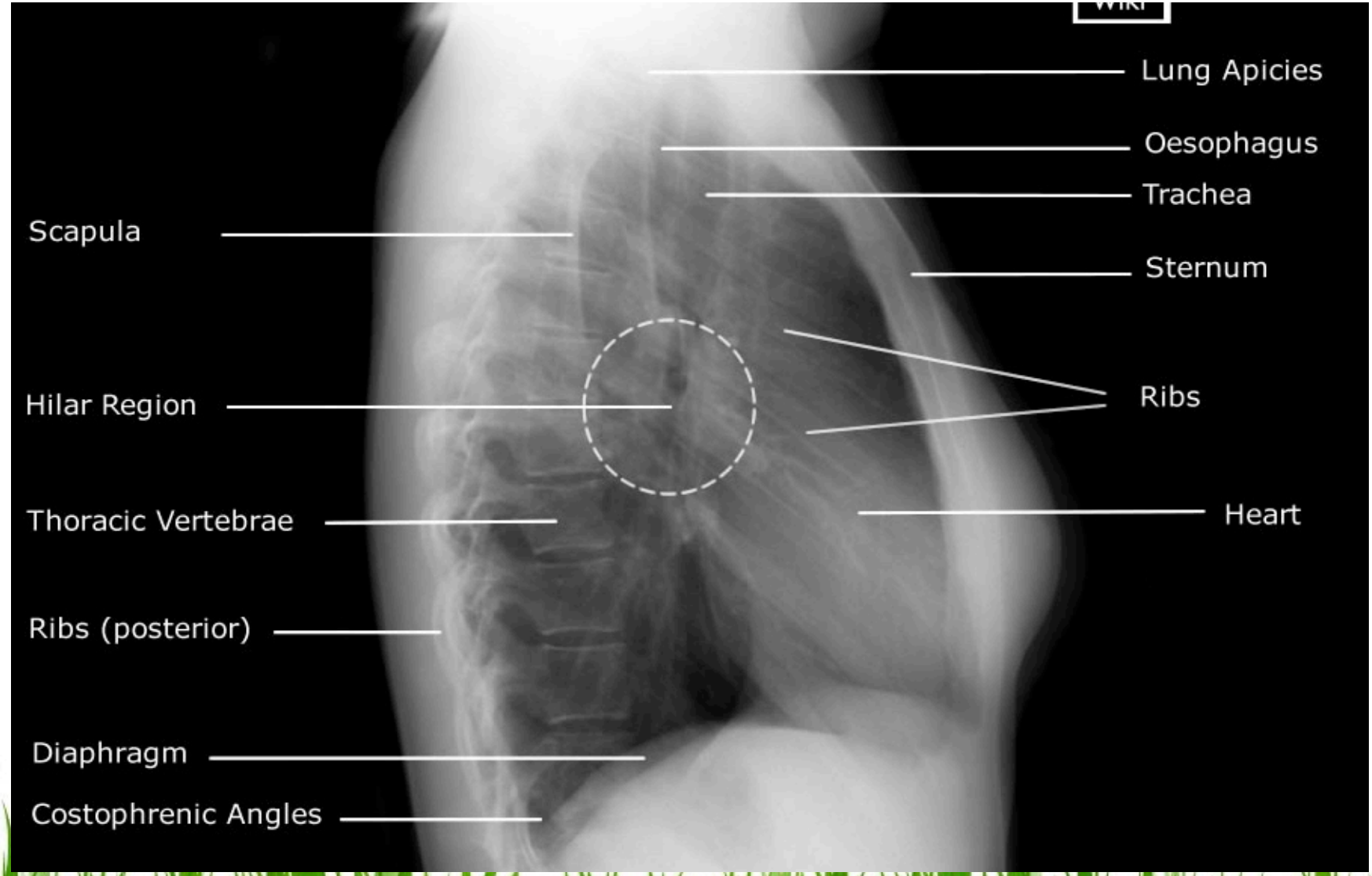
Lateral and Decubitus Films

Other views

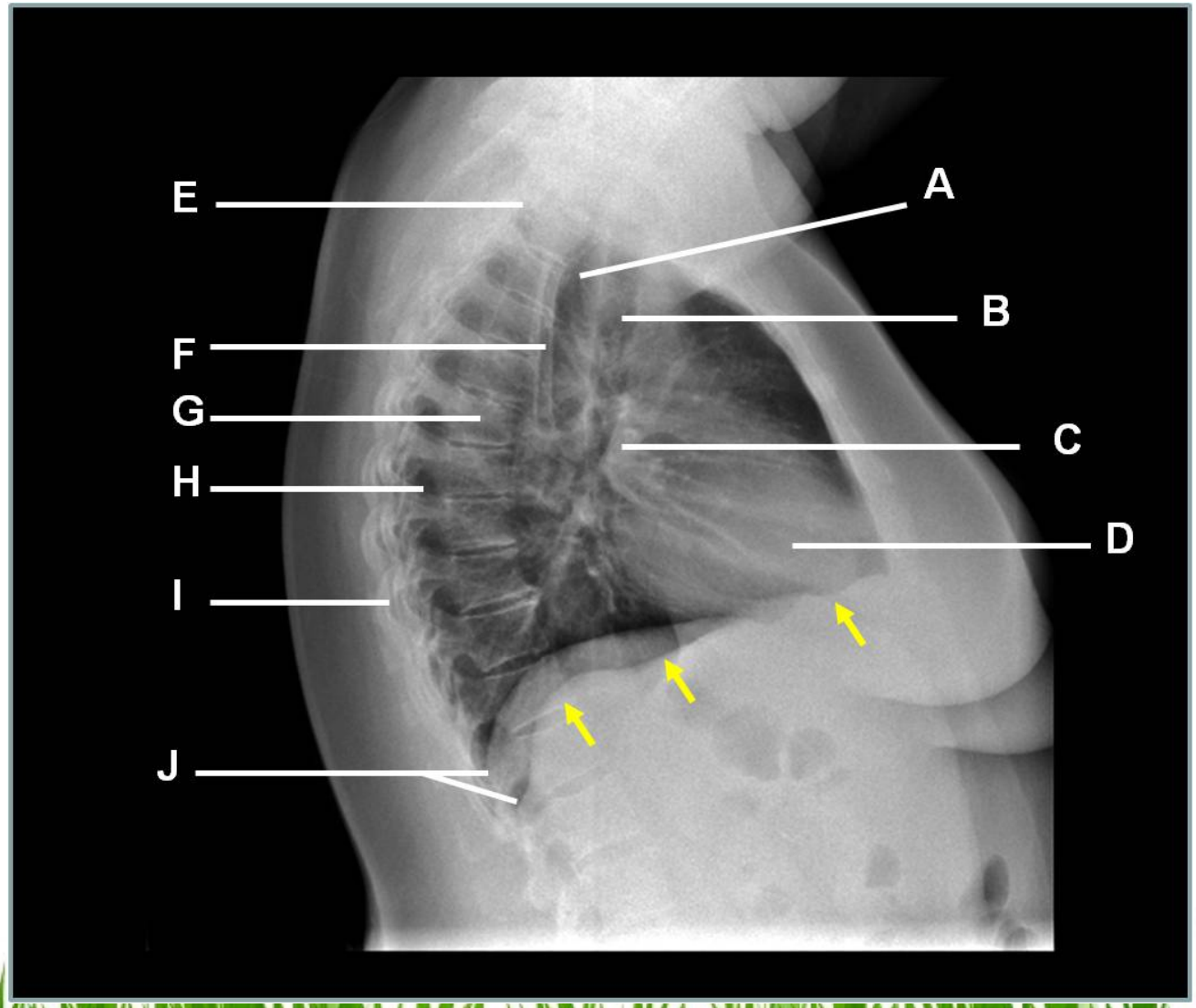
Lateral CXR

Lateral decubitus CXR

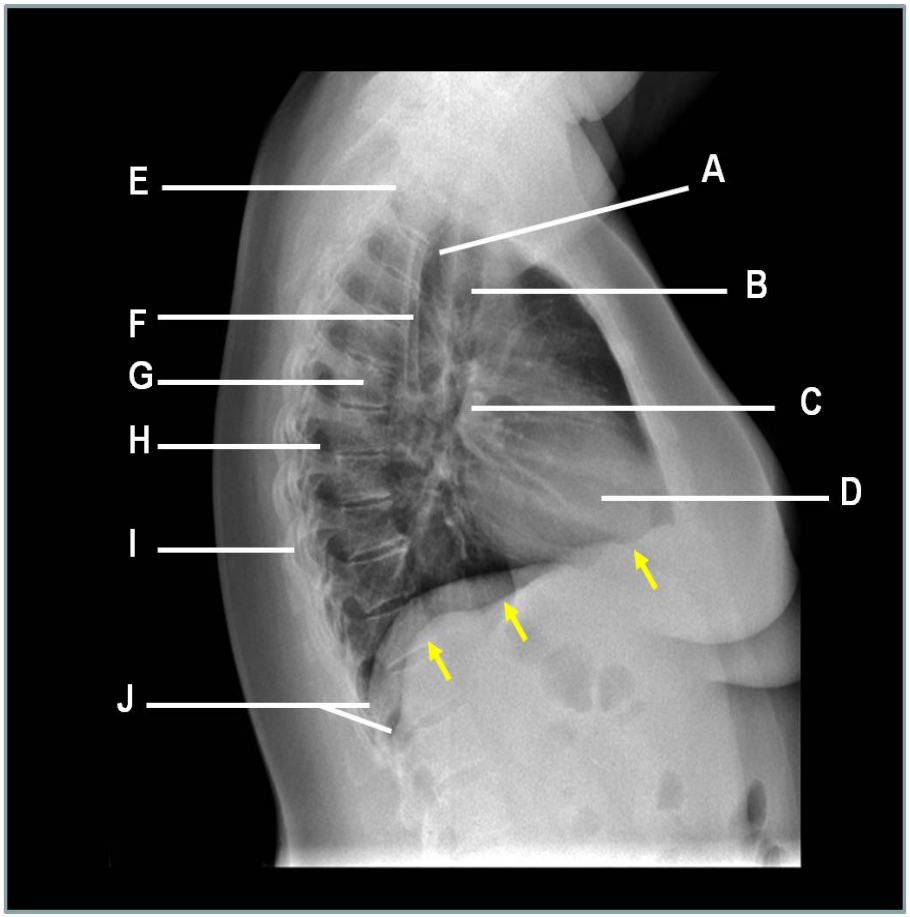
Lateral Radiograph



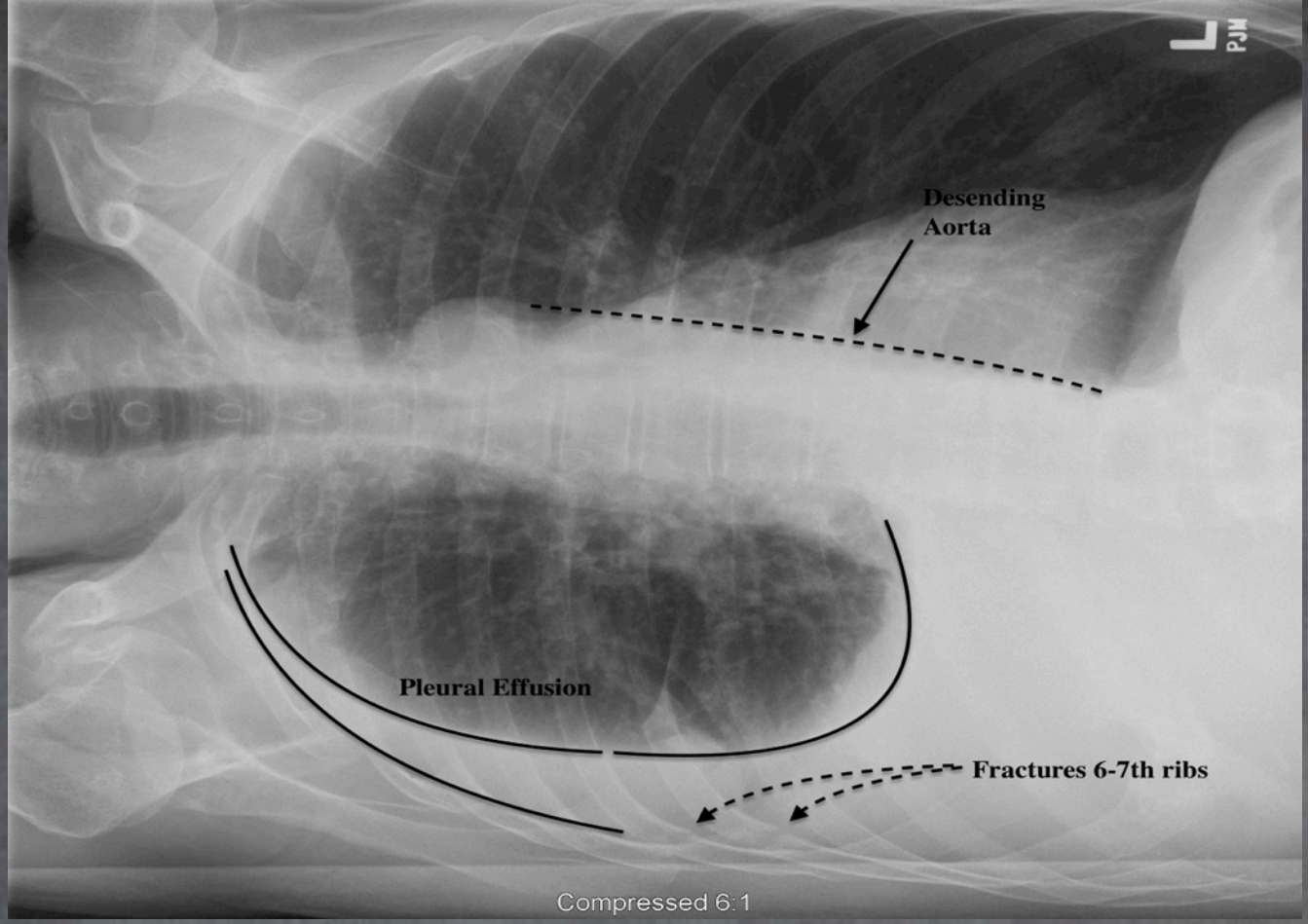
Test your knowledge



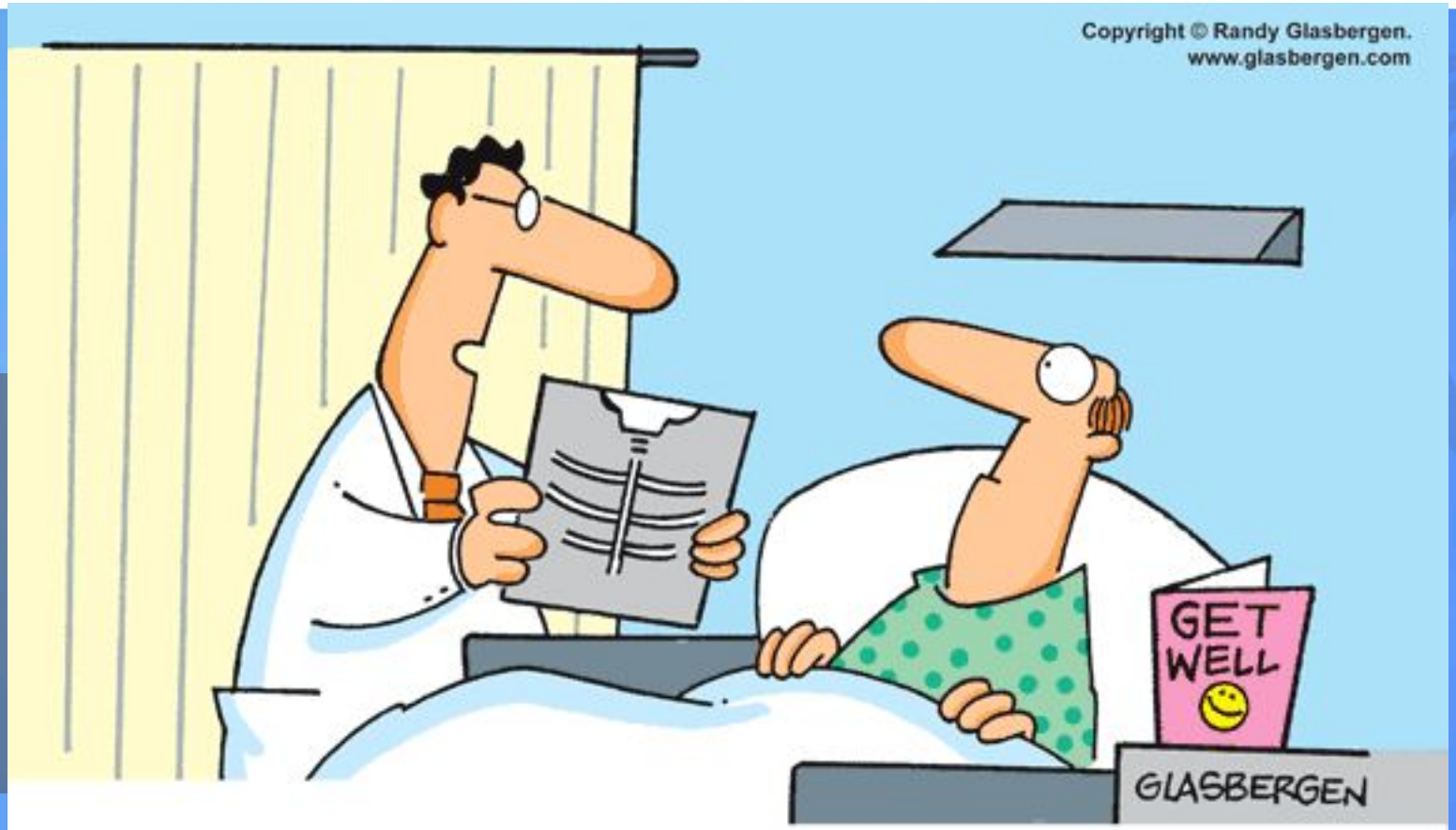
Test your knowledge



- esophagus (A),
- trachea (B),
- lung hili (C),
- heart silhouette (D),
- lung apices (E),
- scapulae (F),
- thoracic vertebra (G),
- thoracic intervertebral foramen (H),
- superimposed posterior ribs (I),
- costophrenic angles (J),
- and diaphragm (yellow arrows).



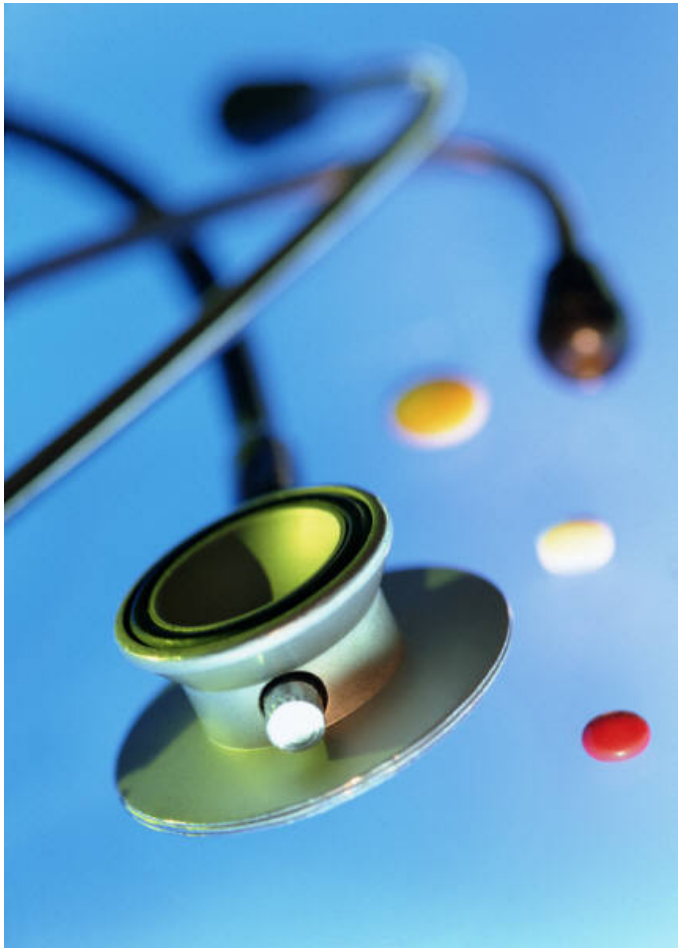
Lateral Decubitus Film



"Your x-ray showed a broken rib, but we fixed it with Photoshop."

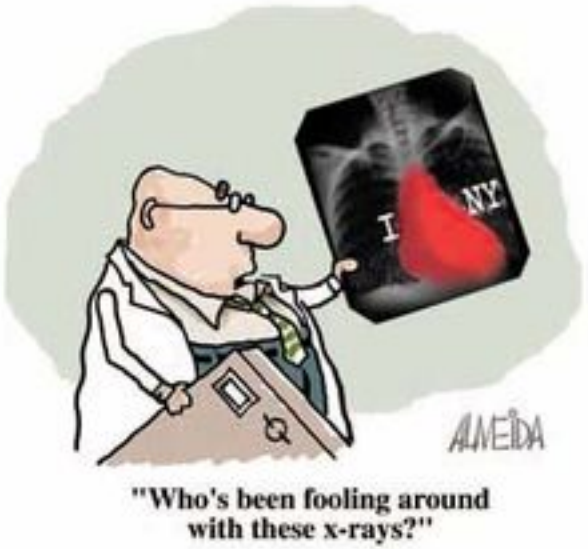
Steps to CXR Interpretation

- → **LABEL**
- → **ORIENTATION**
- → **LANDMARKS**
- → **ARTIFACT**
- → **PATHOLOGY**

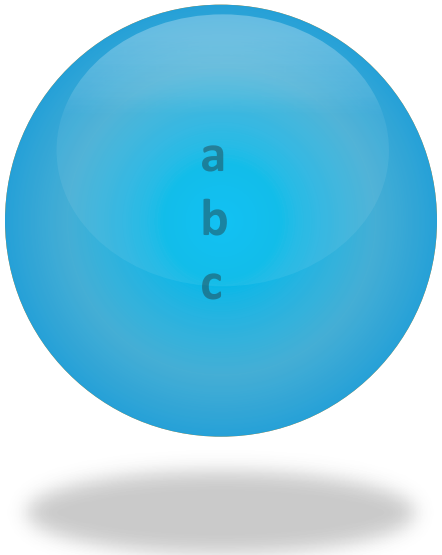


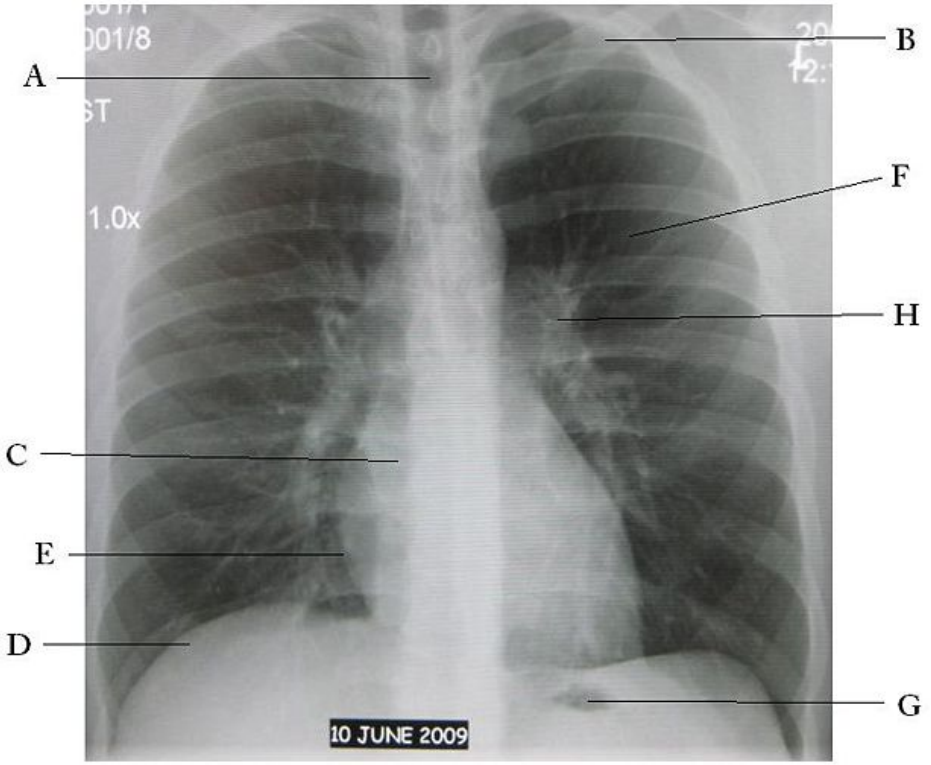
A – B- C way of Reviewing Chest Radiographs

- A- Airway
- B- Bones
- C- Cardiac
- D- Diaphragm
- E- Everything Else
- F- Fields (lung)
- G-Gastric
- H-Hilum



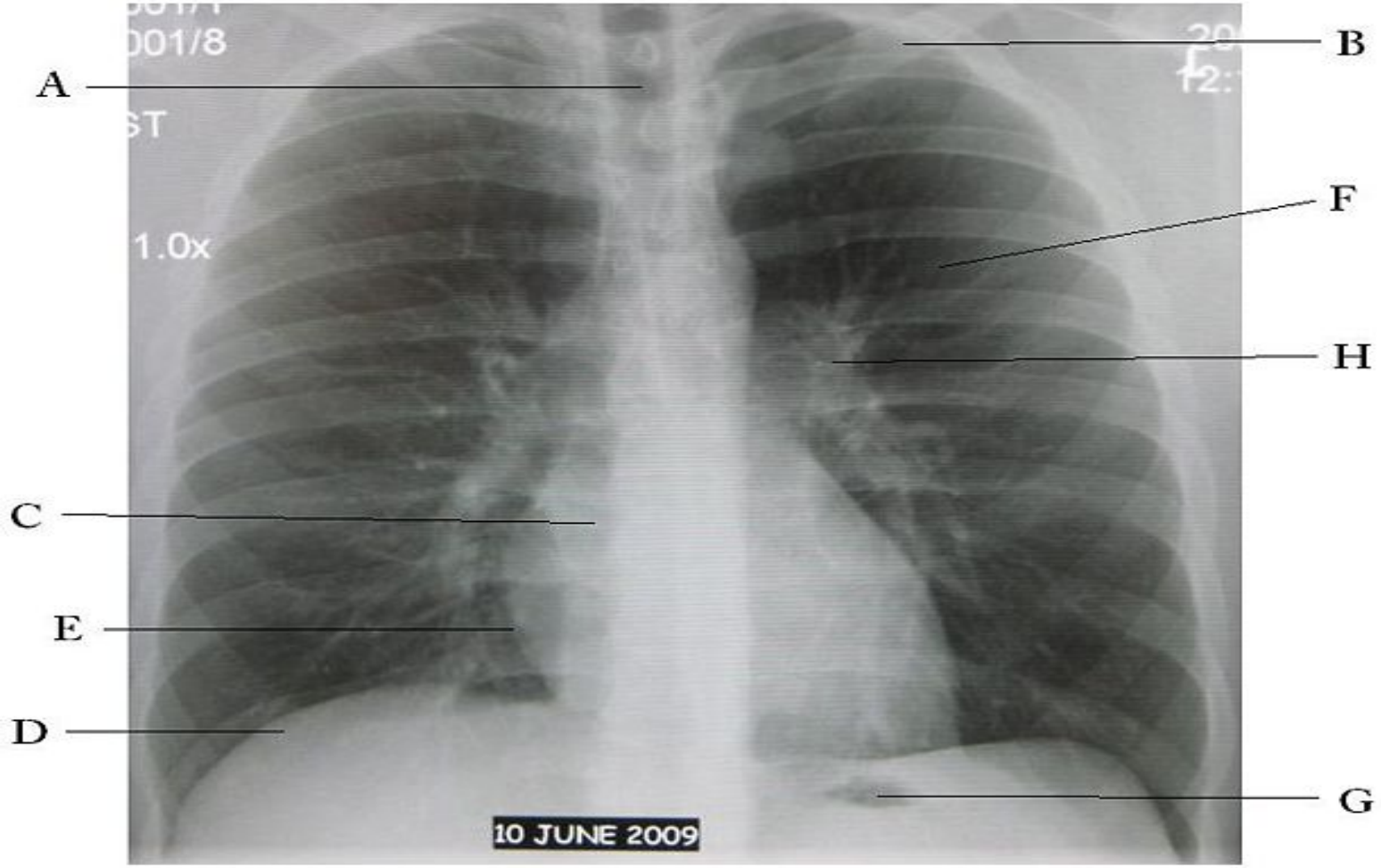
"Who's been fooling around with these x-rays?"



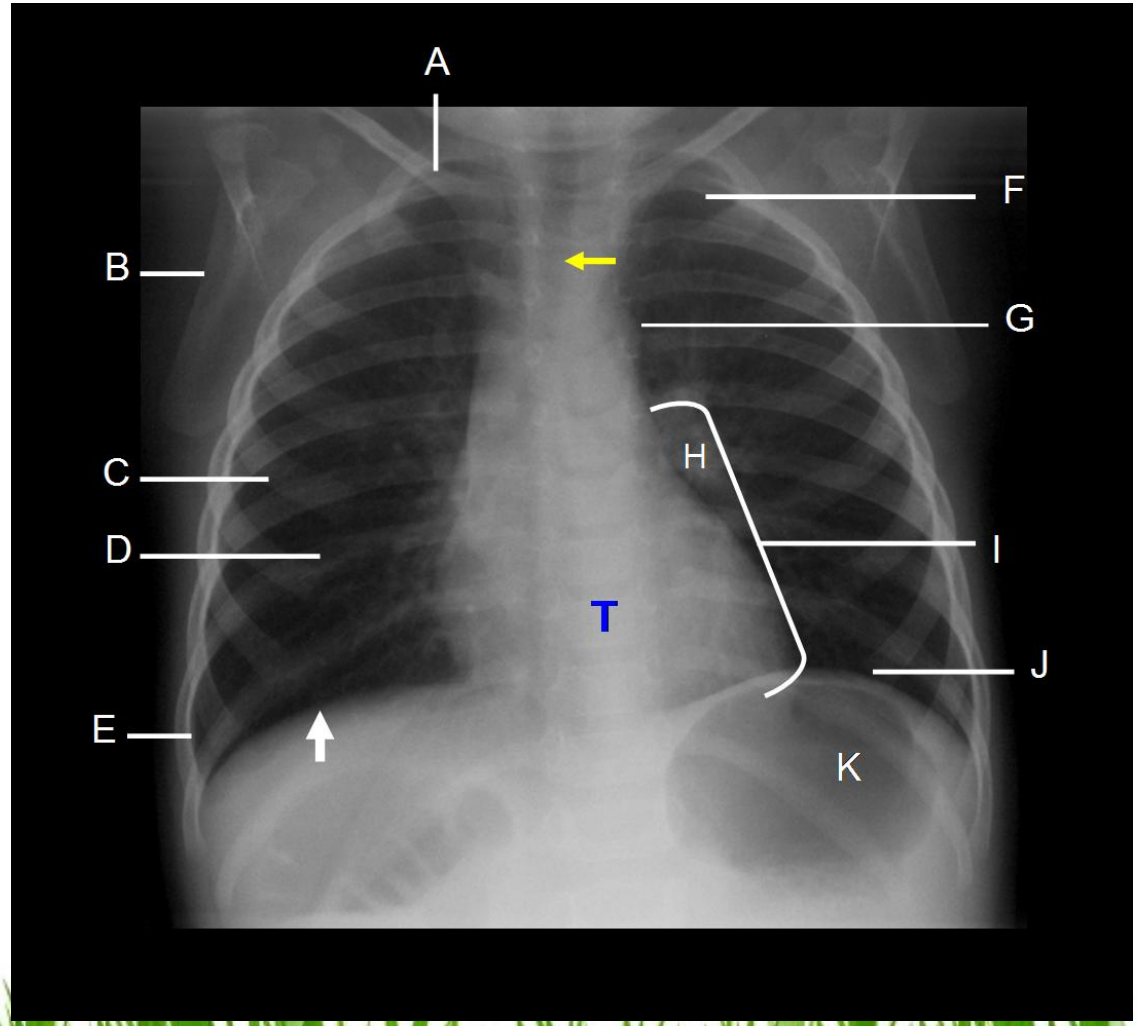


Now you will learn your ABC's

A
B
C
D
E
F
G
H



Lets test your knowledge



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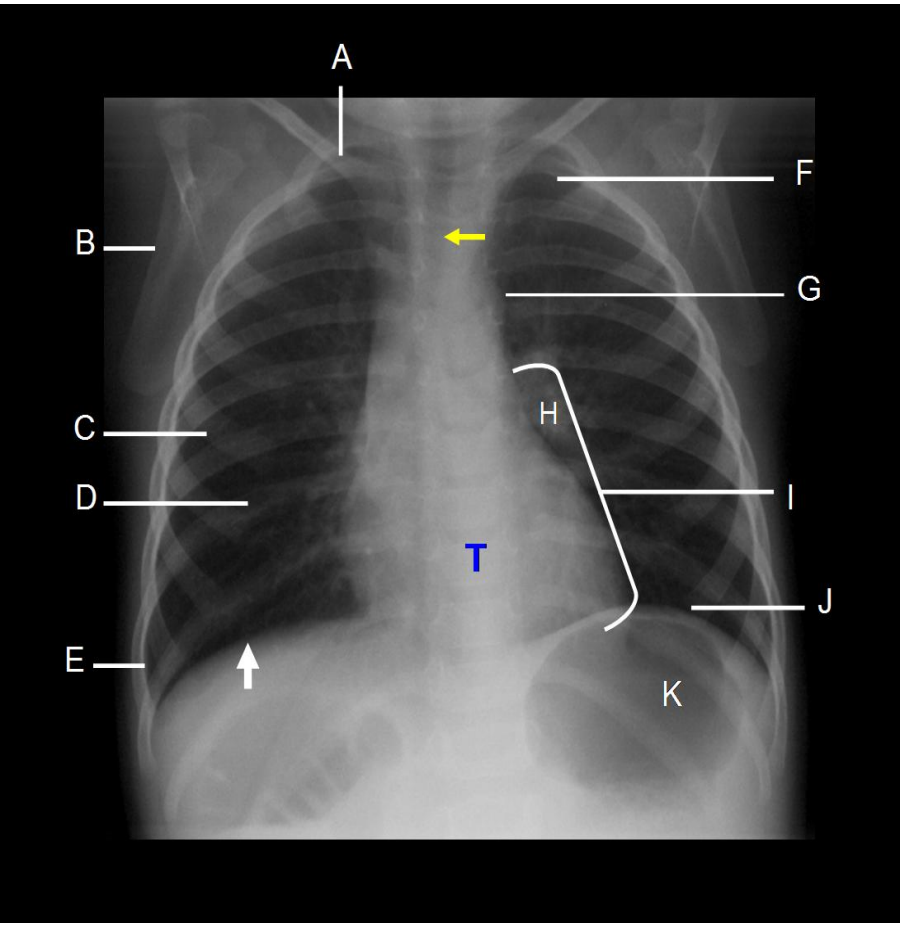
Lets test your knowledge



- Right clavicle (A),
- right scapula (B),
- right fourth anterior rib (C),
- right eighth rib (D),
- right costophrenic angle (E),
- left lung apex (F),
- aortic arch (G),
- hilum (H),
- heart (I),
- left lung base (J),
- right hemidiaphragm (white arrow).

http://www.ceessentials.net/images/critiqueChest/image0024.jpg

CXR Case Studies


You must
clearly explain
your problem

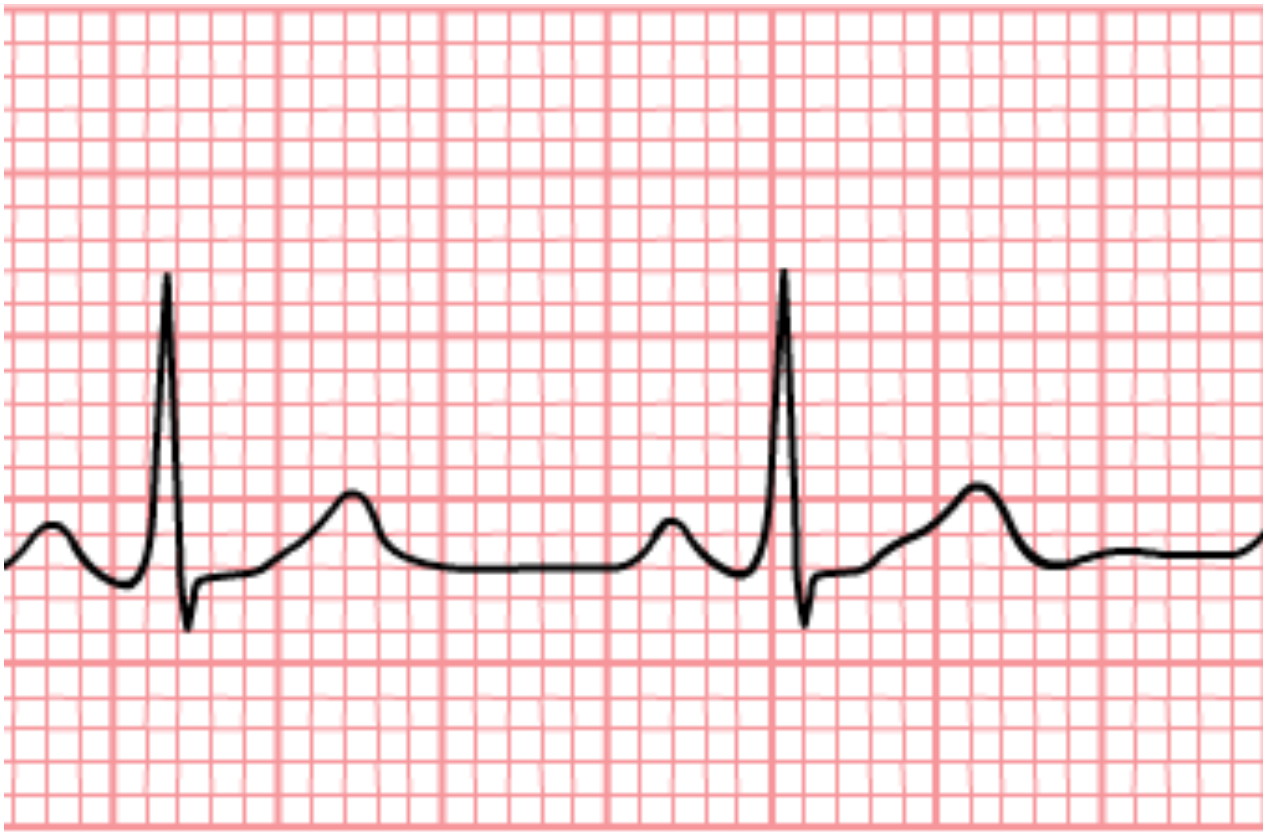
12 – Lead EKG

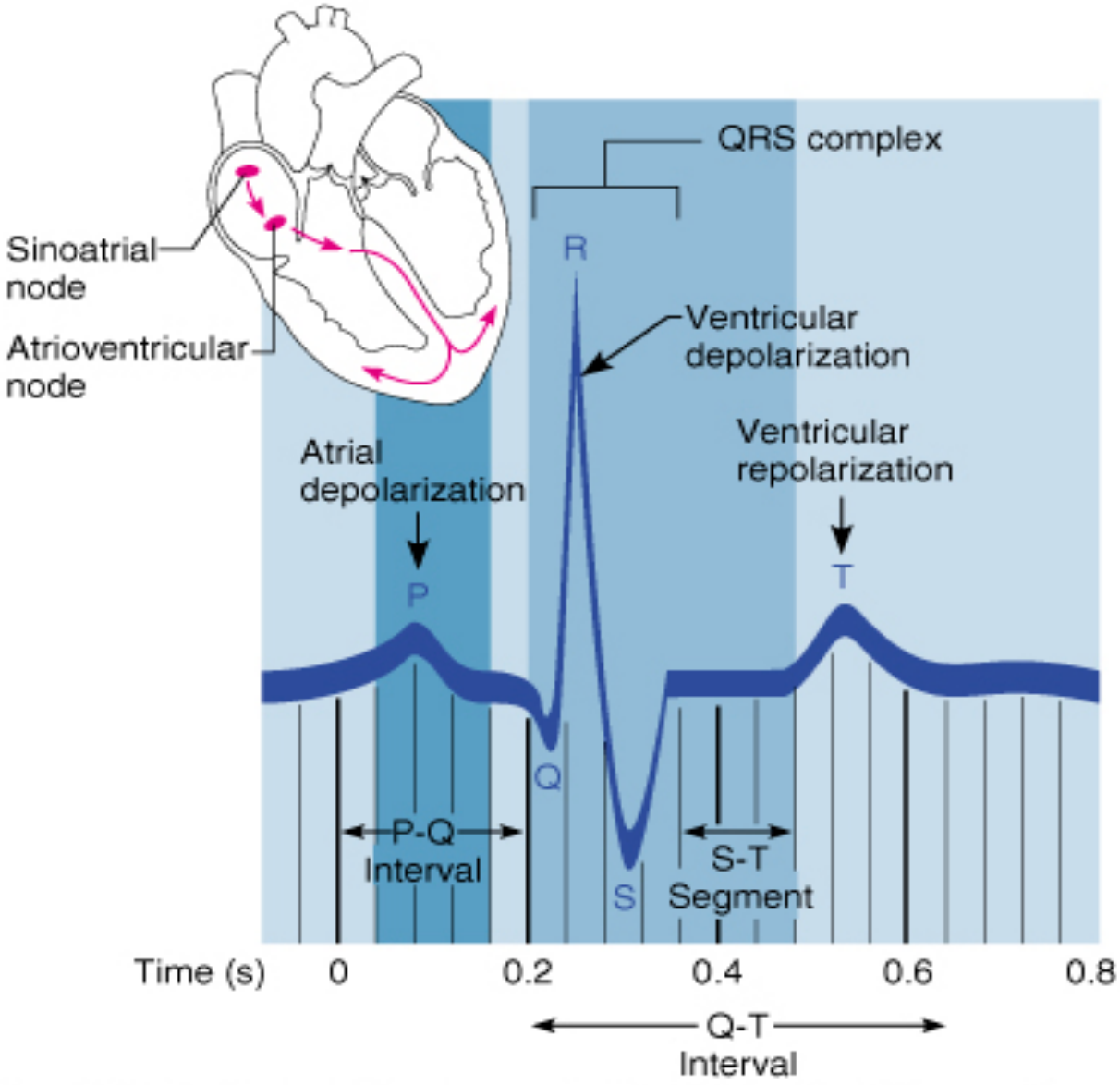
Objectives:

1. Describe the basic electrical conduction of the heart on ECG
2. Describe ECG changes of ischemia, injury & necrosis
3. Interpret in a step-wise approach

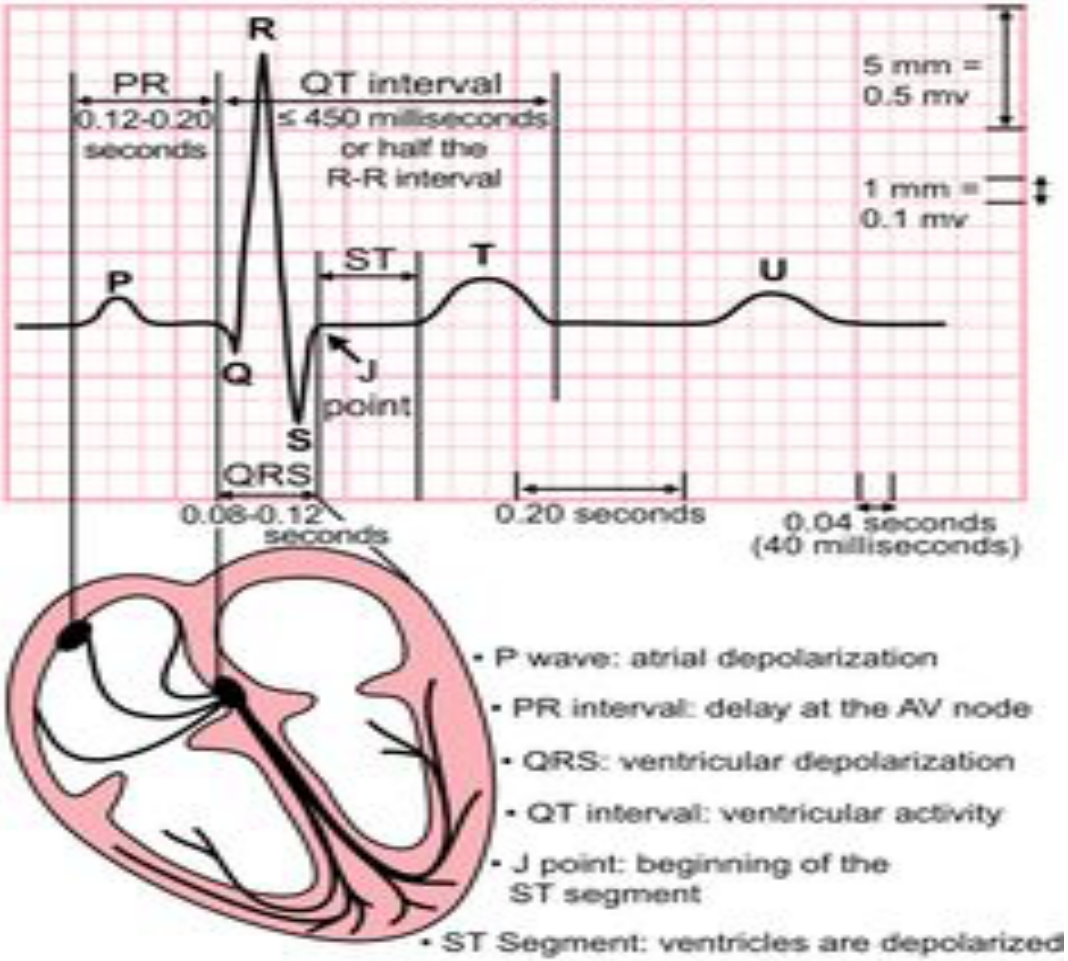
The 7 + 2 Step Approach to ECG

- Rhythm
- Rate
- Conduction
- Axis
- P wave
- QRS
- ST
- Compare
- Conclusion



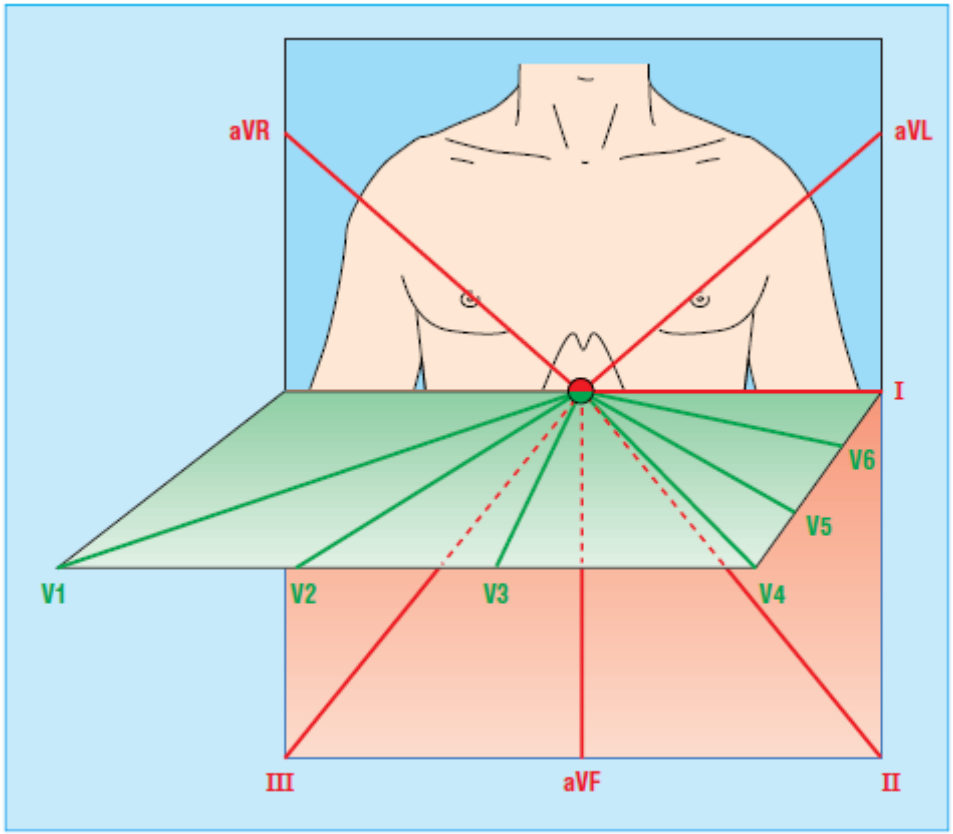


The Normal ECG



- P wave: atrial depolarization
- PR interval: delay at the AV node
- QRS: ventricular depolarization
- QT interval: ventricular activity
- J point: beginning of the ST segment
- ST Segment: ventricles are depolarized
- T wave: ventricular repolarization
- U wave: repolarization of Purkinje

ECG interpretation for MI

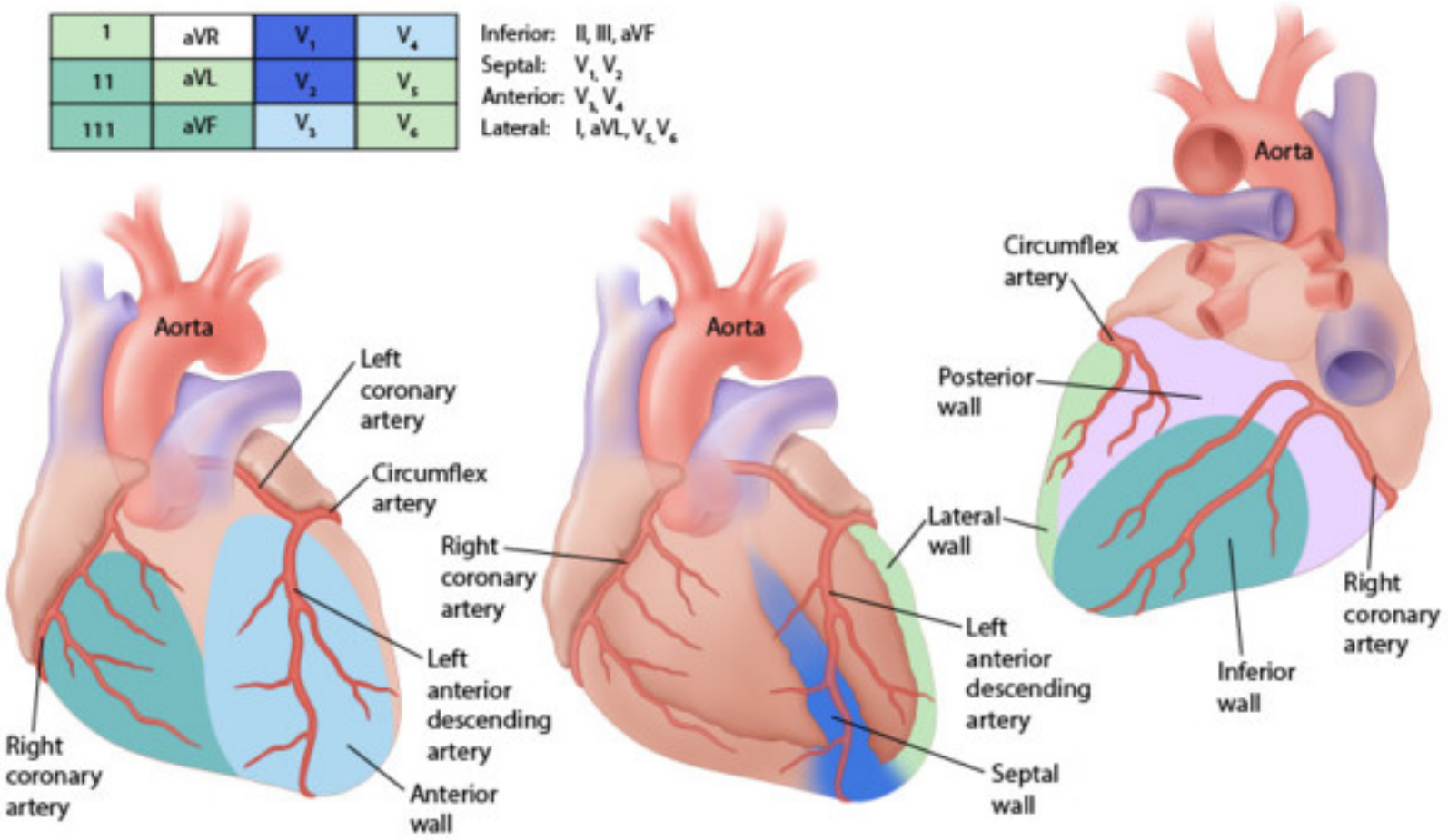


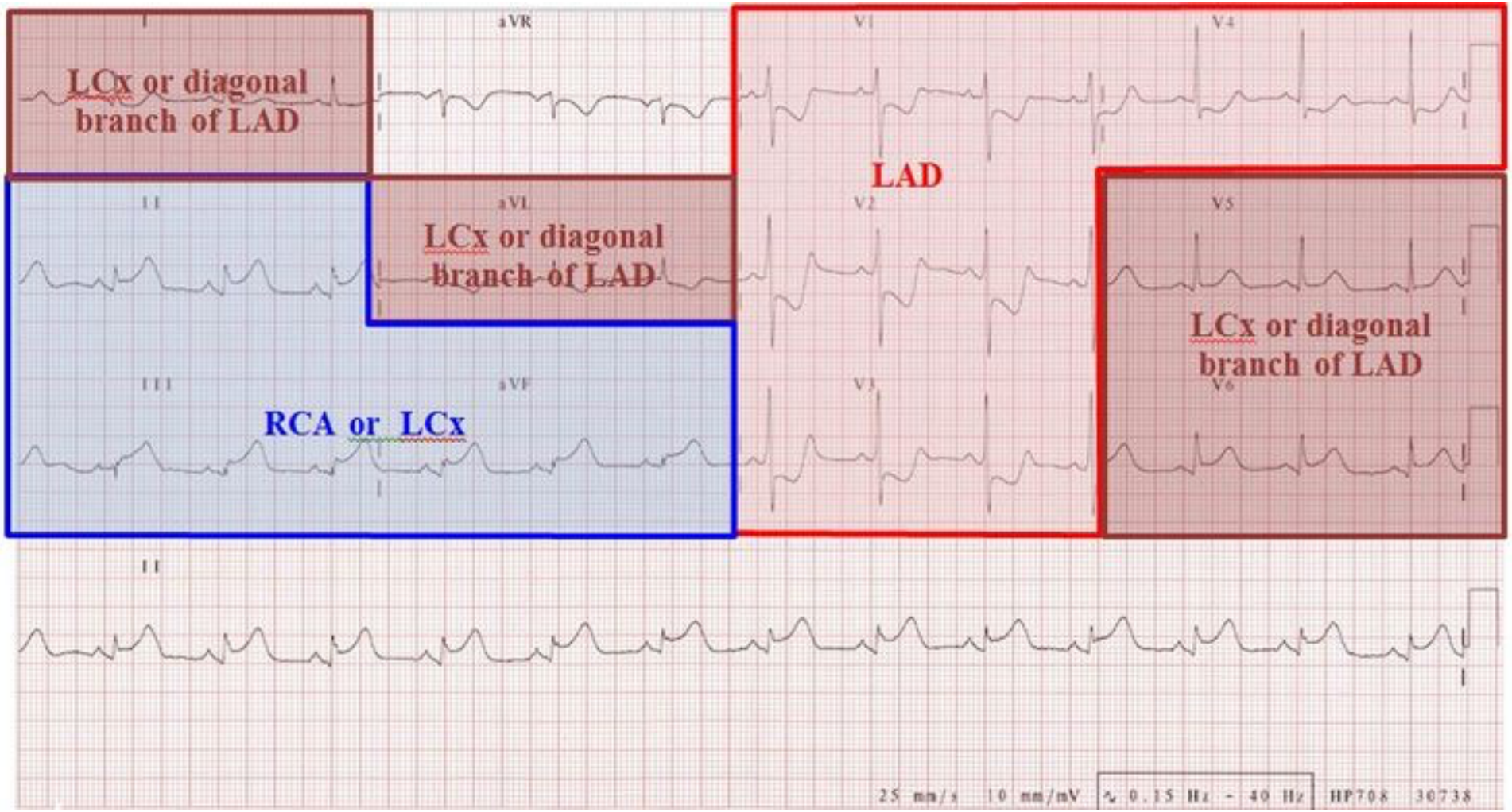
Vertical and horizontal perspective of the leads. The limb leads "view" the heart in the vertical plane and the chest leads in the horizontal plane

- **Perspective of ECG Leads**
  - Anterior: V3 & V4
  - Septal: V1 & V2
  - Lateral: I, aVL, V5 & V6
  - Inferior: II, III & aVF
  - Posterior: ST depression or prominent R waves (reciprocal changes) in V1 or V2
  - RV: Right side leads (V4-6R)

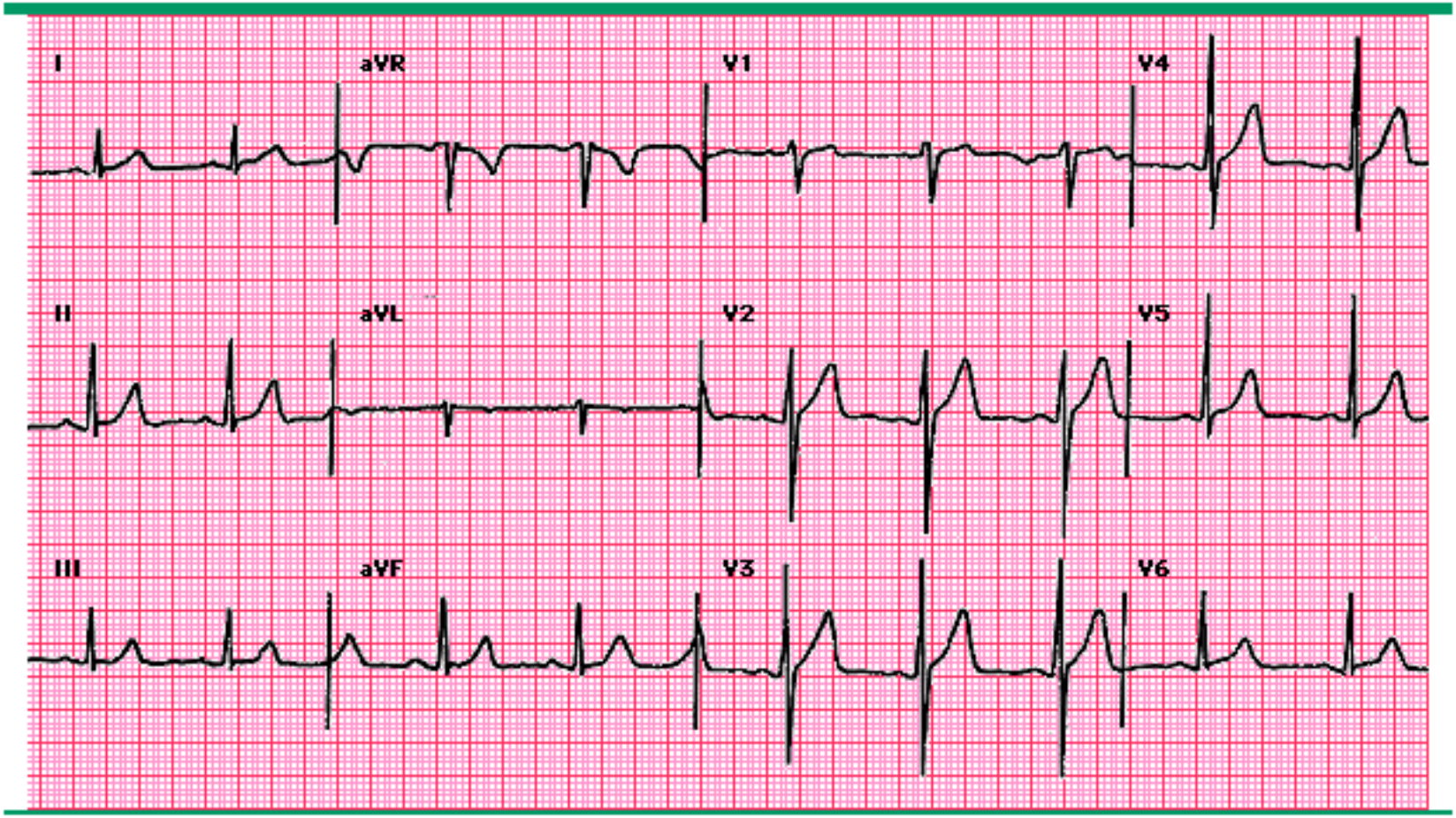
1	aVR	V_1	V_4
11	aVL	V_2	V_5
111	aVF	V_3	V_6

Inferior: II, III, aVF
Septal: V_1, V_2
Anterior: V_3, V_4
Lateral: I, aVL, V_5, V_6




I
aVR
V1
V4
LCx or diagonal branch of LAD
LAD
II
aVL
V2
V5
LCx or diagonal branch of LAD
LCx or diagonal branch of LAD
III
aVF
V3
V6
RCA or LCx
II
25 mm/s
10 mm/mV
0.15 Hz - 40 Hz
HP708
30738

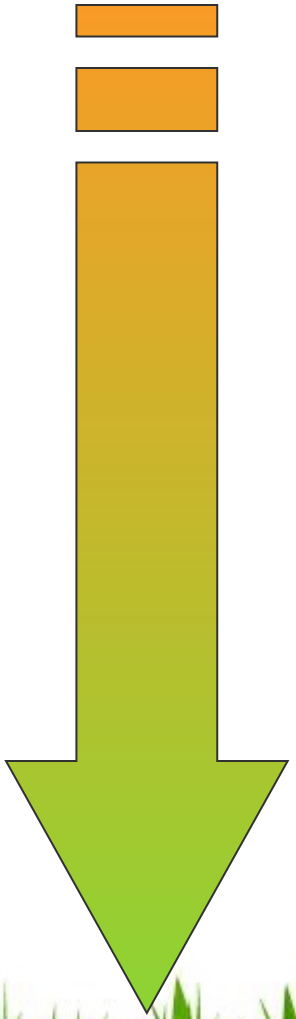
Normal ECG



Normal electrocardiogram showing normal sinus rhythm at a rate of 75 beats/min, a PR interval of 0.14 sec, a QRS interval of 0.10 sec, and a QRS axis of approximately 75°.

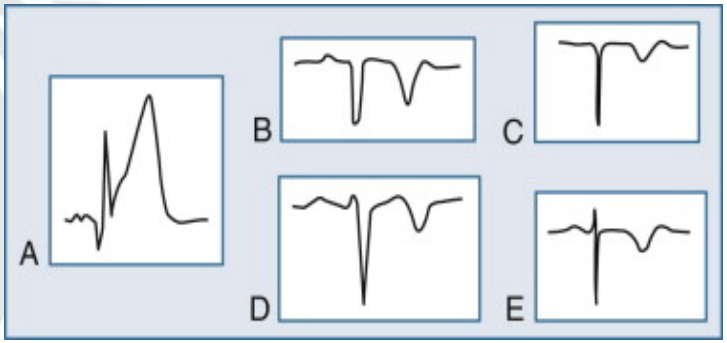
Courtesy of Ary Goldberger, MD.

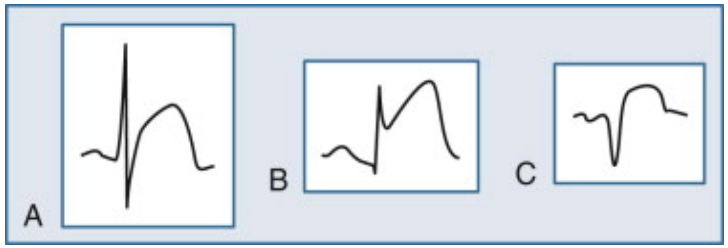
ECG interpretation for MI

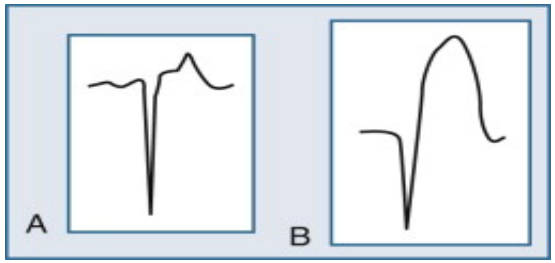


- **Progression of ECG Appearance**
  - *Ischemia*: T wave inversion
  - *Injury*: ST elevation
  - *Infarct*: Tombstone shaped ST elevation begin to see Q waves
  - *Chronic*: ST elevation disappears, T becomes upright, permanent Q waves

ECG-MI Findings







STEMI and NSTEMI

- **STEMI**:
  - New ST elevation at the J point in two anatomically contiguous leads
  - Using the following diagnostic thresholds: ≥0.1 mV (1 mm) in all leads other than V2-V3, where the following diagnostic thresholds apply: ≥0.2 mV (2 mm) in men 40 years; ≥0.25 mV (2.5 mm) in men <40 years, or ≥0.15 mV (1.5 mm) in women.
- **Non ST elevation MI or unstable angina**:
  - New horizontal or down-sloping ST depression ≥0.05 mV (0.5 mm) in two anatomically contiguous leads and/or T inversion ≥0.1 mV (1 mm) in two anatomically contiguous leads with prominent R wave or R/S ratio >1.

ECG interpretation for MI

- **ST elevation MI** — STEMI
  - ECG evolves through a typical sequence
  - Earliest change is development of a hyperacute or peaked T wave
    - reflects localized hyperkalemia
  - ST segment elevates in leads recording electrical activity of involved myocardial region”
    - Initially, elevation of J point and ST segment is concave
    - Over time, ST segment elevation becomes more pronounced → becomes more convex (rounded upward)
    - 2 mm of ST segment elevation in precordial leads for men
    - 1.5 mm for women in precordial leads (tend to have less ST elevation) & greater than 1mm in other leads
  - ST segment may → indistinguishable from the T wave

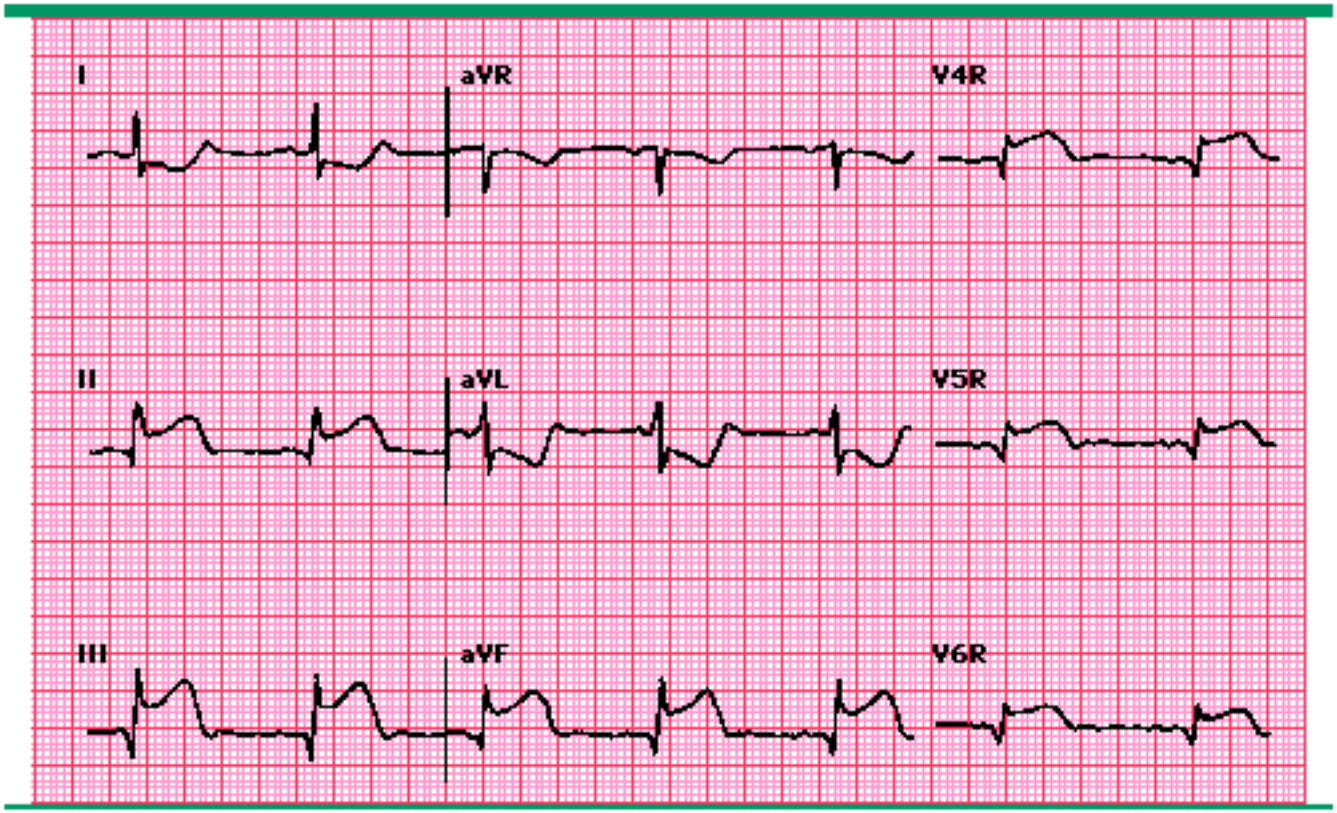
ECG interpretation for MI

- **ST elevation MI** — STEMI (cont.)
  - ECG evolves through a typical sequence (cont.)
  - An initial Q wave or abnormal R wave develops over several hours to days
  - Over time there is further evolution……(hours to 2 weeks)
    - ST segment gradually returns to isoelectric baseline
    - R wave amplitude becomes markedly reduced
    - Q wave deepens
    - T wave becomes inverted

ECG interpretation for MI

- **Non-STEMI**
  - ST depressions and/or T wave inversions without ST segment elevations or pathologic Q waves
  - ST-T wave abnormalities may be present diffusely in many leads
    - more commonly localized to leads associated with region of ischemic myocardium
  - Two forms of non-ST depression
    - frequently indistinguishable initially (prior to biomarker elevation)
    - Non-STEMI
      - ST segment depressions evolve over days
      - residual ST segment depression and T wave inversions
      - No formation of pathologic Q waves
    - Unstable Angina (UA)
      - ST segment and T wave changes usually resolve completely

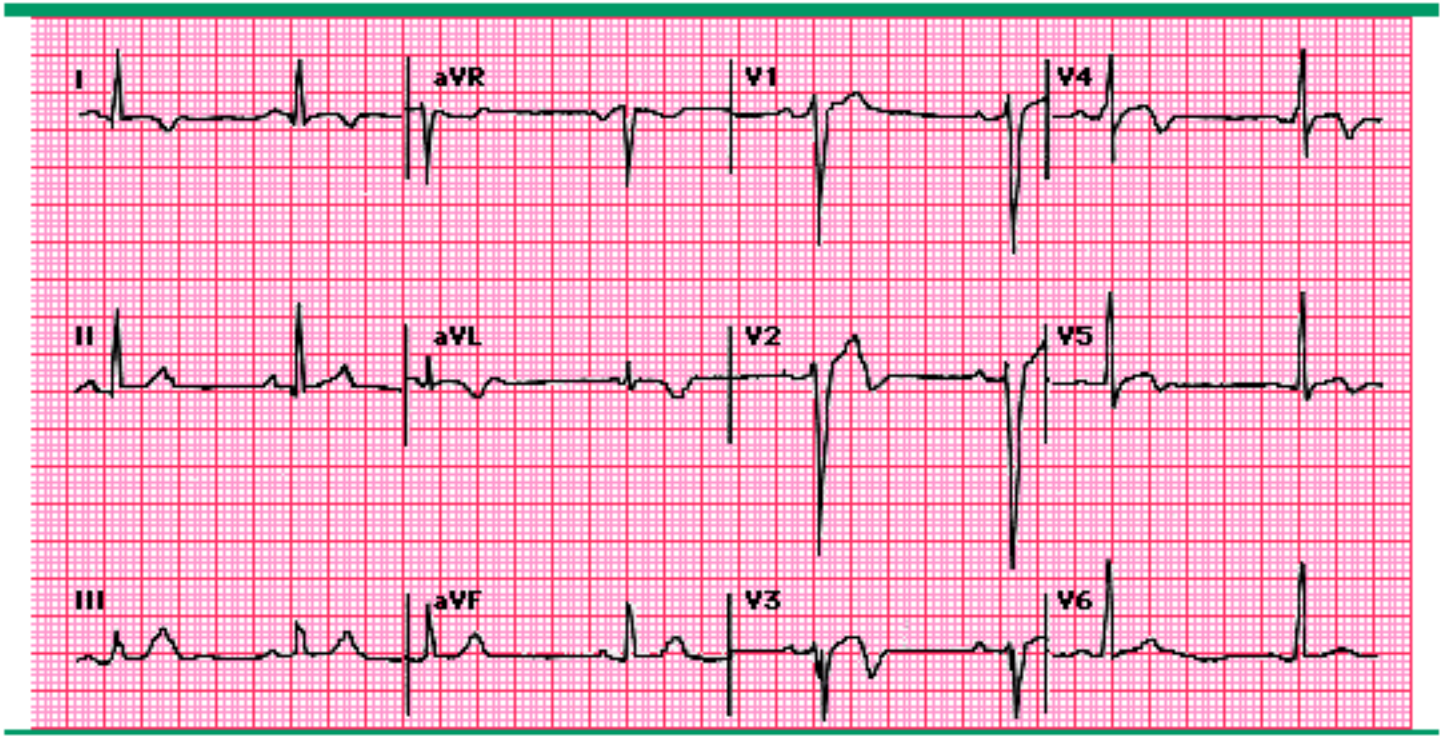
ECG of acute inferior and right ventricular myocardial infarction



Electrocardiogram shows Q waves and prominent doming ST segment elevation in II, III, and aVF, findings which are characteristic of an acute inferior myocardial infarction. ST elevation in the right precordial leads - V4R, V5R, and V6R - indicates right ventricular involvement as well (arrows). The ST depressions in leads I and aVL represent reciprocal changes.

Courtesy of Ary Goldberger, MD.

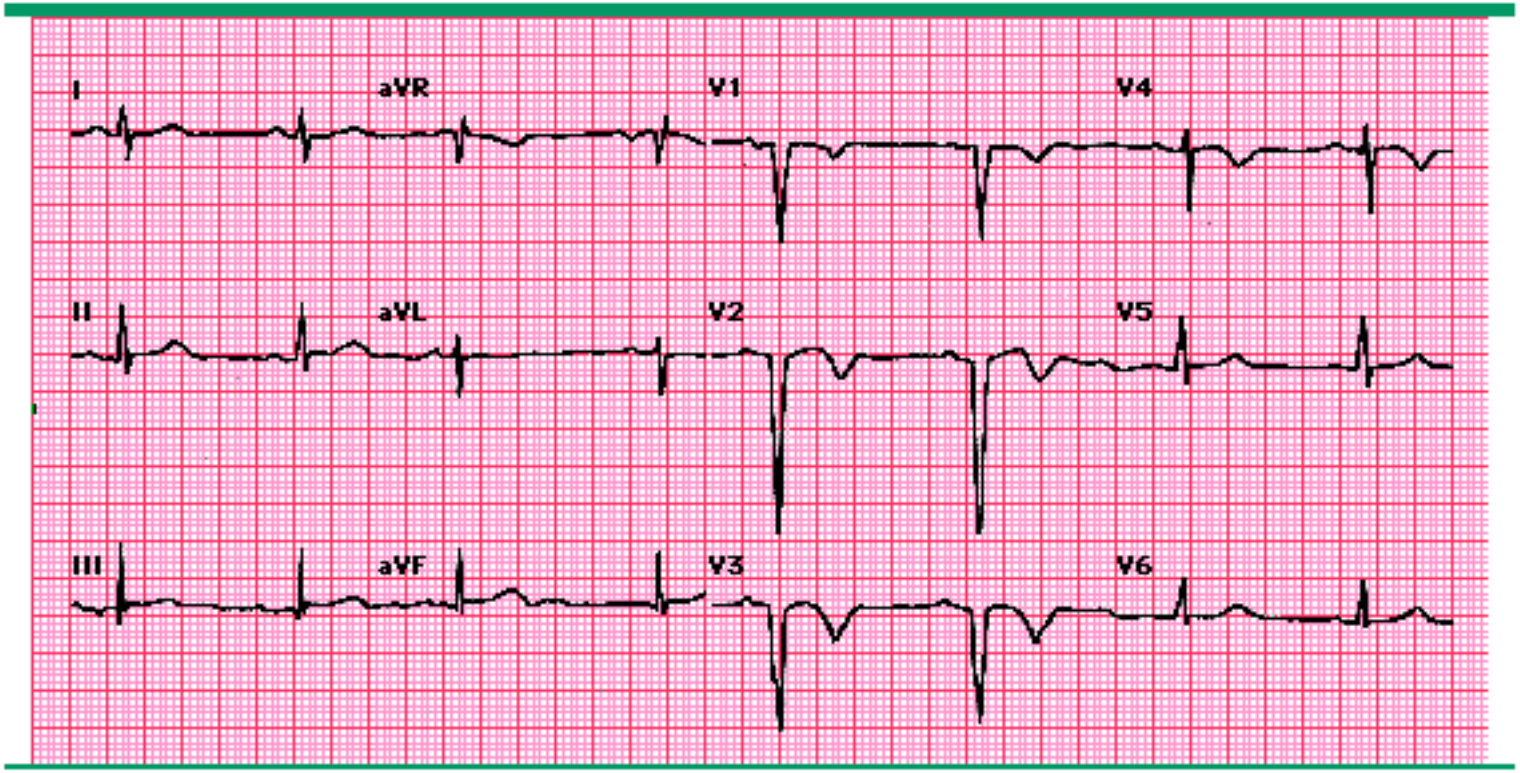
Electrocardiogram (ECG) in an evolving anterior myocardial infarction



Electrocardiogram shows findings typical of an evolving Q-wave anterior MI: loss of R waves in leads V1 to V3, ST segment elevations in V2 to V4, and T wave inversions in leads I, aVL, and V2 to V5. Sinus bradycardia (55 beats/min) is present due to concurrent therapy with a beta blocker.

Courtesy of Ary Goldberger, MD.

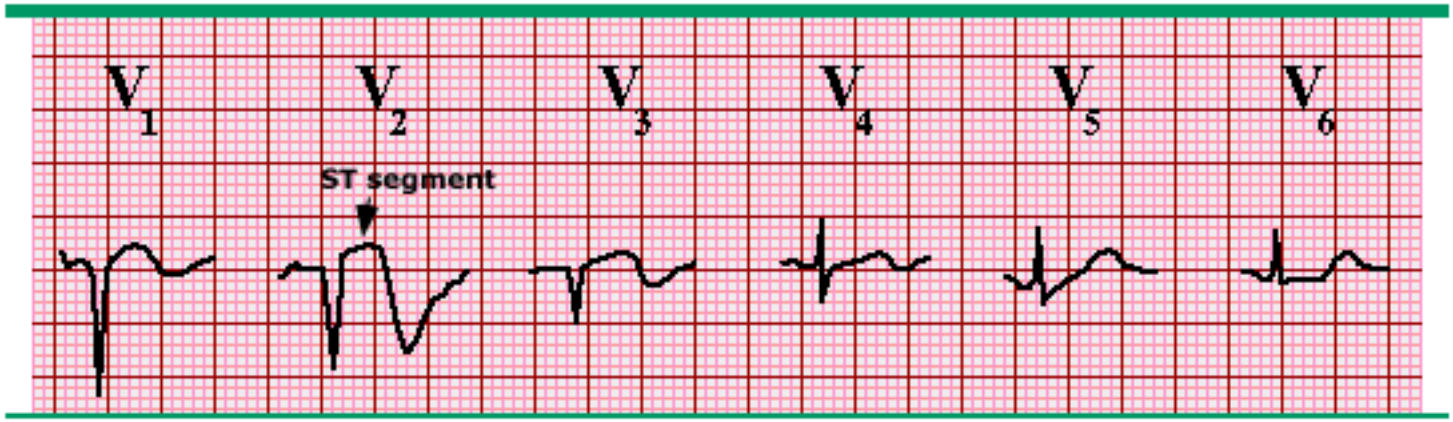
Electrocardiogram (ECG) late in the evolution of an anterior myocardial infarction



Later stage in the evolution of an acute anterior myocardial infarction. There is a QS pattern in leads V1 to V3 and T wave inversion in leads V2 to V4. The ST segment elevations in these leads have almost disappeared.

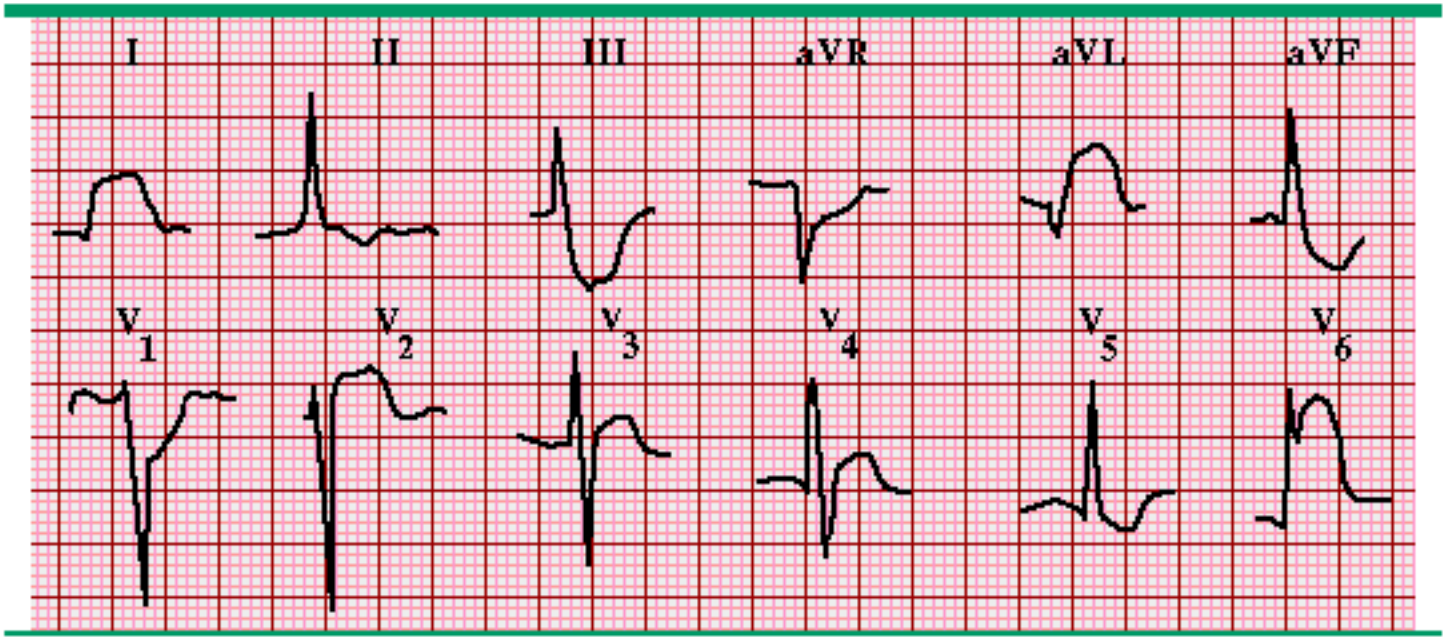
Courtesy of Ary Goldberger, MD.

Acute anteroseptal myocardial infarction



ST segment elevation in V1 and V2 is characteristic of an acute anteroseptal infarct. There is also reciprocal ST segment depression in V5 and V6.

Acute lateral transmural myocardial infarction



ST elevation in leads I and aVL is characteristic of an acute lateral wall infarct. Reciprocal ST depression is evident in this case in the inferior leads (II, III, and aVF) and in V1.

UpToDate®

ECG changes associated with prior myocardial infarction

Any Q-wave in leads V_2-V_3 ≥0.02 s or QS complex in leads V_2 and V_3
Q-wave ≥0.03 s and ≥0.1 mV deep or QS complex in leads I, II, aVL, aVF, or V_4-V_6 in any two leads of a contiguous lead grouping (I, aVL,V_6; V_4-V_6; II, III, and aVF)*
R-wave ≥0.04 s in V_1-V_2 and R/S ≥1 with a concordant positive T-wave in the absence of a conduction defect

* The same criteria are used for supplemental leads V_7-V_9, and for the Cabrera frontal plane lead grouping.

Reproduced with permission from: Thygesen, K, Alpert, JS, White, HD, et al. Universal definition of myocardial infarction: Kristian Thygesen, Joseph S. Alpert and Harvey D. White on behalf of the Joint ESC/ACCF/AHA/WHF Task Force for the Redefinition of Myocardial Infarction. Eur Heart J 2007; 28:2525. Copyright ©2007 Oxford University Press.

Causes of Q waves

Physiologic or positional factors
Normal variant "septal" q waves
Normal variant Q waves in leads V1,V2, aVL, III, and aVF
Left pneumothorax or dextrocardia: loss of lateral precordial R wave progression
Myocardial injury or infiltration
Acute processes: myocardial ischemia or infarction, myocarditis, hyperkalemia
Chronic processes: myocardial infarction, idiopathic cardiomyopathy, myocarditis, amyloidosis, tumor, sarcoid, scleroderma, Chagas' disease, echinococcus cyst
Ventricular hypertrophy or enlargement
Left ventricle: slow R wave progression in which there are small or absent R waves in the mid-precordial leads
Right ventricle: reversed R wave progression in which there is a progressive decrease in R wave amplitude from V1 to the mid-lateral precordial leads, or slow R wave progression, particularly with chronic obstructive lung disease or acute pulmonary embolism
Hypertrophic cardiomyopathy - may simulate anterior, inferior, posterior, or lateral infarcts
Conduction abnormalities
Left bundle branch block - slow R wave progression in which there are small or absent R waves in the mid-precordial leads
Wolff-Parkinson-White patterns

Causes of ST segment elevation

Myocardial ischemia or infarction
Noninfarction, transmural ischemia (Prinzmetal's angina pattern or acute takotsubo syndrome)
Acute myocardial infarction (MI) usually due to coronary atherosclerosis or occasionally to other causes (eg, acute takotsubo syndrome)
Post-MI (ventricular aneurysm pattern)
Acute pericarditis
Normal variants (including benign early repolarization)
Left ventricular hypertrophy or left bundle branch block (V1-V2 or V3)
Other
Myocarditis (may look like MI or pericarditis)
Massive pulmonary embolism (leads V1-V2 in occasional cases)
Brugada-type patterns (V1-V3 with right bundle branch block-appearing morphology)
Myocardial tumor
Myocardial trauma
Hyperkalemia (only leads V1 and V2)
Hypothermia (J wave/Osborn wave)
Hypercalcemia (rarely)
Post-DC cardioversion (rarely)

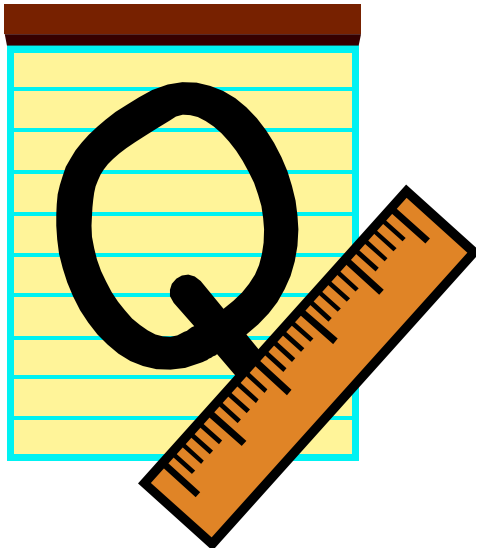
A 12-Lead Electrocardiogram from a Patient with Acute Pericarditis, Demonstrating Widespread ST-Segment Elevation and PR-Segment Depression

Lange, R. A. et al. N Engl J Med 2004;351:2195-2202


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

ECG interpretation for MI

- **Q waves or no Q waves**
  - **Q wave Infarct:**
    - *Usually* involving the entire myocardial wall (AKA "transmural")
    - Higher acute mortality, tissue damage and development of CHF
  - **Non-Q wave Infarct:**
    - *Usually* small enough not to produce Q waves but big enough to cause ST elevation
    - Damage may be anywhere in the myocardial wall
    - Higher long-term mortality without aggressive tx



ECG

- Repeated Q 20-30 minutes
  - Initial ECG nondiagnostic in 45% and normal in 20% of those subsequently shown to have AMI
- LBBB or Pacemaker
  - New LBBB
  - Many do not have CP

MI +LBBB: Indications for Reperfusion Therapy

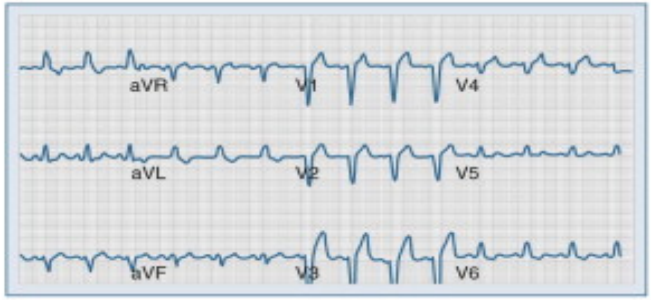
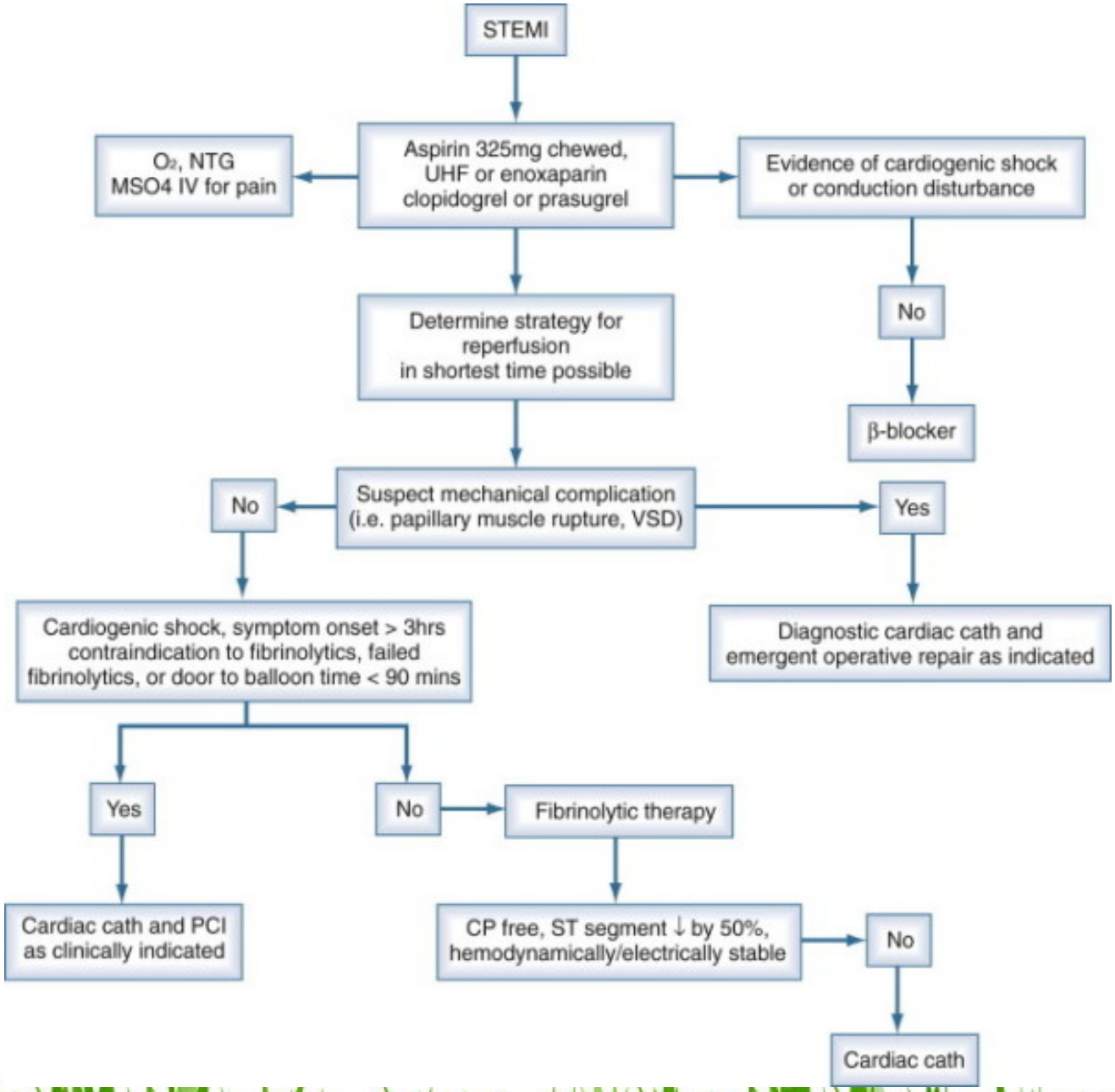
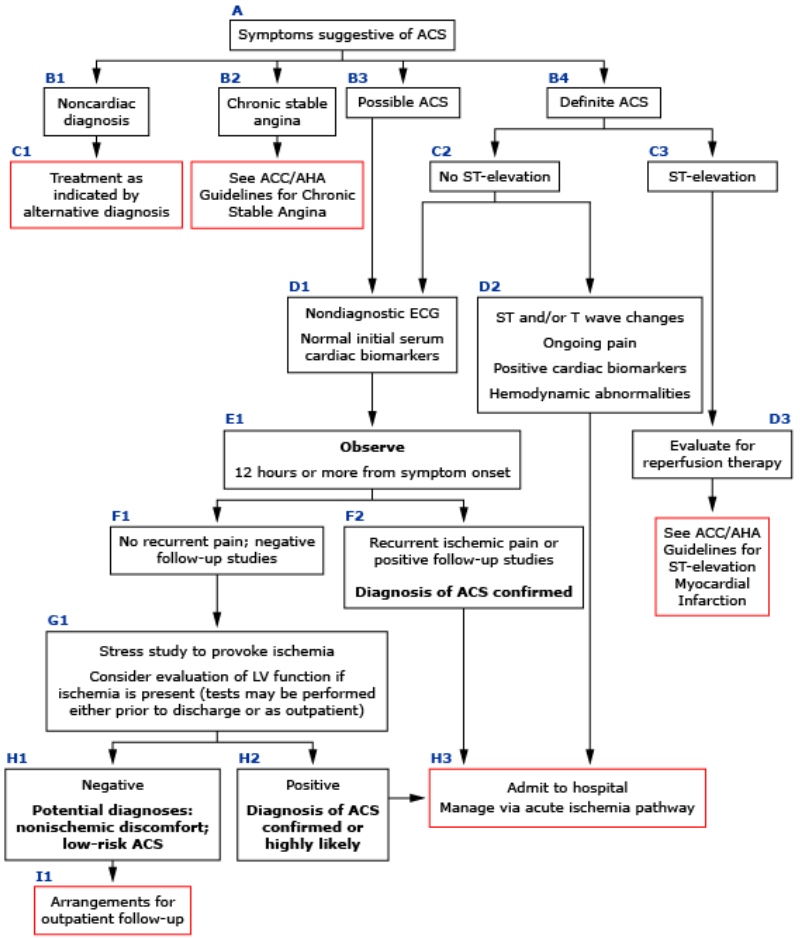
ECG finding	Comment	Example
New LBBB	New onset and with appropriate clinical correlation	
Concordant ST segment elevation	ST segment elevation >1 mm//concordant with QRS complex	
Concordant ST segment depression in leads V1, V2, and/or V3	ST segment depression >1 mm in leads V1, V2, or V3	
Discordant ST segment elevation	ST segment elevation >5 mm discordant with QRS complex	

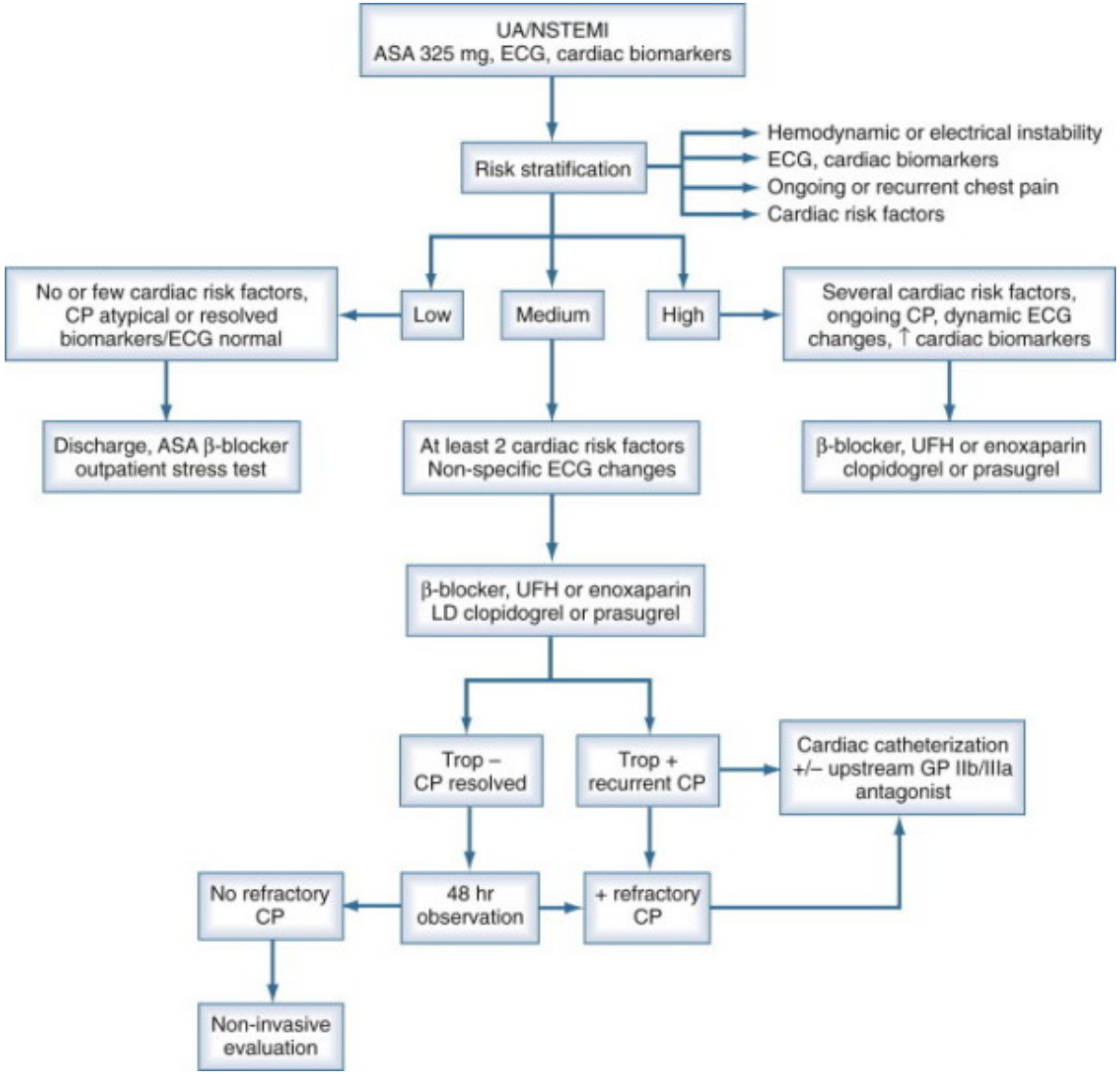
Figure 75-3 Electrocardiographic indications for reperfusion therapy in the left bundle branch block presentation.


STEMI
Aspirin 325mg chewed, UHF or enoxaparin clopidogrel or prasugrel
O2, NTG MSO4 IV for pain
Evidence of cardiogenic shock or conduction disturbance
No
β-blocker
Determine strategy for reperfusion in shortest time possible
Suspect mechanical complication (i.e. papillary muscle rupture, VSD)
No
Yes
Diagnostic cardiac cath and emergent operative repair as indicated
Cardiogenic shock, symptom onset > 3hrs contraindication to fibrinolytics, failed fibrinolytics, or door to balloon time < 90 mins
Yes
No
Fibrinolytic therapy
Cardiac cath and PCI as clinically indicated
CP free, ST segment ↓ by 50%, hemodynamically/electrically stable
No
Cardiac cath

Algorithm for evaluation and management of patients suspected of having ACS



To facilitate interpretation of this algorithm and a more detailed discussion in the text, each box is assigned a letter code that reflects its level in the algorithm and a number that is allocated from left to right across the diagram on a given level.
ACC/AHA: American College of Cardiology/American Heart Associations; ACS: acute coronary syndrome; ECG: electrocardiogram; LV: left ventricular.
Reproduced with permission from: Anderson J, Adams C, Antman E, et al. ACC/AHA 2007 guidelines for the management of patients with unstable angina/non-ST-elevation myocardial infarction: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to revise the 2002 Guidelines for the Management of Patients with Unstable Angina/Non-ST-Elevation Myocardial Infarction): developed in collaboration with the American College of Emergency Physicians, American College or Physicians, Society for Academic Emergency Medicine, Society for Cardiovascular Angiography and Interventions, and Society of Thoracic Surgeons. J Am Coll Cardiol 2007; 50:e1.


UA/NSTEMI
ASA 325 mg, ECG, cardiac biomarkers
Risk stratification
Hemodynamic or electrical instability
ECG, cardiac biomarkers
Ongoing or recurrent chest pain
Cardiac risk factors
Low
Medium
High
No or few cardiac risk factors, CP atypical or resolved biomarkers/ECG normal
Several cardiac risk factors, ongoing CP, dynamic ECG changes, ↑ cardiac biomarkers
Discharge, ASA β-blocker outpatient stress test
At least 2 cardiac risk factors Non-specific ECG changes
β-blocker, UFH or enoxaparin clopidogrel or prasugrel
β-blocker, UFH or enoxaparin LD clopidogrel or prasugrel
Trop – CP resolved
Trop + recurrent CP
Cardiac catheterization +/– upstream GP IIb/IIIa antagonist
No refractory CP
48 hr observation
+ refractory CP
Non-invasive evaluation

ACC/AHA guidelines: Medications

- Oxygen Saturation at least 90%
- Intravenous nitroglycerin - for treatment of persistent pain, congestive heart failure, or hypertension
  - Contraindications (eg, use of phosphodiesterase inhibitor drugs for erectile dysfunction)
  - Goal of therapy is a 10% reduction in the systolic blood pressure or a 30 percent reduction in hypertensive patients
  - Caution with RV infarction or severe aortic stenosis
- Morphine for relief of CP or anxiety
- Beta blockers - recommended universally to all patients without contraindications, irrespective of concomitant fibrinolytic therapy or PCI.
  - No specific BB in guidelines.
  - Most experts recommend a IV cardioselective agent such as metoprolol or atenolol
  - Esmolol can be used if an ultrashort acting beta blocker is required
- Statin therapy for all patients
  - Some initiate on admission (lipid panel)
  - All recommend initiation prior to hospital discharge

ACC/AHA guidelines: Medications

- Electrolyte repletion - guidelines recommend maintaining the serum potassium > 4.0 meq/L and serum magnesium > 2.0 meq/L in patients with an acute MI
- Heparin - use of unfractionated heparin (UFH) in:
  - patients with STEMI undergoing percutaneous or surgical revascularization
  - patients undergoing thrombolysis with selective fibrinolytic agents
  - Low molecular weight heparin (LMWH) might be considered (class IIb recommendation) an alternative to UFH in patients receiving thrombolysis provided they are younger than 75 years of age and have no renal dysfunction

Treatment in NSTEMI

- **Reperfusion** — Thrombolytic therapy **should NOT** be administered to patients with UA or NSTEMI
  - EXCEPTION: subsequent ECG documentation of persistent ST segment elevations
  - PCI is best suited for patients with an elevated troponin level or a TIMI risk score ≥5
  - For patients at lower risk, approaches vary based upon hospital protocol
- **Medications** — similar to STEMI
  - Cornerstone is antiplatelet therapy

ECG Case Studies