CARDIAC FUNCTION TEST

Dr.Siby Joseph

CARDIAC FUNCTION TESTS

- To know whether heart is functioning normally or not.
- To find out abnormalities of heart functions.
- To find out the extend of abnormal heart functions.
- To assess the efficacy of intervention to correct the cardiac abnormalities

- ✓ Biochemical markers
- ✓ Electrocardiogram
- ✓ Imaging studies.

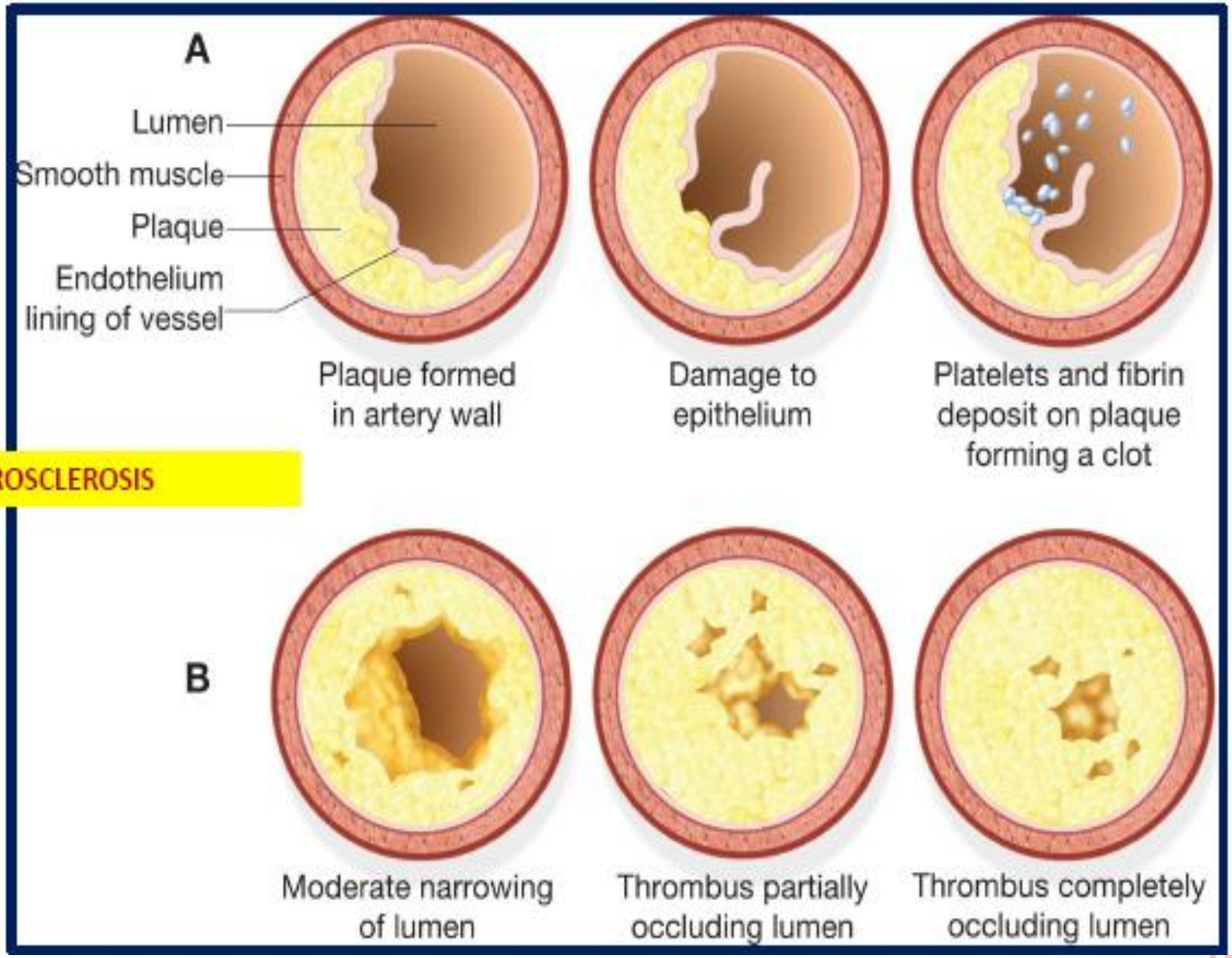
BIOCHEMICAL CARDIAC MARKERS

The criteria for an ideal BCM for the diagnosis of Acute coronary Syndrome include

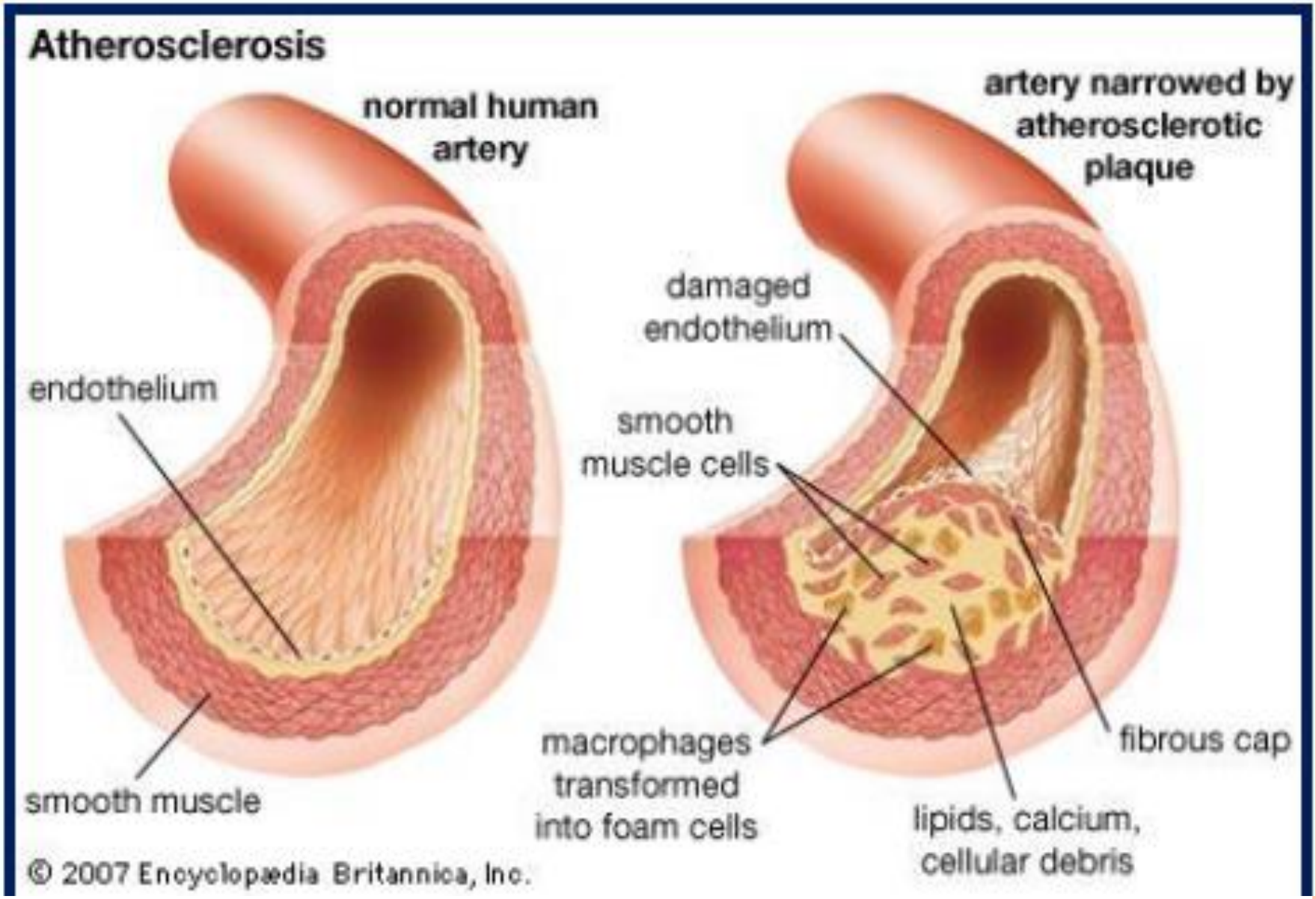
- ✓ High specificity.
- ✓ High sensitivity.
- ✓ Release and clearance kinetics provide expedient and practical diagnosis.
- ✓ Measured level of marker is in direct proportional relationship to the extent of myocardial injury.
- ✓ Assay technique is commercially available and is easy to perform, inexpensive and rapid

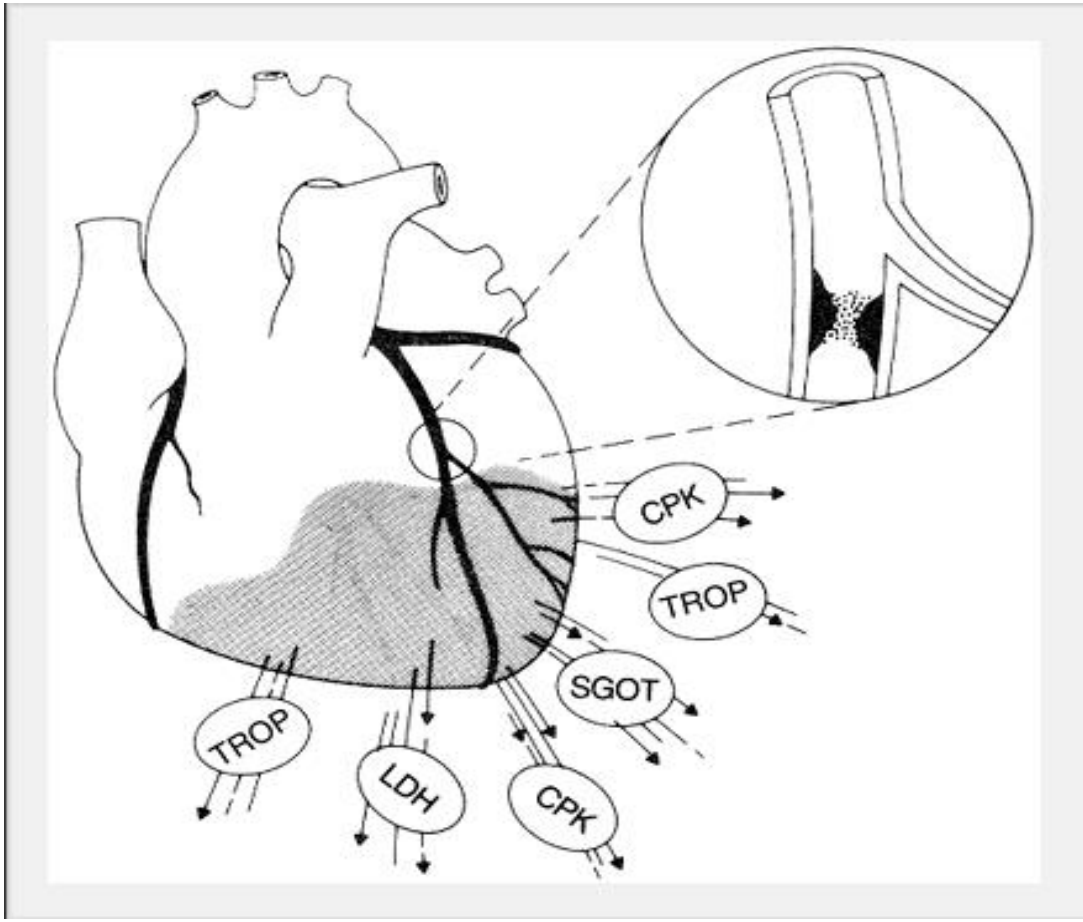
- The heart muscles uses chemical energy to contract and pump.
- Energy is obtained from aerobic metabolism of fatty acid and other nutrients (lesser extent). Eg: glucose & lactate.
- Due to this energy requirement, cardiac tissues are rich in enzymes, isoenzymes and proteins than other body tissues.
- When heart is injured, irreversible damage takes place to cardiac cells as during ACS and these enzymes and proteins leak through cell membrane entering the vascular compartment.

- **Cardiac biomarkers** are substances that are released into the blood when the heart is damaged.
- Measurement of these biomarkers is used to help diagnose, evaluate, and monitor patients with suspected ACS.
- The symptoms of ACS may also be seen with non-heart-related conditions.
- So, increases in one or more cardiac biomarkers can identify patients with ACS, allowing rapid diagnosis and appropriate treatment of their condition.



ATHEROSCLEROSIS


Atherosclerosis
normal human artery
endothelium
smooth muscle
artery narrowed by atherosclerotic plaque
damaged endothelium
smooth muscle cells
macrophages transformed into foam cells
lipids, calcium, cellular debris
fibrous cap
© 2007 Encyclopædia Britannica, Inc.



Intracellular enzymes are released by the dying myocardial cells after complete coronary occlusion resulting in acute infarction

The best choice of BCM in the evaluation of pts with ACS is **TROPONINS.**

- Troponins is a protein complex consisting of 3 subunits: TnC, TnI, TnT
- Three subunits are located along the thin filaments of myofibrils.
- They regulate Ca2+ mediated interaction of actin and myosin necessary for the contraction of cardiac muscles
- Their level rise within 4-8 hrs after injury & remain elevated for 7-14 days
- Troponin T is found in cardiac and skeletal muscle cells whereas Troponin I is found only in cardiac muscles.
- (TnI < 1.5 ng/ml, TnT <0.2 ng/ml)

CARDIAC TROPONINS

- **I. Troponin :** "contractile "proteins of myofibrils→ regulatory proteins

a) **Troponin C** (the calcium binding component)

b) **Troponin I** (the inhibitory component)

c) **Troponin T** (Tropomyosin binding component)

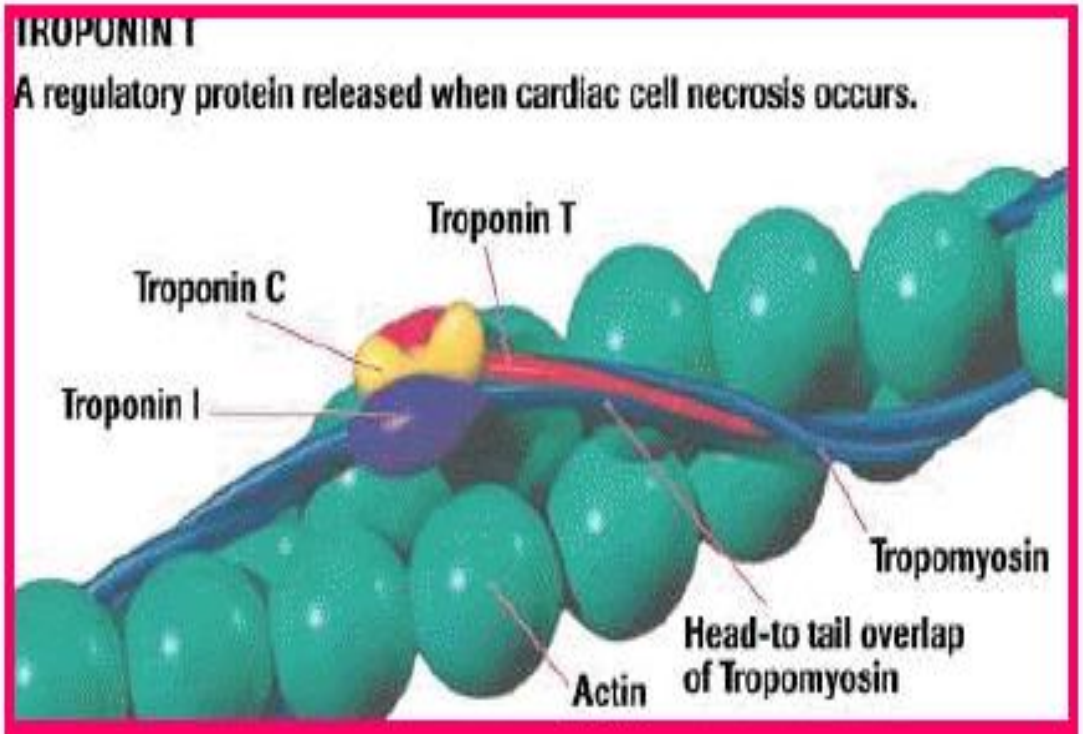
- **II. Component of cardiac muscles**
- **III. Longer Half Time** –Insoluble Troponin released from infarcted heart muscle therefore circulatory levels remain high .
- **IV .Rise in serum Troponin continues for longer than enzymes**

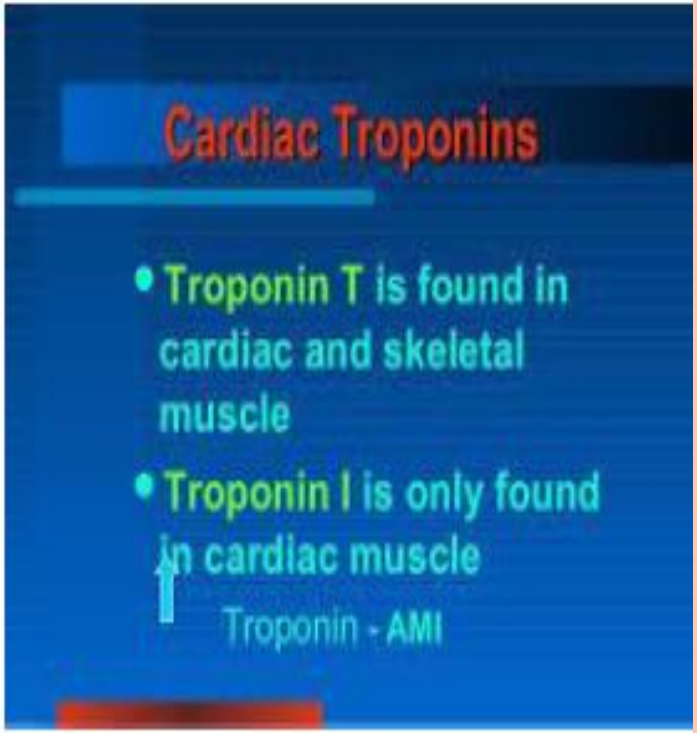
Comparison between cardiac Troponin I & Troponin T

Human cardiac Troponin I	Human cardiac Troponin T
30 AMINO ACIDS	11 AMINO ACIDS
CARDIAC MARKERS ---SPECIFIC &SENSITIVE	MUSCLUAR DYSTROPHY CHRONIC RENAL FAILURE POLYMYOSITIS
2 ISOFORMS—GENETIC ORIGIN	
INCREASE OBSERVED AFTER AMI—CPK –MB& CPK -TOTAL	

TROPONINS (MARKER OF MYOCARDIAL INFARCTION)

TROPONINS TYPE	PROPERTY
TROPONINS C	CALCIUM BINDING
TROPONINS I	ACTINO MYCIN INHIBITORY ATPase
TROPONINS T	TROPOMYOSIN BINDING ELEMENT





Cardiac Troponin I &T

❖ **Clinical interpretation :cardiac troponin (C Tn I & C Tn T)**

1. Longer half life → insoluble troponin released from infarcted heart muscles within 4 to 8 hrs after onset of symptoms
2. Troponin I &C increase significantly after AMI
3. Increase in serum levels of Troponin I &C synchronizes CPK –MB & CPK – TOTAL
4. circulatory levels are maintained for 4-5 days
5. Half life of C Tn I & C Tn T is 5- 10days
6. peak values of C Tn T observed at → 72 -100 hrs.
7. peak values of C Tn I observed at → 24 -48 hrs.
8. low levels & undetectable serum levels are observed in individuals without cardiac disease(no false + ve or –ve values for C Tn I)

CREATININE KINASE

- Normal range:
- males:0-200 IU/L or 667-3334 nmol sec/L
- females:35-150 IU/L or 583-2501 nmol sec/L
- CK is an enzyme that stimulates the transfer of high energy phosphate groups.
- CK is found in skeletal muscle, myocardium, and the brain.
- Concentrated in cytoplasm of tissues that require high amount of energy specially in striated (skeletal) muscles

- Amount of circulating CK is related to individual's muscle mass. Therefore males have higher value than females.
- In AMI, Sr. CK rise sharply 6-8 hrs after onset of chest pain d. Max conc. (5-7 times normal) is reached within 24 hrs and returns to normal in 3-4 days.
- Vigorous exercise, fall, deep IM injection, trauma, surgery can also cause elevation in CK levels.

- Some drugs like amphotericin B, clofibrate, ethanol, lithium can also increase total CK. Therefore isoenzymes of CK should be considered.
- There are 3 CK isoenzymes and their tissue source are skeletal (CKMM), heart (CKMB), brain (CKBB).
- **Sr. CKMB (<12 IU/L)** begin to rise 4-8 hrs after AMI symptoms and peak concentration reached within 12-24 hrs.
- Because of faster clearance from Serum, CKMB returns to baseline sooner than CK (2-3 days).

MYOGLOBIN:

- Its an oxygen storing protein found in heart & other muscles.
- Serum levels are detected within 1-4 hrs and peak 6-7 hrs after the onset of symptoms.
- The early rise of myoglobin levels after onset of symptoms make it the most appropriate and convenient test for early detection of AMI.
- Time period :in which CPK -2 & CARDIAC TROPONIN IS VERY SHORT.

- **Disadvantages of Myoglobin as cardiac marker :**

- ✓ Non specific eg myoglobin increases in any form of muscle damage.
- ✓ Renal failure can give false positive results.
- ✓ Methods not tissue specific (muscle /cardiac)
- ✓ Muscle injury increase in myoglobin misdiagnosis of AMI
- ✓ Its sensitivity is lost due to its rapid clearance.

- **LACTATE DEHYDROGENASE**:

Normal range:100-210 IU/L or 1667-3501 nmol sec/L

- LDH is an enzyme that catalyses the reversible formation of lactate from pyruvate in the final step of glycolysis.

- Following AMI, serum LDH rises in 24-48 hours, peaks at 2-3 days, and returns to normal in 8-14 days after onset of chest pain.

- The major limitation to LDH is the lack of specificity as it is found in numerous organs and tissues including the heart, liver lungs kidney ,skeletal muscle, red blood cells and lympocytes.
- Electrophoretic fractionation of LDH to its five major isoenzymes (LDH1-LDH5) better distinguishes the site of origin, but these test are not specific for AMI.
- The heart contains mainly LDH1 and to a lesser extent LDH2.In past, elevation of LDH1 or a ratio of LDH1:LDH2 greater than one was used in the differential diagnosis of AMI

- *Due to development of more cardiac-specific markers, LDH and LDH isoenzymes are no longer recommended for the evaluation of the patient with ACS.*

ASPARTATE AMINOTRANSFERASE:

- Normal range :0-45IU/L
- AST is widely distributed in the liver, heart, skeletal muscle, red blood cells, kidney and pancreas.
- Serum levels of AST rises within 12 hours of AMI, peak in 24-48 hrs, return to baseline in 3-4 days
- Poor specificity of AST for myocardial cell damage led to its replacement by more cardiac-specific markers.

HOMOCYSTEINE

- Increase in serum homocystein

- Molecular basis of coronary arterial disease (CAD) respect to Homocysteinemia

1.Damage cell lining of blood vessels

2. Increase growth of smooth muscles

3. Homocystein alters anti coagulant properties of endothelial cells to pro coagulant

4. Dysfunction of vascular endothelium

5. Damage vascular endothelium

- Atherosclerosis & CAD

BNP (B type natriuretic peptide)

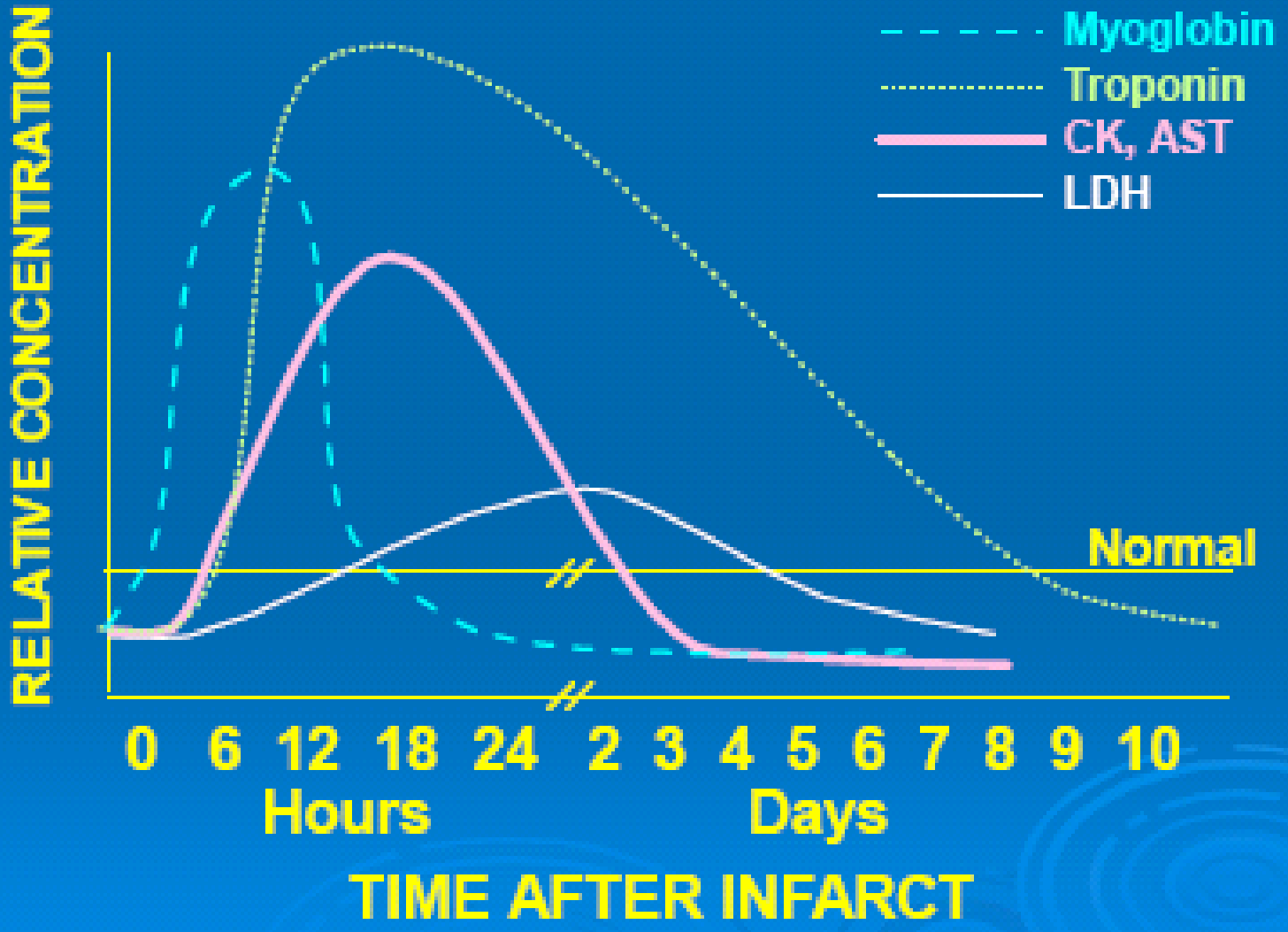
- It is a cardiac specific peptides first identified in porcine brain extracts and hence the name brain natriuretic peptide.
- BNP is a neurohormone released by ventricular myocardium in response to volume overload.
- Its primary utilization is for the evaluation of patient with HF.
- It has shown to be a strong predictor of short and long-term mortality in patients with ACS.
- The clinical diagnostic cutoff level for heart failure is BNP LEVEL of >100 pg/mL

- N- Terminal-ProBNP(NT-proBNP) is a more stable form of BNP
- It is formed by the enzymatic cleavage of PreproBNP,a precursor of BNP.

- NT- proBNP levels are elevated in the elderly, and accordingly, the clinical diagnostic cutoff level for heart failure is >125 pg /mL in pts younger than 75 yrs of age and >450 pg/mL in pts older than 75 yrs.

CRP, C-REACTIVE PROTEIN:

- Is a non specific acute-phase reactant that is released in the presence of inflammatory processes caused by various etiologies (infections, malignancy, trauma, rheumatoid arthritis and other inflammatory process).
- Synthesized in the liver and normally present in trace amounts in peripheral circulation.
- CRP production is stimulated by systemic cytokines.
- Serum & plasma levels of CRP are elevated in pts with CAD and ACS
- Cut off time is (<0.3 mg/dl)


RELATIVE CONCENTRATION
Myoglobin
Troponin
CK, AST
LDH
Normal
0 6 12 18 24 2 3 4 5 6 7 8 9 10
Hours
Days
TIME AFTER INFARCT

Comparison of cardiac markers

1. Increase in CPK -2 in conditions individuals without cardiac disease Therefore better risk assessment than CPK –MM .
2. CPK -2 (CPK –MB) increase in muscle injury
3. (C Tn I & C Tn T) don't increase in muscle injury
4. C Tn T excellent marker for AMI VERSES muscle injury when concomitant.
5. C Tn T increases in sepsis, drug induced toxicities ,chronic diseases ,malignancies ,hematological disorder ,non cardiac surgery
6. **C Tn I →sensitive & specific for AMI**

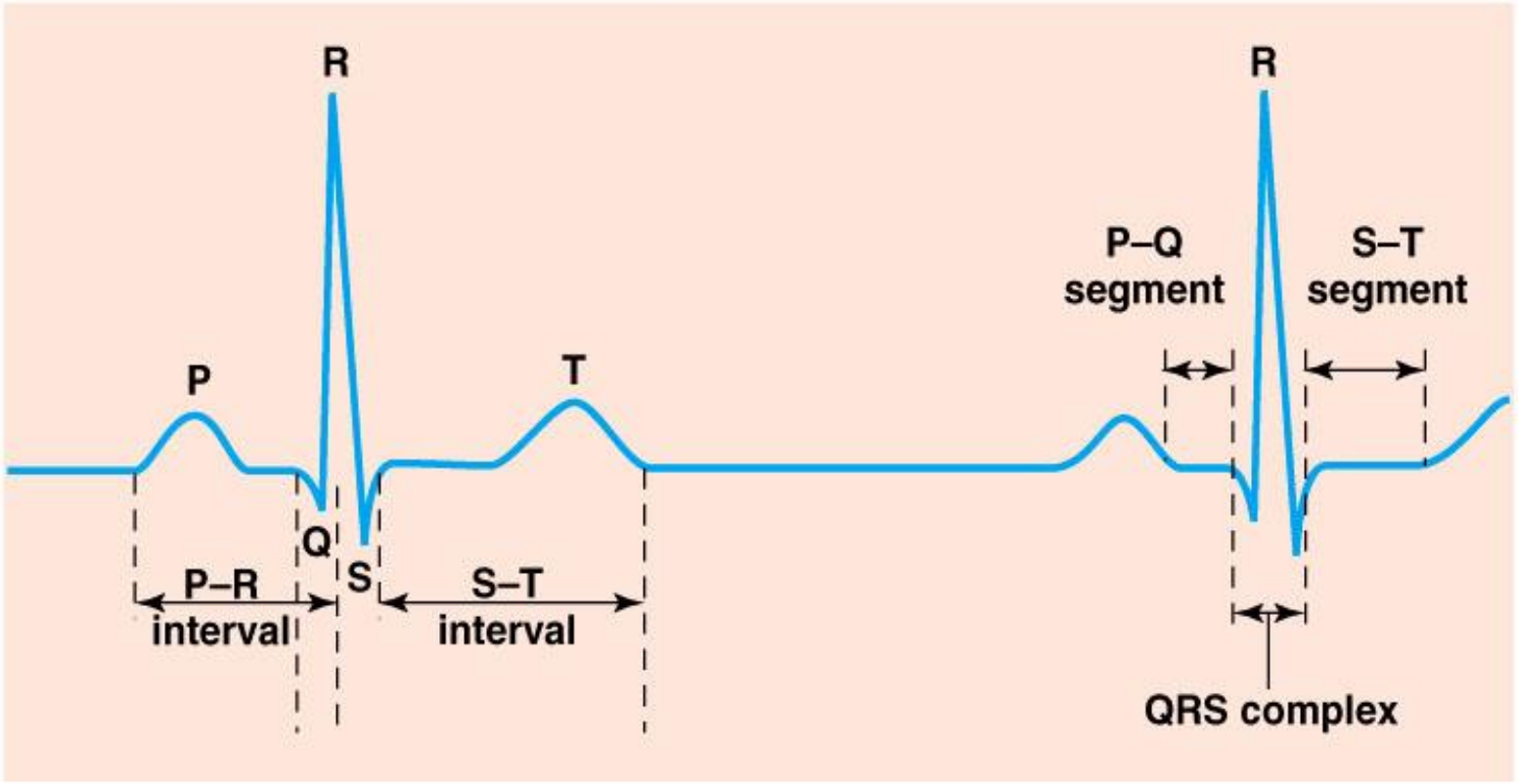
Biochemical Cardiac Markers

	CARDIAC MARKER	ABNORMAL ACTIVITY DETECTABLE IN (hrs)	PEAK VALUE OF ABNORMALITY (hrs)	DURATION OF ABNORMALITY (DAYS)
1	CPK (TOTAL)	3-8 HRS	10-24	3-4
2	CPK -MB	3-8 HRS	10-24	2-3
3	LDH (TOTAL)	8-12HRS	72-144	8-14
4	SGOT (AST)	6TO 12HRS (	24-48	4-6
5	MYOGLOBIN	1TO 3HRS	6-9	1
6	TROPONIN I (C –T n I)	3-8 HRS	24-48	3-5
7	TROPONIN T (C –Tn T)	3-8 HRS	72-100	5-10

ELECTROCADIOGRAPHY

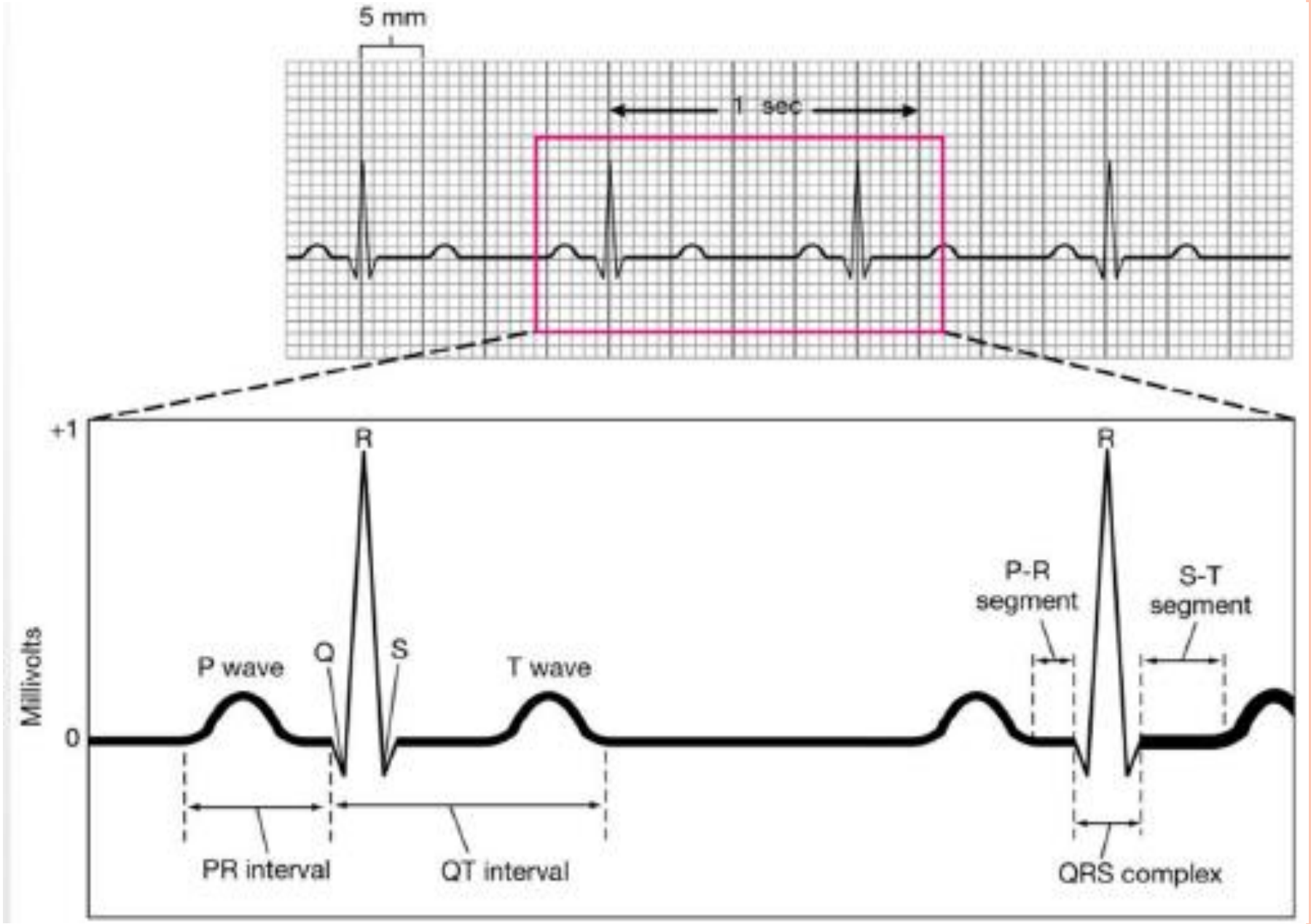
- Recording of the electrical activity of the heart on an electrocardiogram (ECG)

(b)



- Significance of ECG.
- ECG gives information about rate and rhythm of the heart.
- The physical orientation of heart i.e axis.
- Its a diagnostic tool for various heart conditions like hypertrophies , ischemia, infarction , arrhythmias
- Conduction problems and pace maker activity.

- ECG does not provide information about mechanical activity.


5 mm
1 sec
+1
0
Millivolts
R
R
P wave
Q
S
T wave
P-R
segment
S-T
segment
PR interval
QT interval
QRS complex

ECG leads.

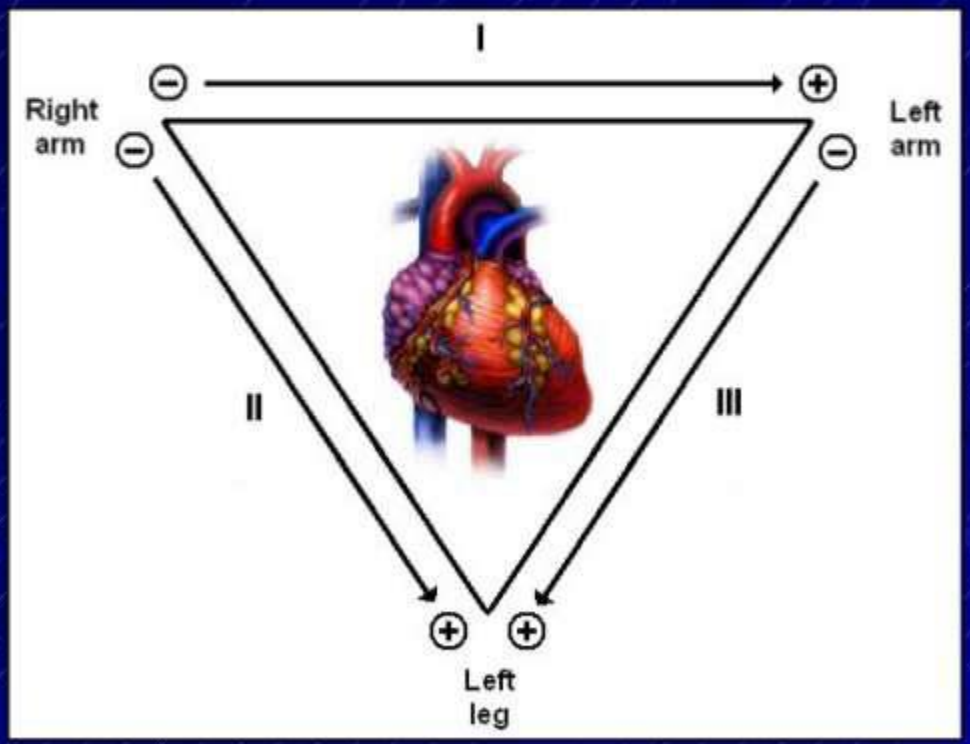
- leads are electrodes which record the electrical potential of heart at different sites.
- There are 12 ECG leads.
  a) 3 bipolar limb leads.
  b) 3 augmented limb leads.(unipolar).
  c) 6 chest leads.

- Different leads provide specific information on different aspects of heart chambers and coronary arteries.

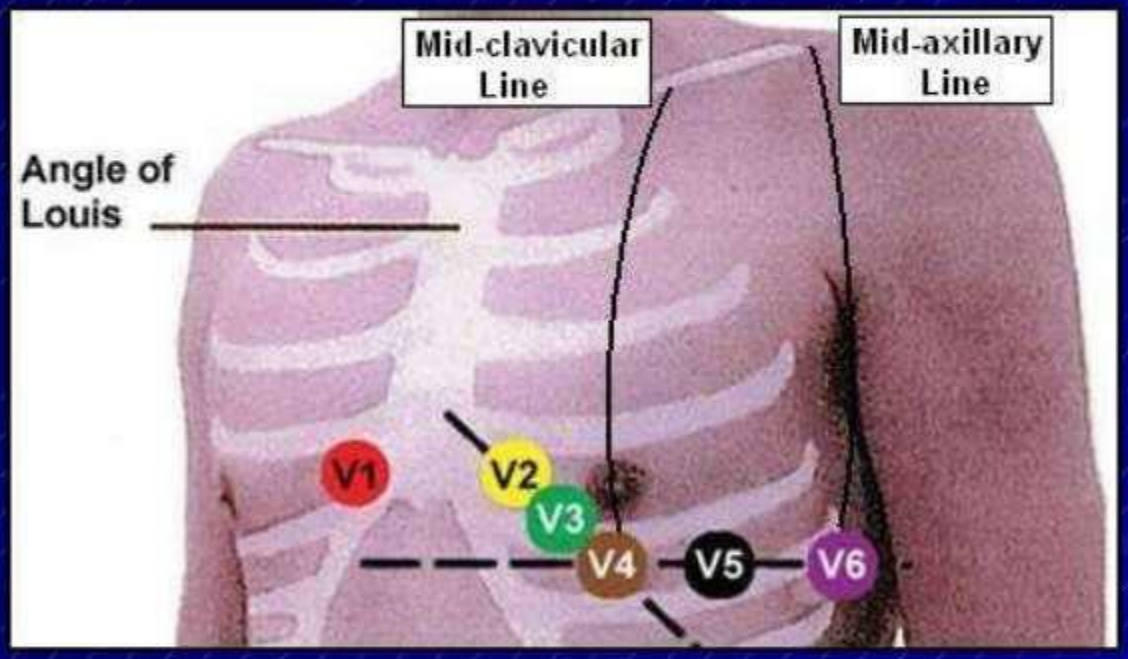
Bipolar limb leads.

- Lead 1 = left arm + ve , right arm _ ve
- Lead 2 = right arm _ ve ,left leg + ve.
- Lead 3 = left arm _ ve , left leg +ve.

Standard Limb Leads



Precordial Leads



Augmented limb leads.

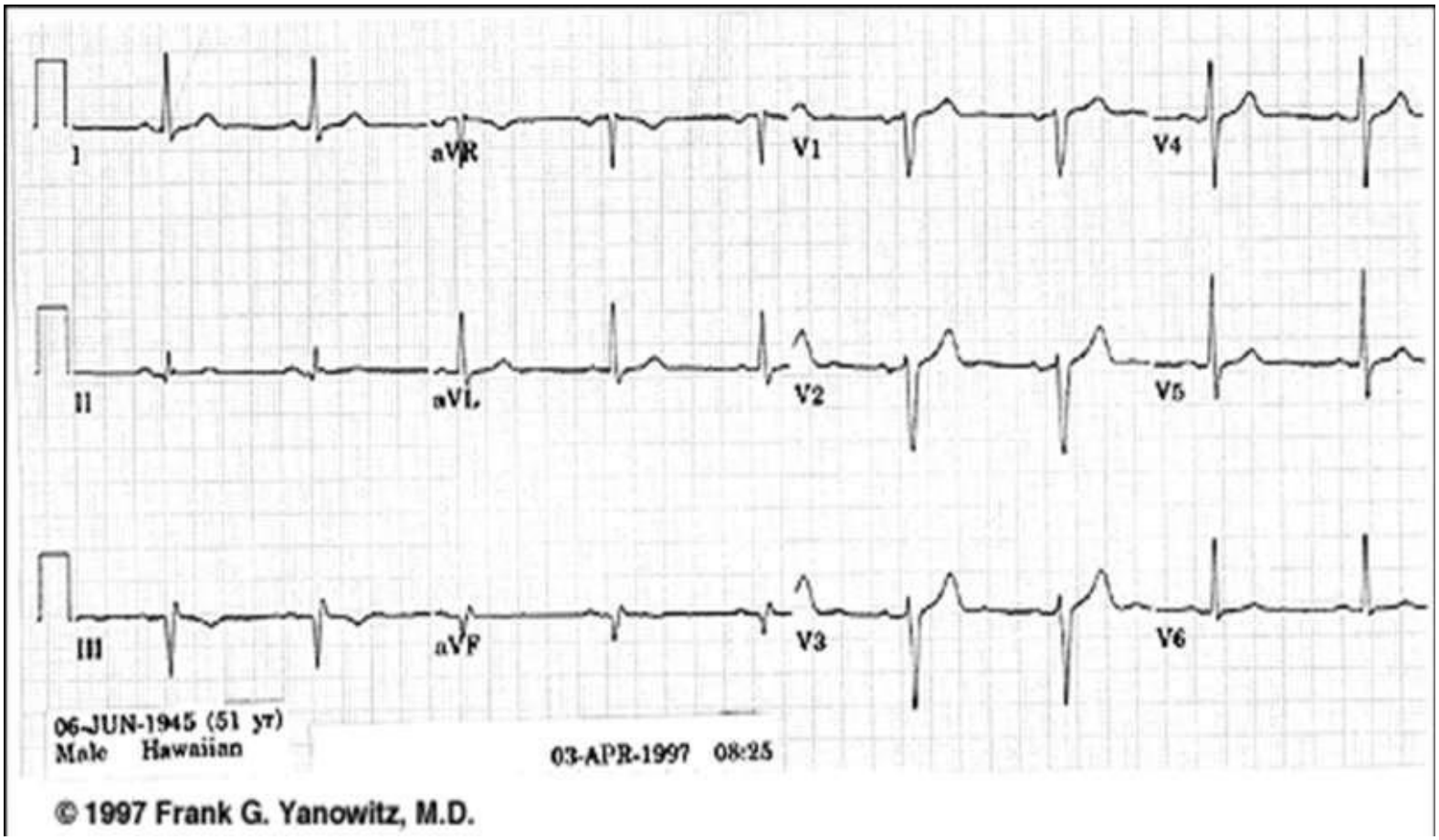
- aVR attach to right arm.
- aVL attach to left arm.
- aVF attach to left foot.

Summary of Leads

	Limb Leads	Precordial Leads
Bipolar	I, II, III (standard limb leads)	-
Unipolar	aVR, aVL, aVF (augmented limb leads)	V_1-V_6

Remember

- Positive electrode is always a reference electrode.
- Depolarization wave moves toward the positive electrode gives positive deflection.
- Depolarization wave move away from positive electrode gives the negative deflection.


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
06-JUN-1945 (51 yr)
Male Hawaiian
03-APR-1997 08:25
© 1997 Frank G. Yanowitz, M.D.

Basic terminologies

- Base line : flat, straight and isoelectric line
- Wave form :deviation or movement away from base line may be upward or downward
- Segment : A line between two waves
- Interval : a wave form plus a segment this shows time duration
- Complex : combination of several wave form without segment.

Normal Impulse Conduction

Sinoatrial node

↓

AV node

↓

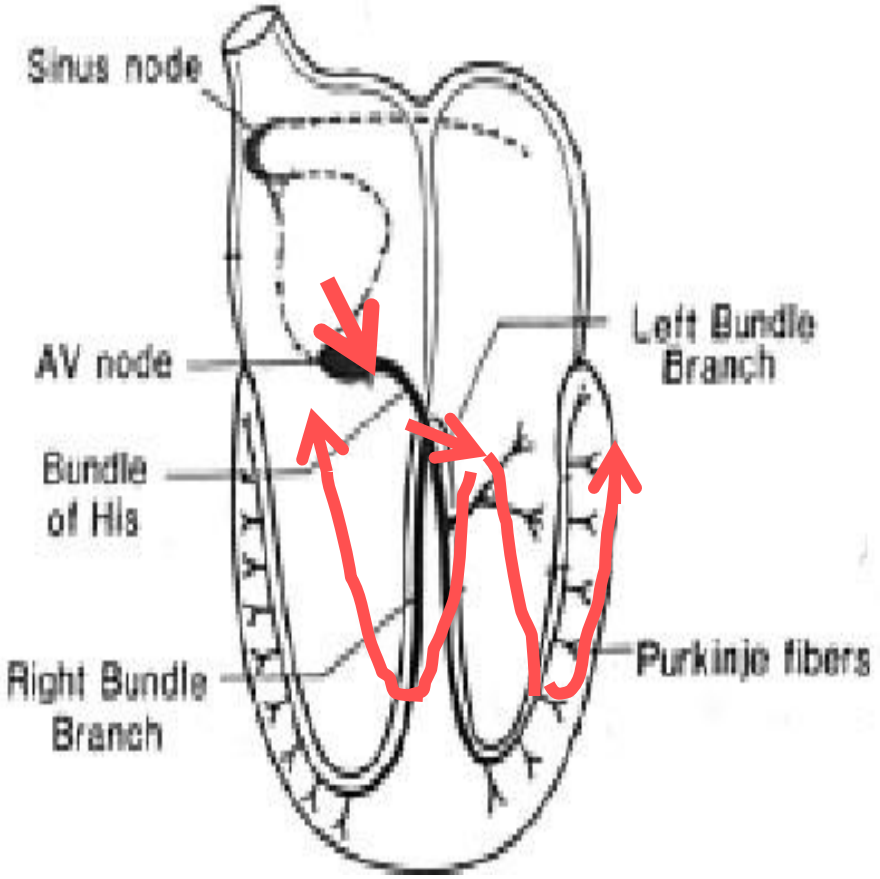
Bundle of His

↓

Bundle Branches

↓

Purkinje fibers



Impulse Conduction & the ECG

Sinoatrial node

↓

AV node

↓

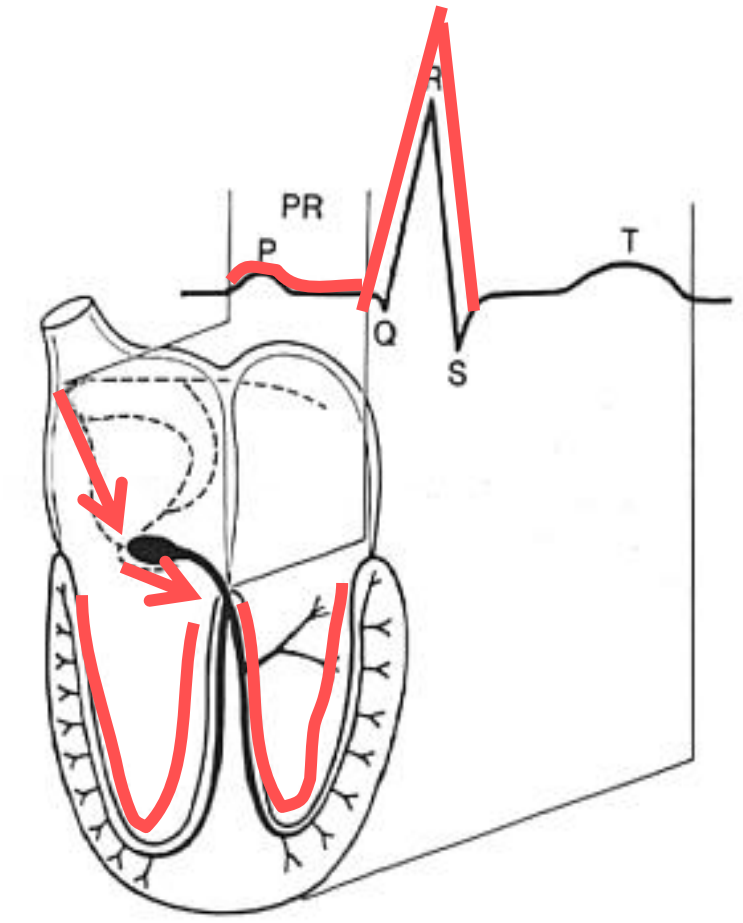
Bundle of His

↓

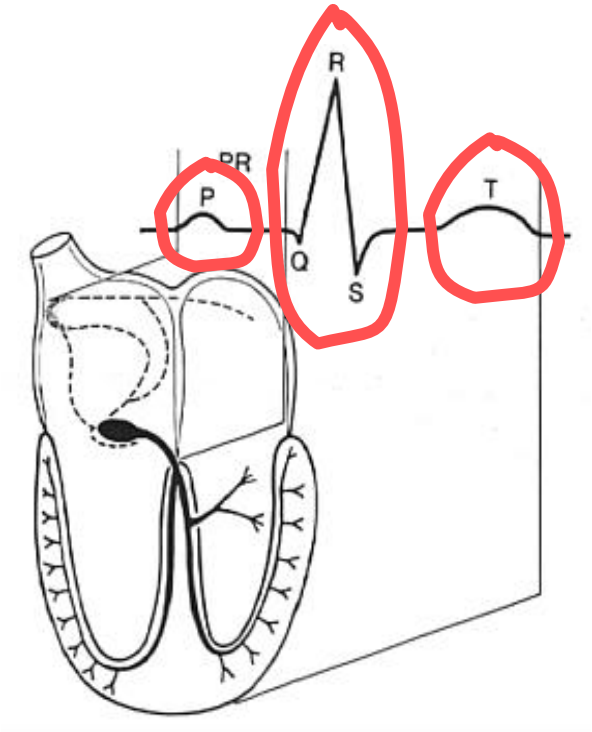
Bundle Branches

↓

Purkinje fibers



The "PQRST"



- P wave - Atrial depolarization
- QRS - Ventricular depolarization
- T wave - Ventricular repolarization

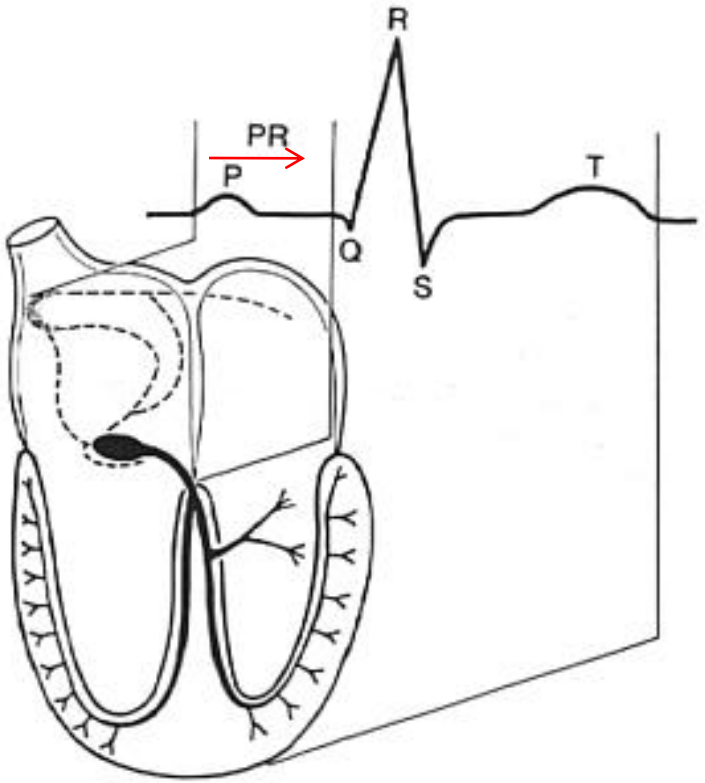
PR INTERVAL

Atrial depolarization

+

delay in AV junction

(AV node/Bundle of His)



(delay allows time for the atria to contract before the ventricles contract)

- The **PR interval** is the time from the onset of the P wave to the start of the QRS complex.
- It reflects conduction through the AV node.

Elements of the ECG:

- P wave: Depolarization of both atria;
  - ✓ Relationship between P and QRS helps distinguish various cardiac arrhythmias
  - ✓ Shape and duration of P may indicate atrial enlargement
  - ✓ Normal duration is 0.12- 0.16 secs
- PR interval: from onset of P wave to onset of QRS
  - ✓ Normal duration = 0.12-2.0 sec
  - ✓ Represents atria to ventricular conduction time (through His bundle)
  - ✓ Prolonged PR interval may indicate heart block

- QRS complex: Ventricular depolarization
  - ✓ Normal duration = 0.08-0.12 seconds
  - ✓ Its duration, amplitude, and morphology are useful in diagnosing cardiac arrhythmias, ventricular hypertrophy, MI, electrolyte derangement, etc.
  - ✓ Q wave greater than 1/3 the height of the R wave, greater than 0.04 sec are abnormal and may represent MI

- ST segment:
  - ✓ Connects the QRS complex and T wave
  - ✓ Duration of 0.08-0.12 sec (80-120 msec

- T wave:
  - ✓ Represents repolarization or recovery of ventricles
  - ✓ Interval from beginning of QRS to apex of T is referred to as the absolute refractory period

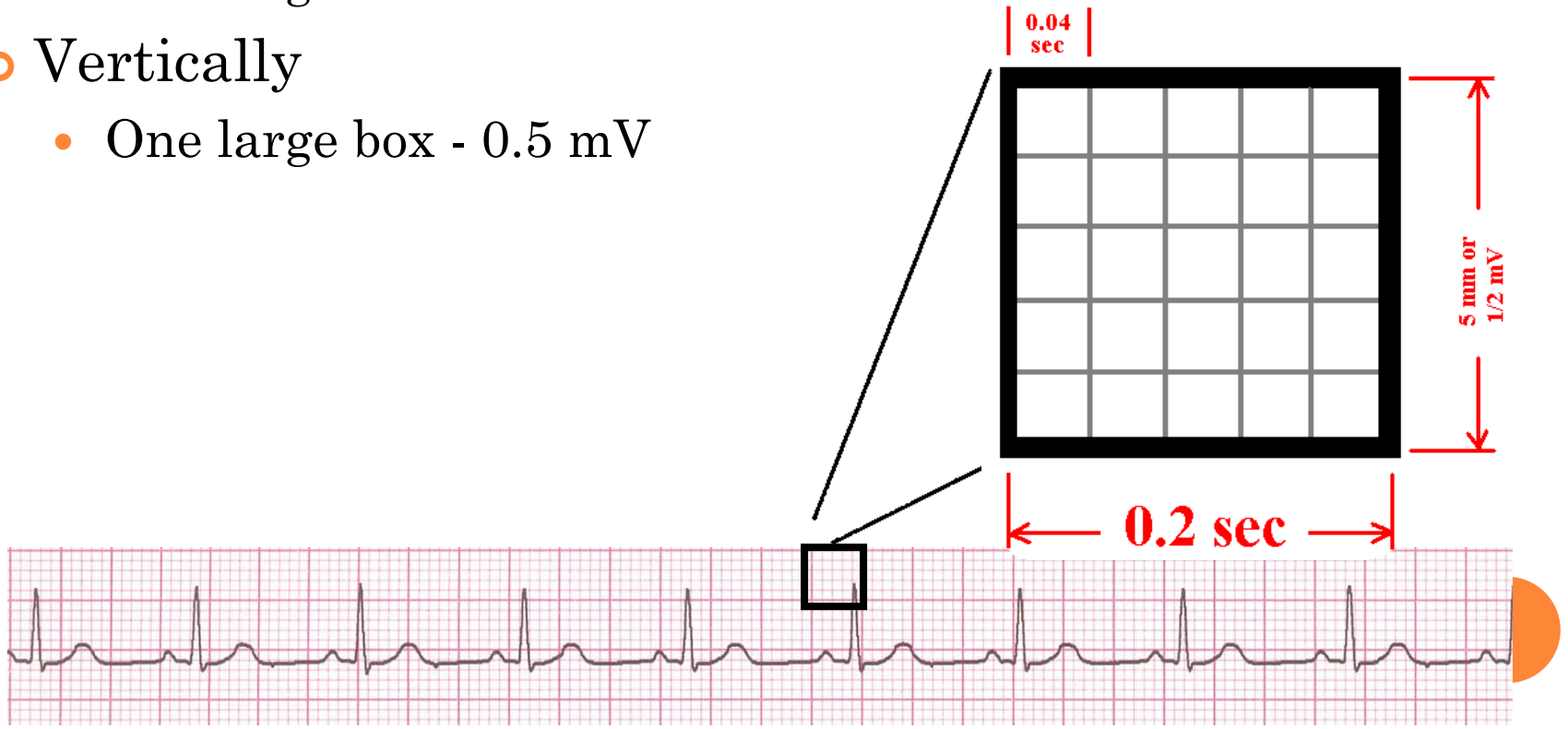
- QT Interval
  - ✓ Measured from beginning of QRS to the end of the T wave
  - ✓ Normal QT is usually about 0.40 sec
  - ✓ QT interval varies based on heart rate

Pacemakers of the Heart

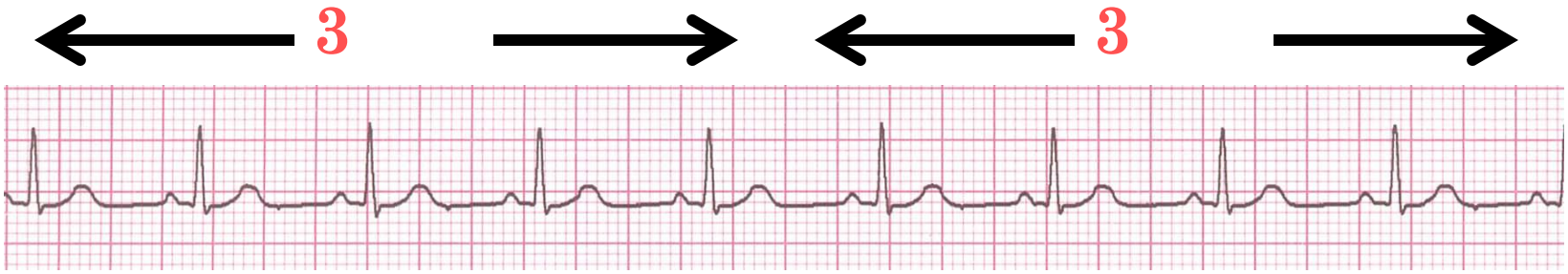
- SA Node - Dominant pacemaker with an intrinsic rate of 60 - 100 beats/minute.
- AV Node - Back-up pacemaker with an intrinsic rate of 40 - 60 beats/minute.
- Ventricular cells - Back-up pacemaker with an intrinsic rate of 20 - 45 bpm.

The ECG Paper

- Horizontally
  - One small box - 0.04 s
  - One large box - 0.20 s
- Vertically
  - One large box - 0.5 mV

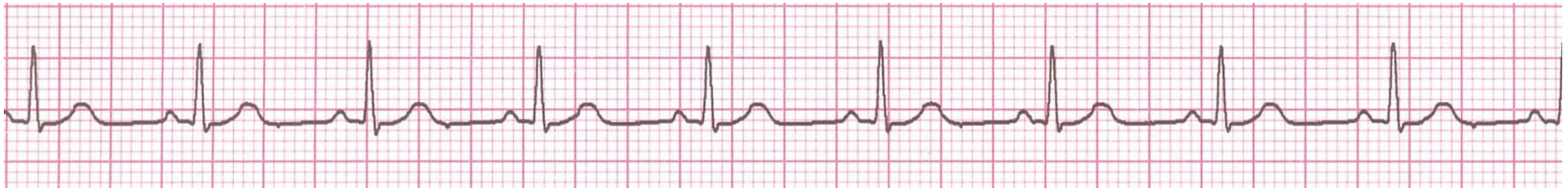


The ECG Paper (cont)



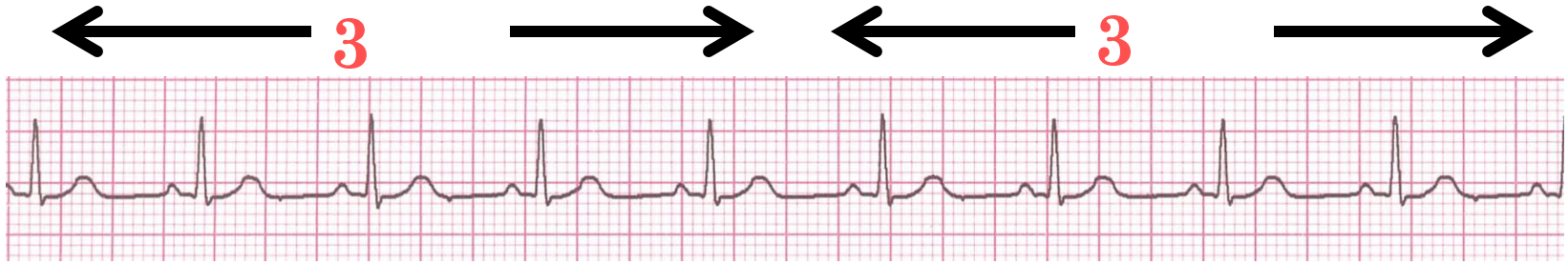
- Every 3 seconds (15 large boxes) is marked by a vertical line.
- This helps when calculating the heart rate.

Rhythm Analysis



- Step 1: Calculate rate.
- Step 2: Determine regularity.
- Step 3: Assess the P waves.
- Step 4: Determine PR interval.
- Step 5: Determine QRS duration.

Step 1: Calculate Rate



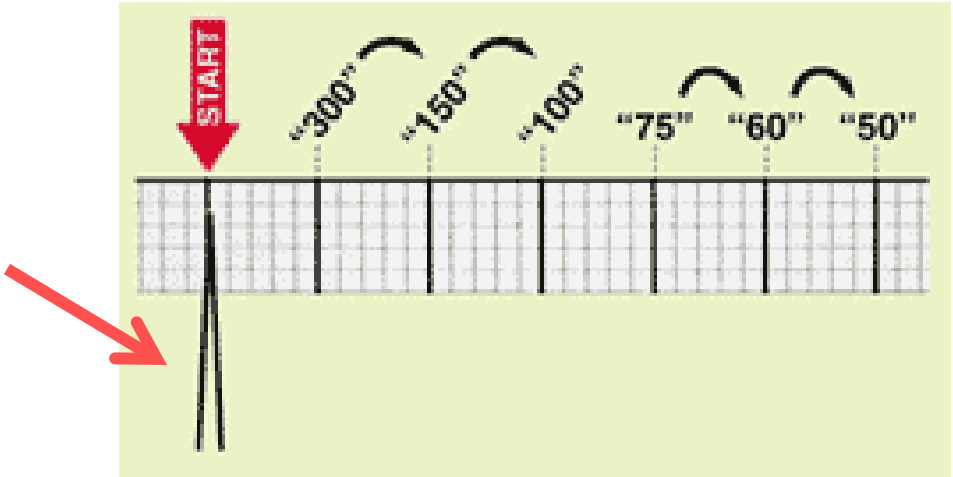
- Option 1
  - Count the # of R waves in a 6 second rhythm strip, then multiply by 10.

Interpretation?

9 x 10 = 90 bpm

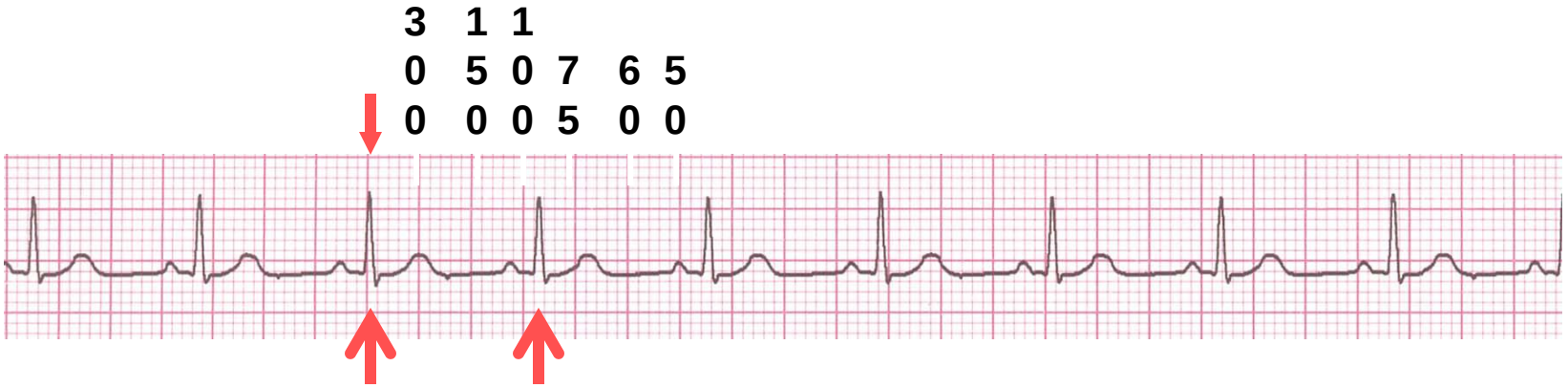
Step 1: Calculate Rate

R
wave



- Option 2
  - Find a R wave that lands on a bold line.
  - Count the # of large boxes to the next R wave. If the second R wave is 1 large box away the rate is 300, 2 boxes - 150, 3 boxes - 100, 4 boxes - 75, etc. (cont)

Step 1: Calculate Rate



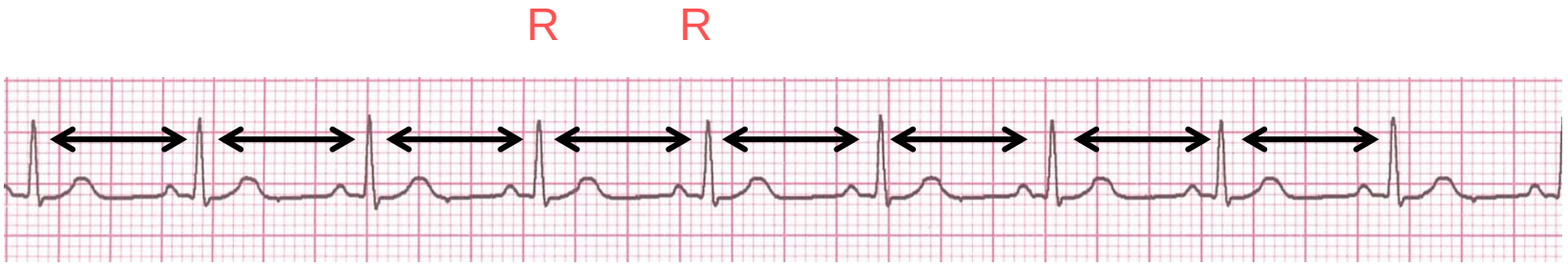
- Option 2 (cont)
  - Memorize the sequence:

    300 - 150 - 100 - 75 - 60 - 50

Interpretation?

Approx. 1 box less than 100 = 95 bpm

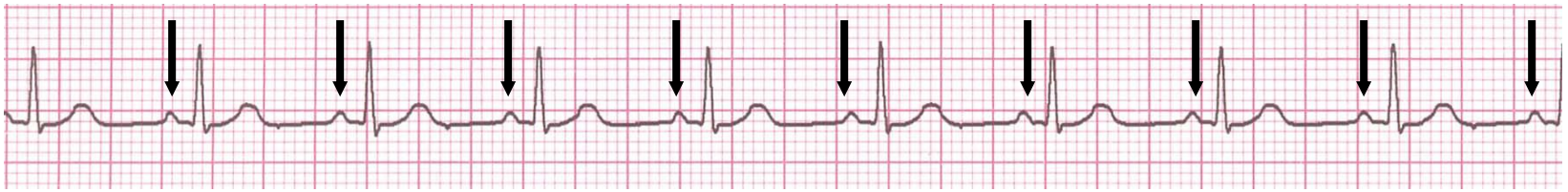
Step 2: Determine regularity



- ✓ Look at the R-R distances (using a caliper or markings on a pen or paper).
- ✓ Regular (are they equidistant apart)? Occasionally irregular? Regularly irregular? Irregularly irregular?

Interpretation? *Regular*

Step 3: Assess the P waves

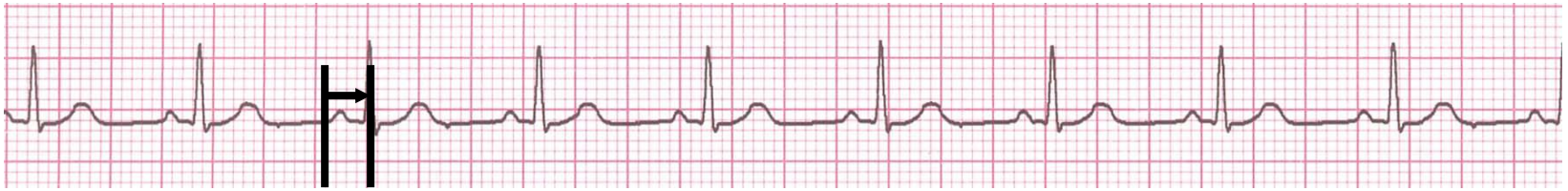


- ✓ Are there P waves?
- ✓ Do the P waves all look alike?
- ✓ Do the P waves occur at a regular rate?
- ✓ Is there one P wave before each QRS?

Interpretation?

Normal P waves with 1 P wave for every QRS

Step 4: Determine PR interval

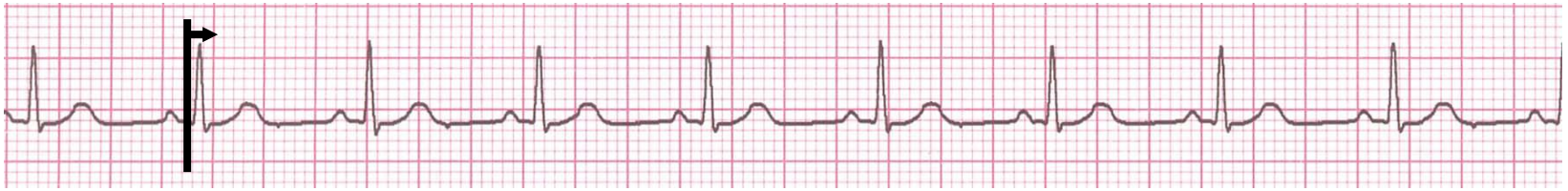


- Normal: 0.12 - 0.20 seconds.
  (3 - 5 boxes)

Interpretation?

0.12 seconds

Step 5: QRS duration

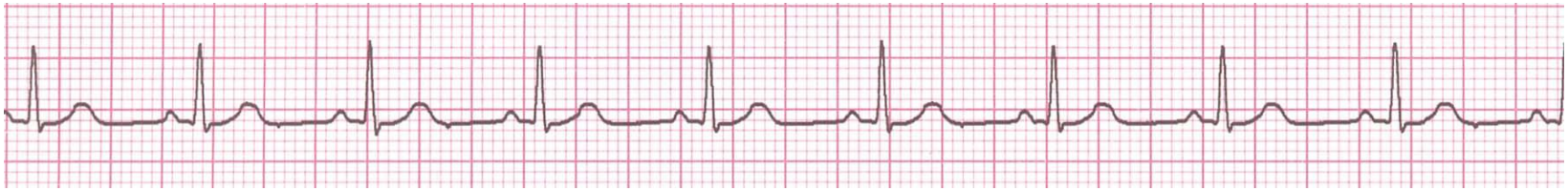


- Normal: 0.04 - 0.12 seconds.
  (1 - 3 boxes)

Interpretation?

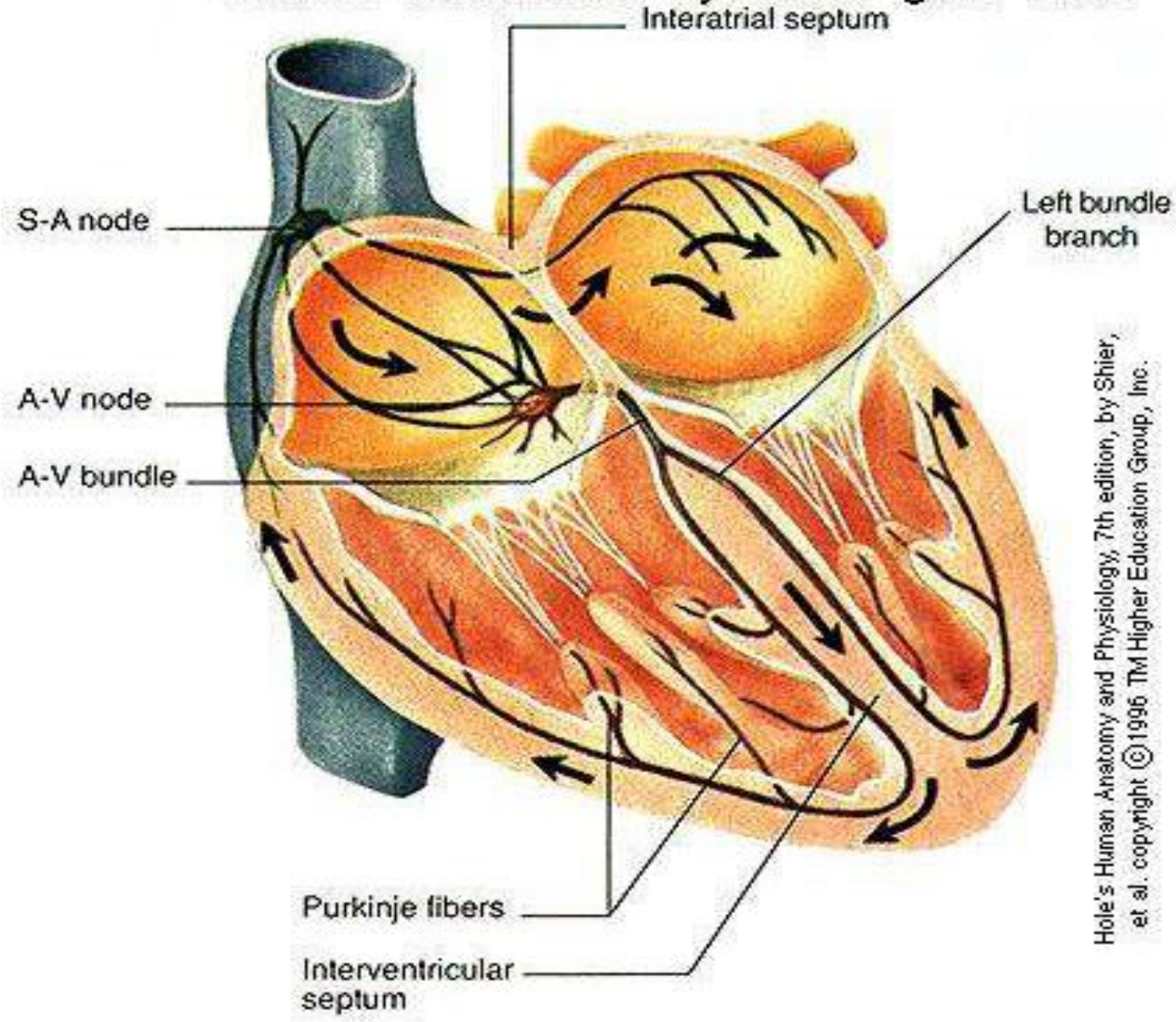
0.08 seconds

Rhythm Summary



✓ Rate	90-95 bpm
✓ Regularity	regular
✓ P waves	normal
✓ PR interval	0.12 s
✓ QRS duration	0.08 s
Interpretation?	*Normal Sinus Rhythm*

Cardiac Conduction System. Figure 15.19



Absent P.Wave

- P-waves are not apparent in

1. Atrial fibrillation-P waves replaced with numerous small irregularly occurring fibrillatory waves.
2. Atrial flutter-P waves are replaced with flutter waves with saw toothed appearance.
3. Junctional rhythm-P waves may just precede, just follow or are buried in QRS complexes anterogradely and the atria retrogradely.
4. Vetricular tachycardia-P waves are difficult to identify as they lie buried in the wide QRS complexes.
5. Hyperkalemia-P waves are reduced in amplitude or altogether absent. This is associated with tall T waves and wide QRS complexes.

Inverted P waves

- If the activation of atria occurs retrogradely from below upwards. usually seen in

1. Junctional rhythm.-Inverted P waves may just precede or just follow the QRS complexes.

2. Bypass tract-If atria are activated retrogradely through an acessary pathway bypassing AV node-Wolf Parkinson White syndrome.

- Tall P Wave
- Normal P wave is less than 2.5 mm in height.
- It is the sum of right and left atrial activation, right preceding the left.
- Tall P wave represents right atrial enlargement
- Tall P wave is known as P-Pulmonale-as it is often caused pulmonary hypertension or P.congenitale-as it is often found in congenital heart disease.

- Broad P.Wave
- Normal P wave is less than 2.5mm in width.
- It is the sum of right and left atrial activation, right preceding the left.
- A broad and notched P wave is representative of left atrial enlargement.
- It is known as P Mitrale-as it is often associated with mitral valve disease.

Prolonged PR interval.

- Prolong PR interval shows delayed conduction from SA to AV node….
- In first degree heart , 2nd degree and complete heart block.
- Digitalis therapy.
- Hyperkalemia.

Q Waves

- Physiological Q waves do not exceed 0.04 seconds in duration and they do not exceed one –fourth of R wave height.
- Pathological Q wave-exceede 0.04 seconds in duration and more than one fourth of R amplitude.
- It is present in several leads instead of isolated leads seen in physiological Q wave.
- Severe angina, hypoxia,hypothermia or hypoglycemia may cause transient appearance of pathological Q wave.
- Pathological Q waves are commonly seen in myocardial Infarction as infarcted tissue is electrically inert and does not get depolarized.

Low voltage QRS complex.

- When the height of R or S wave is not more than 5mm… it is seen in..

- ✓ Hypothyroidism.
- ✓ Pericardial effusion.
- ✓ Thick chest wall.
- ✓ Problem in ECG machine.

High voltage QRS complex.

- This is present in ventricular hypertrophies.
- The maximum voltage of QRS complex may be 35 mv(35 small square).
- V1 and V2 show high voltage QRS complex in right ventricular hypertrophy.
- V5 and V6 show such QRS complex in left ventricular hypertrophy.

T Wave.

- It represent the ventricular repolarization.
- It is repolarizing wave but shows the upward deflection because the part depolarized in the last is first to be repolarized,, that is base of heart depolarized in the last but is first to be repolarized.

- T wave should not be more than one third of R wave.
- T wave inversion represent ischemia of heart.
- Tall and peaked R wave is present in hyperkalemia.
- Flattened R waves in pericarditis and myocarditis.

QT interval

- Measured from beginning of Q to the end of the T wave
- Its duration is about (10 small sqrs).
- It indicates total systolic time of ventricles.
- QT interval shortens at faster heart rates and lengthens at slow heart rate.
- QT interval needs to be corrected for the heart rate
- The corrected QT is known as QTc.

- QTc =QT/Square root of RR Interval.

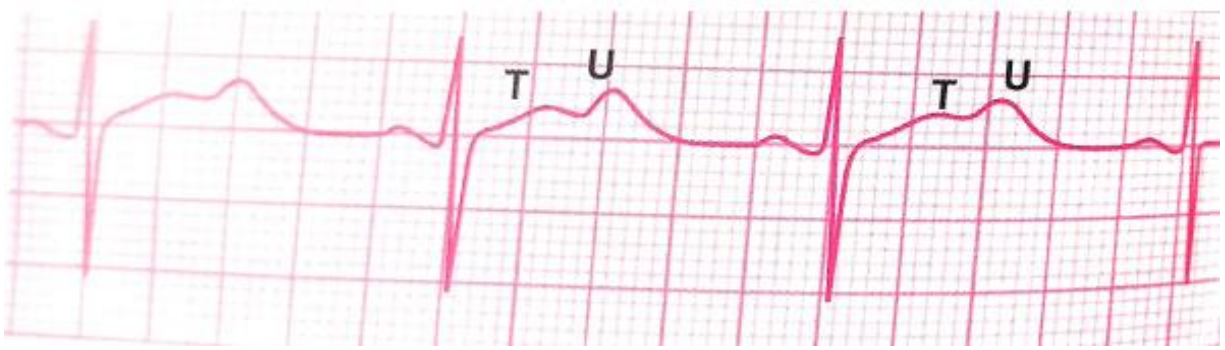
Shortened QT Interval

- QTc less than0.35 seconds is considered short.
- Causes
- ✓ Hyperkalemia
- ✓ Hypercalcemia
- ✓ Digitalis effect
- ✓ Acidosis
- ✓ Hyperthermia
- Hyperkalemia is associated with tall T waves, wide QRS complexes and diminished P waves.
- Hypercalcemia- is not associated with changes deflection of QRS or P and T waves.

Prolonged QT interval

- QTc greater than 0.43 seconds
- Causes can be either congenital or acquired
- Congenital-Romano ward syndrome or Jerwell – Lange-Neilson syndrome
- Acquired can be
- Eletrolyte deficiency –potassium ,Calcium
- Antiarrhythmic drugs-quinidine,Amiodarone.
- Acute myocardial infarction.
- Acute myocaditis –viral myocarditis,Rheumatic fever.
- Intracranial event-head injury,hemorrhage
- AV block,sinus bradycardia.
- Psychotropic drugs-TCAs.

- Hypocalcemia produces true prolongation of QT interval without any alteration in ST segmentor T wave.
- In Hypokalemia the T wave is flattened and the prominent U wave may be mistaken to be T wave.
- It may be Pseudo-prolongation of QT interval.

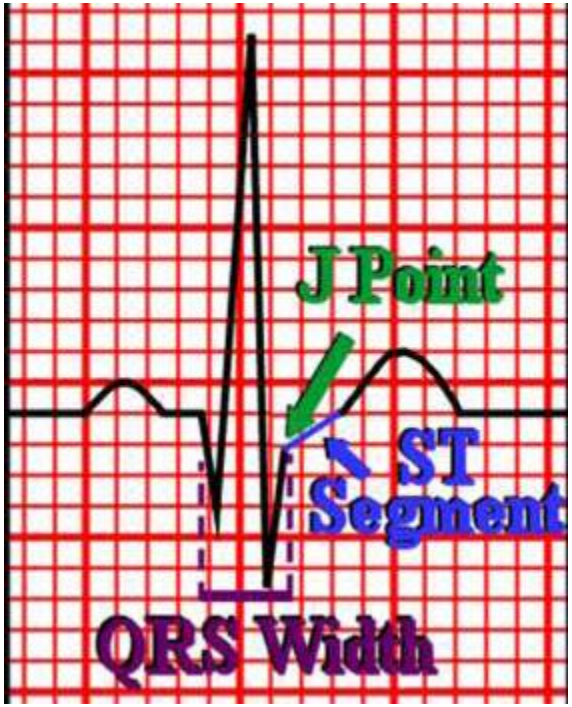


ST segment.

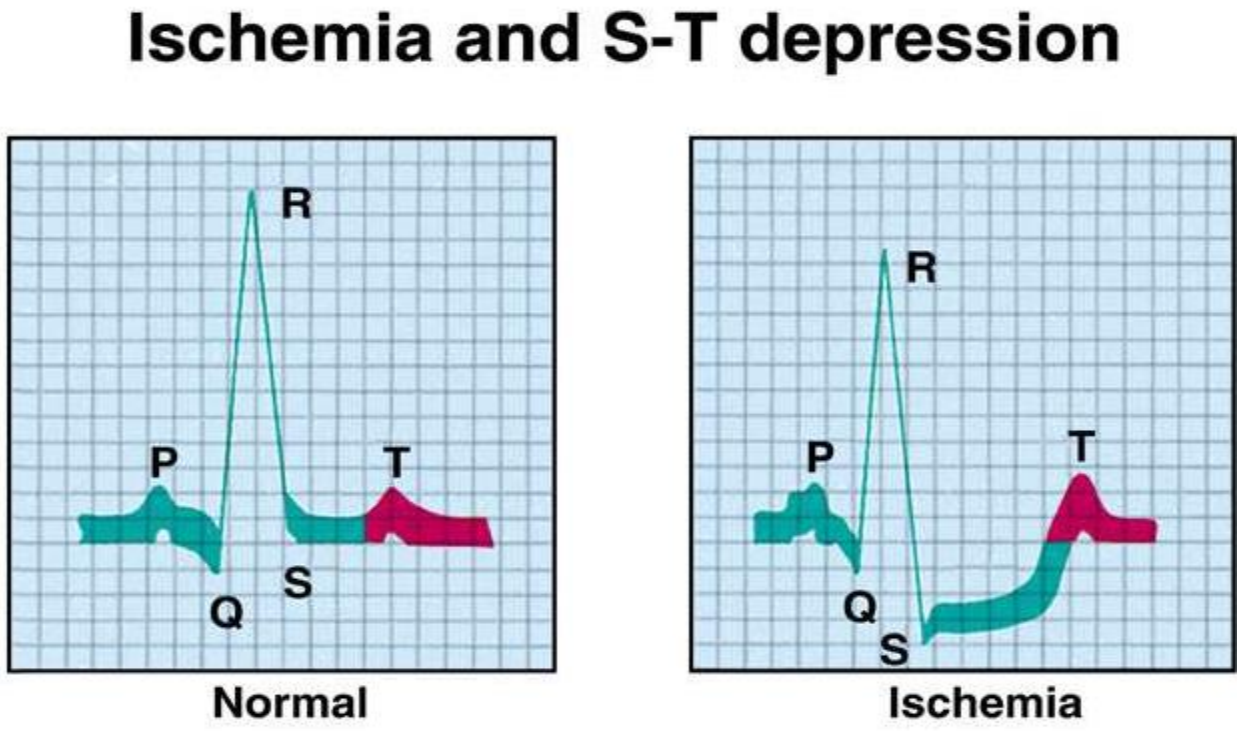
- This segment present between S wave and T wave.
- It represent the plateau phase.
- its duration is 0.04 sec .

J point.

- The exact point at which all parts of ventricles are depolarized i.e at the just end of QRS complex and just at the beginning of ST segment.


J Point
ST
Segment
QRS Width

- ST segment Elevation .
- Seen in recent MI and hyperkalemia.
- ST segment Depression.
- Seen in ischemia, digitalis therapy and hypokalemia.

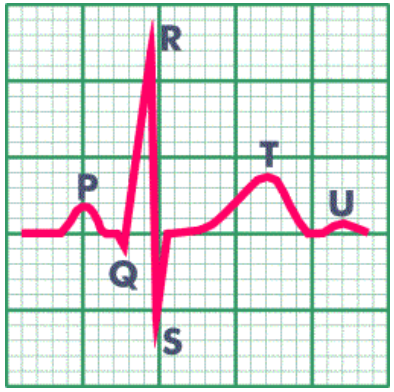


The classical ECG pattern of myocardial infarction is reflected by three changes.

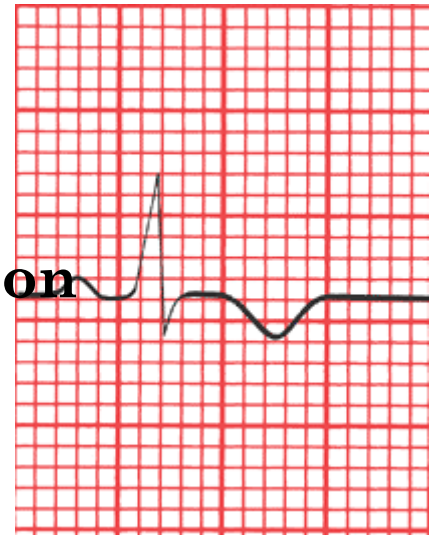
- T-wave inversion
- ST-segment elevation
- Appearance of Q waves

The leads in which ECG changes occur reflect the anatomic site of the infarction

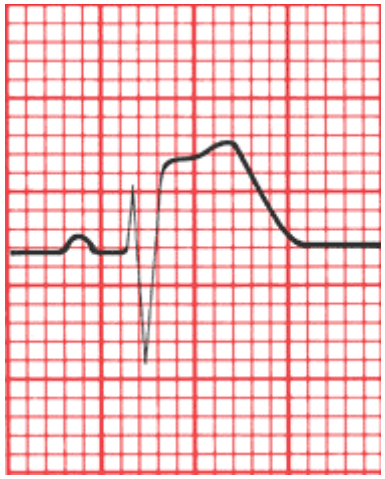
The normal ECG

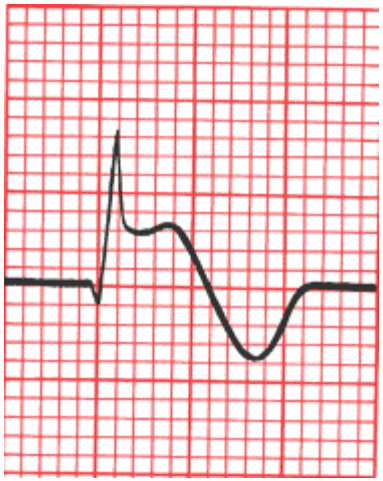


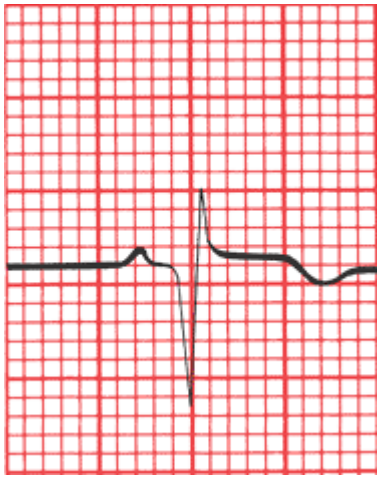
T wave inversion



ST Segment elevation







Q waves

- T- wave inversion or ST segment depression suggest acute ischemia
- ST segment elevation or Q wave suggest acute infarction.
- ST segment elevation is the early sign of MI and occurs within hours of onset of symptoms.
- As the infarction evolves changes to the QRS complex include loss of R- wave height and development of pathologic Q waves.
- Q waves may appear within 1-2 hours, and occasionally, upto 24 hours

IMAGING STUDIES

- No: of imaging modalities contribute to the diagnosis and assessment of ACS, they include

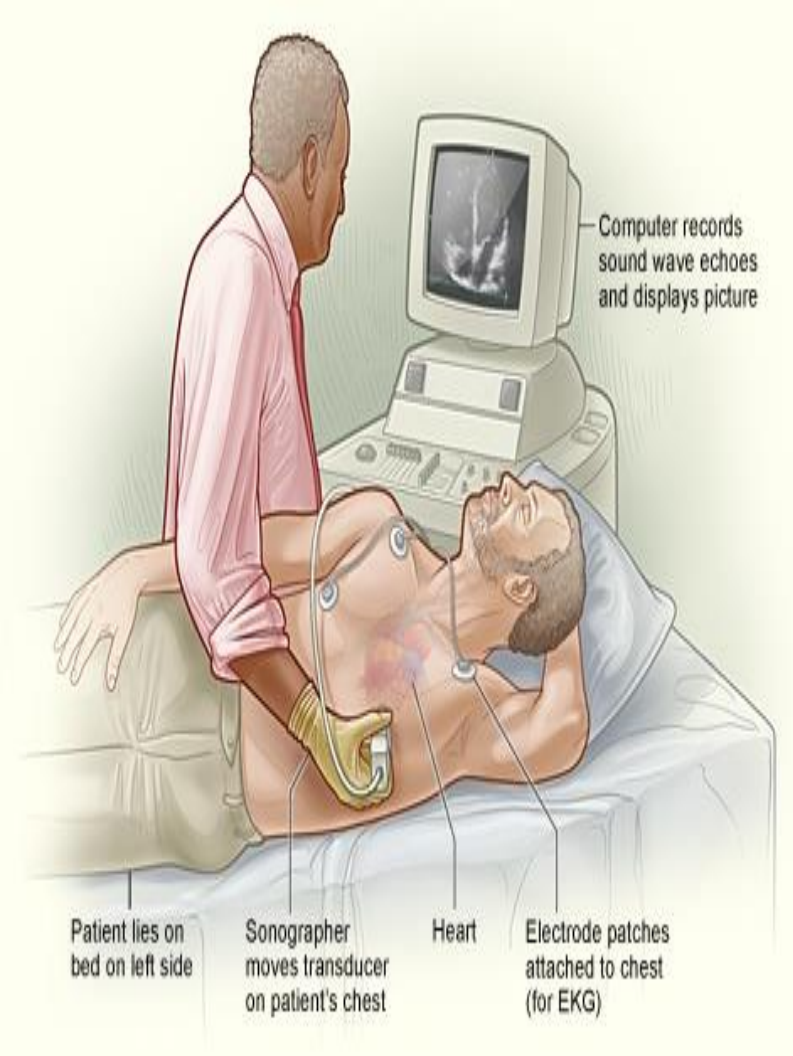
- ✓ CHEST RADIOGRAPHY
- ✓ ECHOCARDIOGRAPHY
- ✓ CARDIAC CATHETERIZATION
- ✓ PERFUSION IMAGING
- ✓ COMPUTED TOMOGRAPHY
- ✓ MAGNETIC RESONANCE IMAGING
- ✓ POSITRON-EMISSION TOMOGRAPHY

CHEST RADIOGRAPHY

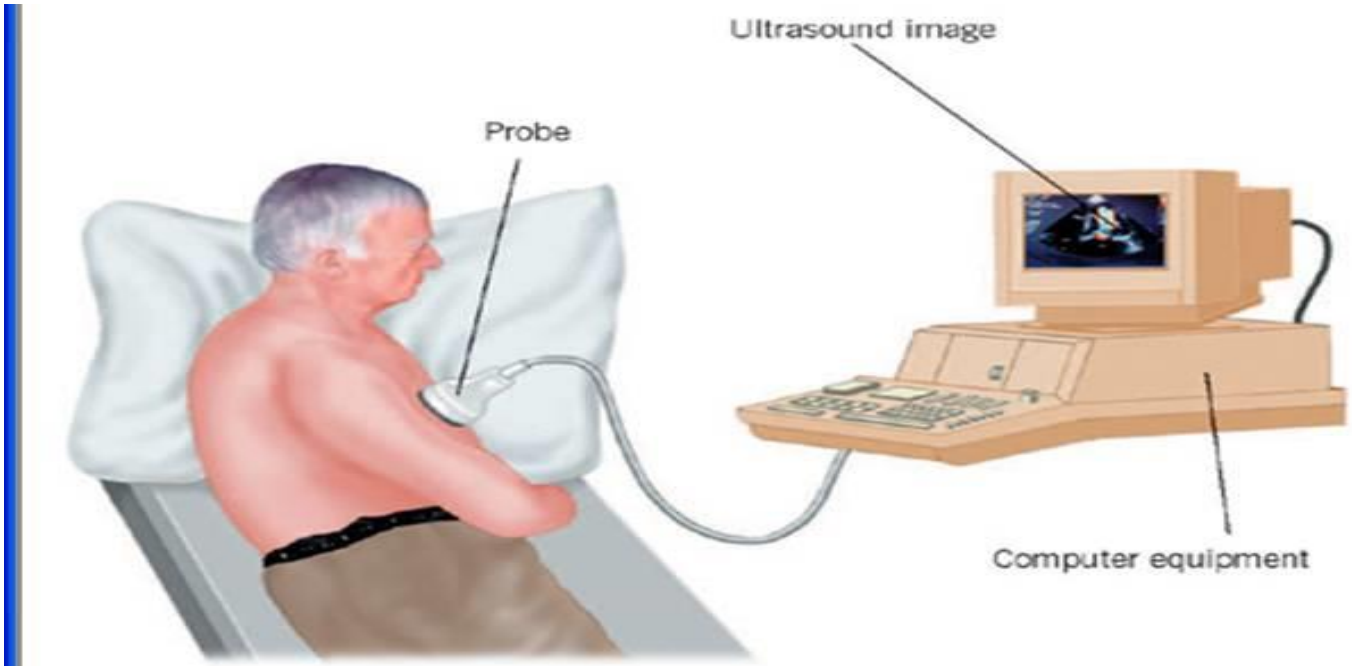
- Chest radiography at the initial presentation of patients with ACS provides an early estimation of the size of the left heart chambers
- In addition, the presence and degree of pulmonary congestion indicates elevated left-ventricular end diastolic pressure, which may result from the sizable infarction of the left ventricle.
- Chest radiography is a std study in evaluating pts presenting with symptoms suggestive of HF.
- Chest radiography findings of heart failure include cardiac enlargement, vascular redistribution, interstitial and alveolar edema, peribronchial cuffing and pleural effusions

ECHOCARDIOGRAPHY

- It is based on the sound transmitted to and through the heart.
- Different tissues present varying resistance to transmitting sound.
- Recording of the position and motion of the heart walls or internal structures of the heart by the echo obtained from the beams of ultrasonic waves directed through the chest wall.

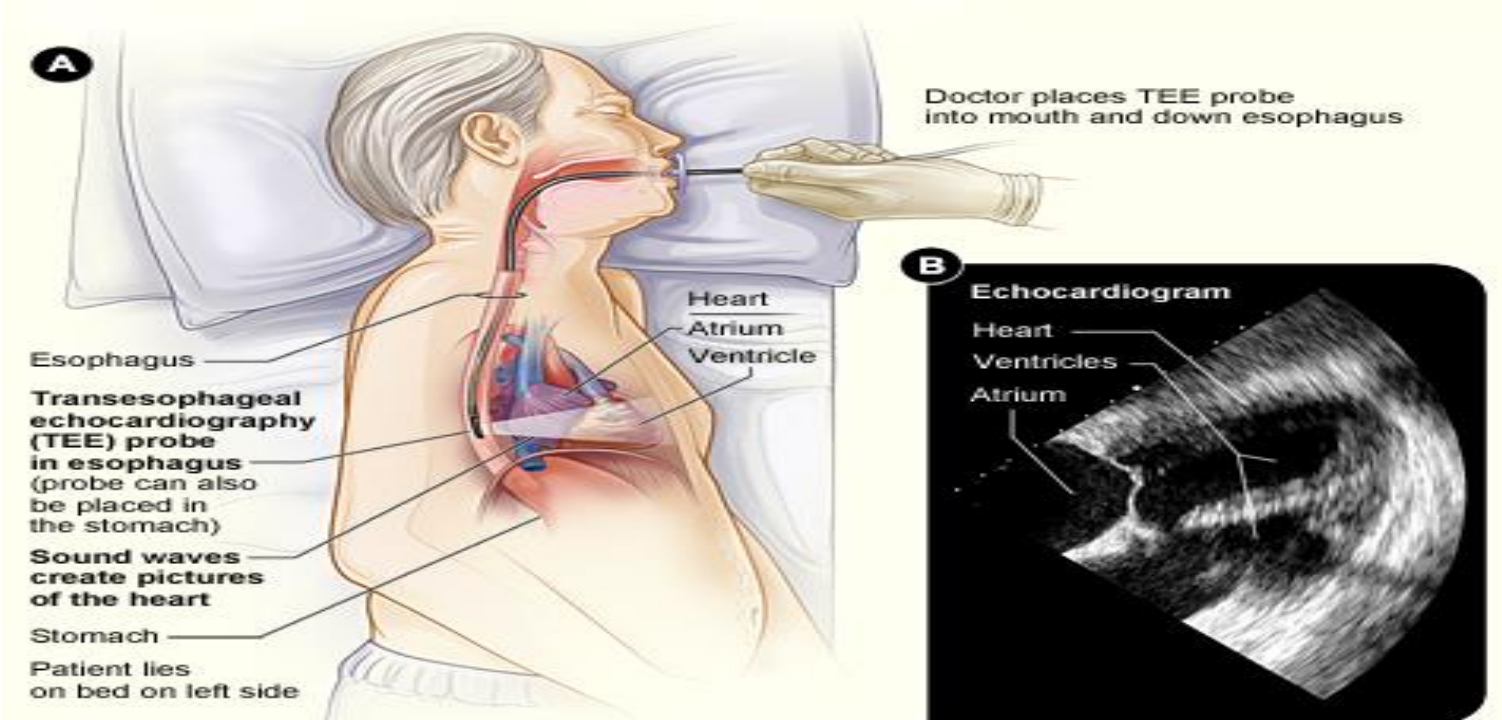


- Transthoracic echocardiography (TTE)involves sound waves from a transducer positioned on the anterior chest directed across cardiac tissues.
- The sound is reflected back in different frequencies and images of cardiac anatomy are displayed on an electronic monitor



Echocardiography uses inaudible sound beams (ultrasound) to take pictures of the

- Transesophageal echocardiography involves mounting the transducer at the end of a flexible endoscope and passing it through the esophagus to position it closer to the heart.
- TEE provides higher resolution of the posterior cardiac structure making it ideal for viewing the atria, cardiac valves and aorta.TEE has no role in the diagnosis or assessment of pts with ACS.



- Two dimensional echocardiography records multiple views providing cross sectional images of the heart. Clinical use of this include anatomic assessment of heart and functional assessment of cardiac chambers and valves.

- Doppler echocardiography use sound or frequency ultra sound to record the velocity and direction of blood and wall motion. It is based on the principle of bouncing ultrasound off of a moving object.(eg RBC).This method permits the assessment of valvular and wall motion abnormalities..

CARDIAC CATHETERIZATION

- It involves the introduction of a radio opaque catheter through the arm vein (femoral or brachial artery), which is advanced to the heart chambers or great vessels. its passage through the heart can be watched on a screen.
- Measurement collected include intracardiac pressure, hemodynamic data and blood flow in the heart chamber and coronary arteries, thus aids in the diagnosis of heart abnormalities.

MYOCARDIAL PERFUSION IMAGING

- Clinically most imp. application of MPI is detection of AMI.
- Myocardial Perfusion Imaging is a nuclear medicine test which is performed in conjunction with exercise or pharmacologic stress testing.
- A radiotracer (Myoview) is administered during stress and rest, followed by images of cardiac perfusion obtained with a gamma camera.
- Thallium and Myoview are radiotracers which are used to assess myocardial perfusion.Thallium-201 is the original cardiac perfusion radiotracer utilized for non-invasive evaluation of coronary artery disease.

- Myoview (tetrofosmin) is a 99m-technetium labeled cardiac perfusion tracer. The physical characteristics of 99m-technetium are optimal for today's gamma camera.
- (MPI) shows how well blood flows through (perfuses) heart muscle. It can show areas of the heart muscle that aren't getting enough blood flow. This test is often called a nuclear stress test. It can also show how well the heart muscle is pumping.
- This may be used for the assessment of thrombolytic therapy effectiveness and early risk stratification of pts. presenting with AMI or ACS.

COMPUTED TOMOGRAPHY

- CT scanning is used rarely as a primary diagnostic procedure in the evaluation of CVD and function because it provides similar information as other diagnostic procedures (e.g., ECHO) and is significantly more expensive.
- Enhanced definition and spatial resolution of structures makes CT scan useful in some specific indications such as to evaluate aortic and pericardial disease and assess paracardiac and cardiac masses.

- CT scanning helps in more accurate determination of chamber volume and size .
- Also mass calculations of myocardial wall thickness can be obtained from CT scanning than with other methods.
- New techniques such as ultrafast CT (cine-CT) scanning have resolved the problems of cardiac motion that distorted conventional CT images.

- With cine-CT scanning, complete tomograms are assembled within one cardiac cycle (50 ms), thus providing real-time images

- Cardiac CT is useful in the assessment of cardiac structure including cardiac masses, pericardial conditions, evaluation of aortic and pulmonary disease

MAGNETIC RESONANCE IMAGING

- MRI is a noninvasive imaging technique capable of detailed tissue characterization and blood flow measurements.
- The procedure involves placing pts in a device generating a powerful magnetic field and aligning the protons of the body's hydrogen atom relative to the magnetic field.
- Radio waves pulsed through the field force the proton to shift their orientation.
- When the radio waves stop, the protons return to their previous orientation, releasing energy in the form of radio waves

- The waves are detected by the scanner and converted to images. The images are physiologically gated to an ECG.

LIMITATIONS OF MRI

- During the procedure the patients are required to remain motionless.
- Claustrophobic patients may not be able to undergo the procedure and sedation may be necessary.
- In addition patients with metal prosthesis should not undergo MRI.(pacemaker, ferro magnetic intracerebral clips)

CLINICAL USE OF MRI:

- Clinical use of cardiac MRI include assessment of congential, aortic and pericardial diseases, tumors and intravascular thrombus.
- Currently it is used in the evaluation of chest pain syndrome with use of vasodilator perfusion CMR or dobutamine stress function CMR in patients with intermediate pretest probability of ischemic heart disease in the setting of uninterpretable ECG or inability to exercise or for evaluating suspected coronary anomalies.

POSITRON EMISSION TOMOGRAPHY (PET SCAN)

- It is a nuclear imaging technique capable of measuring myocardial blood flow and cellular metabolism of substrates(fatty acids,glucose, oxygen)invivo.
- The mechanism uses the properties of short lived positron- emitting ,isotope labelled compounds(N-13,O-15,C-11,F-18)coupled with mathematical models of physiological function.

CLINICAL USE OF PET:

- Used for detecting ischemic but viable myocardium that appears irreversibly necrotic by other diagnostic tests.

MISCELLANEOUS LABORATORY TESTS:

- The number of non cardiac specific laboratory abnormalities may be manifested in patients with AMI.
- Recognition of these abnormalities as secondary to AMI precludes misinterpretation of other disorders.

Serum Glucose:

- *Normal range:*70-110 mg/dl or 3.9-6.1mmol/l(when fasting)
- In case of AMI:

An elevation of serum glucose is seen in patients(both diabetic and nondiabetic) for several weeks due to stress.

This can be differentiated by measurement of glycosylated haemoglobin.

WBC:

- *Normal range:* 4.8- 10.8×10^3cells/mm^3 .

In case of AMI:

- An elevation of WBC is seen in patients due to myocardial tissue necrosis or secondary to increased adrenal glucocorticoid secretion.
- Depending upon extent of tissue necrosis polymorphonuclear leukocytosis of 10000-20000 cells/mm^3may be seen after 12-24 hrs after onset of symptoms and may last for 1-2 weeks.
- Fever can also accompany leukocytosis.

ESR:

- *Normal range:M*ales:1-15 mm/hr

  Females:1-20mm/hr.

In case of AMI:

- ✓ Acute phase reactants levels increases in patients with AMI by which levels of ESR can also vary.
- ✓ It usually peaks on day 4 or 5 and remains elevated for 3-4 weeks of post AMI.

Lipid Panel:

- Total cholestrol and LDL may be decreased 48-72 hrs of post MI and may persist for 6-8 weeks afterward.

NON INVASIVE STRESS TESTING

- EXERCISE STRESS TESTING

- ✓ a noninvasive test used to evaluate clinical and cardiovascular responses to exercise.
- ✓ It is a simple test that can be conducted in a physician's office and is less expensive.
- ✓ The principle behind EST is to increase myocardial oxygen demand above myocardial oxygen supply and coronary reserve, thereby provoking ischemia (inadequate myocardial perfusion), using exercise as a stressor.

- EST is a very practical test in that it can assess patients' functional capacity.
- LIMITATIONS:
- patients with orthopedic, neurological, peripheral vascular problems.
- patients who receive agents that may blunt heart rate response to exercise(beta blockers,CCBs may not be able to achieve the target heart rate necessary for diagnostic and prognostic purposes.

PHARMACOLOGICAL STRESS TESTING

- ✓ People who are unable exercise may be stressed pharmacologically using either

(1) Vasodilating agents(adenosine,dipyridamole)

(2) Positive inotropic agents(dobutamine)

- ✓ Both modalities produce vasodilatory or increased blood flow response leading to heterogeneity of myocardial blood flow between vascular areas supplied by normal and significantly stenosed coronary arteries.