Acute cardiac ischemia (Acute Coronary Syndromes)

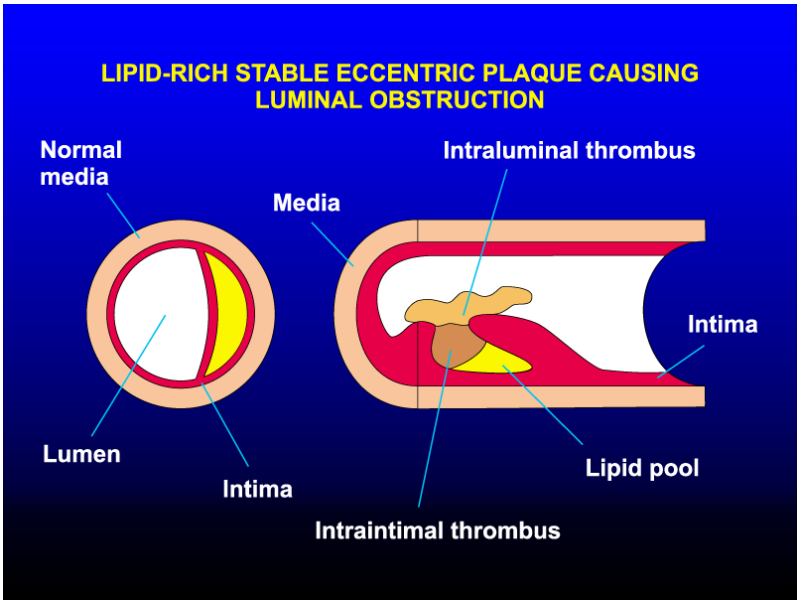
Learning Outcomes

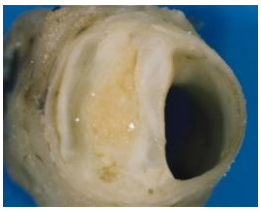
- To *understand* the clinical spectrum of coronary disease
- To *recognise* different presentations of the disease process
- To be *aware* of the different treatment options for each clinical presentation

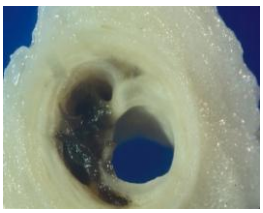
Acute Coronary Syndromes ” ACS”

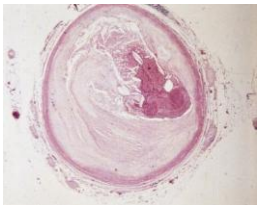
Spectrum of clinical presentation caused by:

- Atherosclerotic plaque rupture
- Smooth muscle constriction
- Thrombus formation





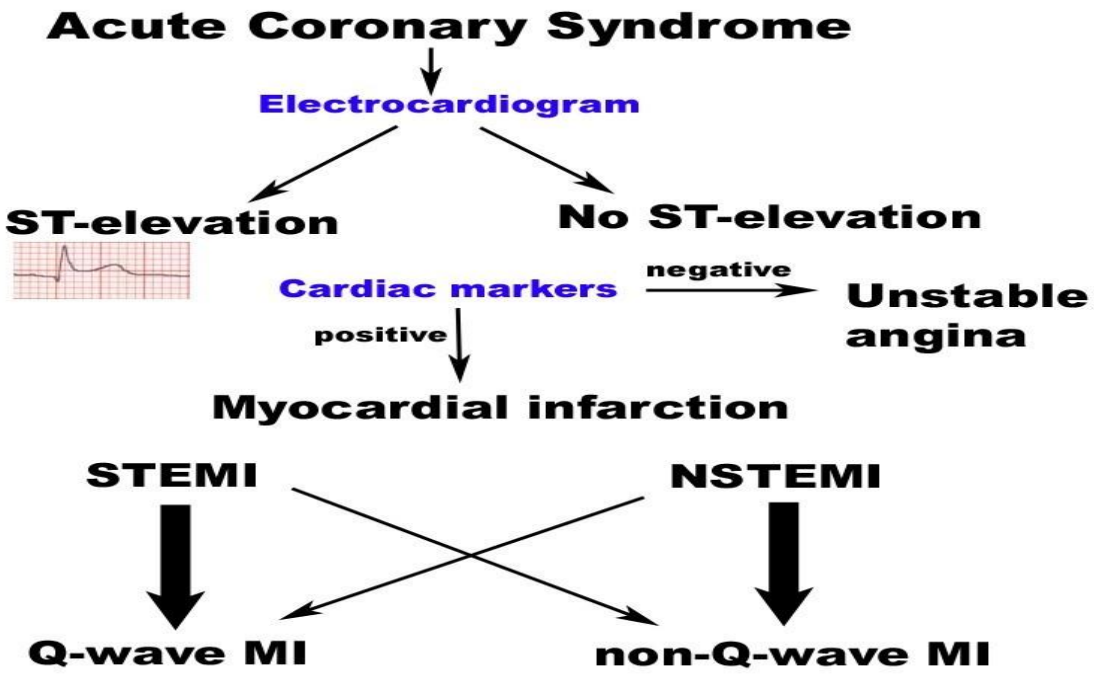




Classification of ACS

1- **Unstable angina**
2- **Non-ST-elevation myocardial infarction**
3- **ST-elevation myocardial infarction**

Diagramatic classification



Stable angina

Pain or discomfort from myocardial ischemia:

- **Tightness , chest ache**
- **May radiate to throat, arms, shoulder, back,epigastrium**
- **Consistently provoked by exercise**
- **Settles when exercise stops**

NOT an acute coronary syndrome

Unstable Angina

1. **Angina on exertion with increasing frequency over a few days, provoked by less exertion**

OR

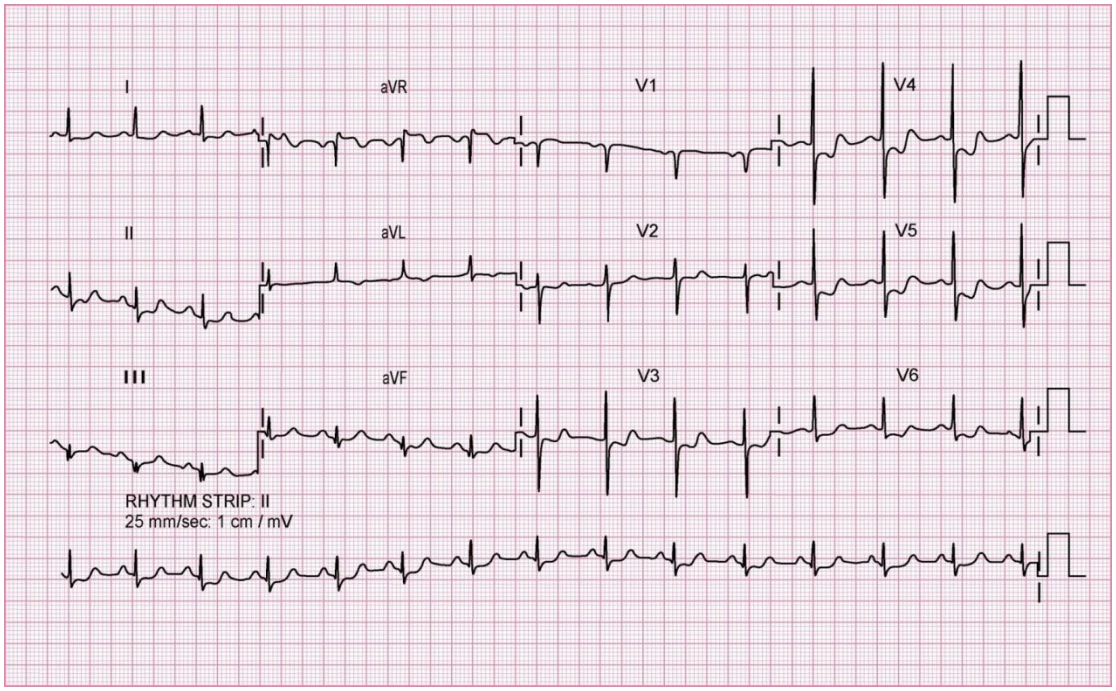
1. **Angina occurring recurrently and unpredictably - not specific to exercise**

OR

1. **Unprovoked and prolonged episode of chest pain**

- **ECG may be normal**
- **ST segment depression suggests high risk**
- **No troponin release**
- **Cardiac enzymes usually normal**

Acute ST depression



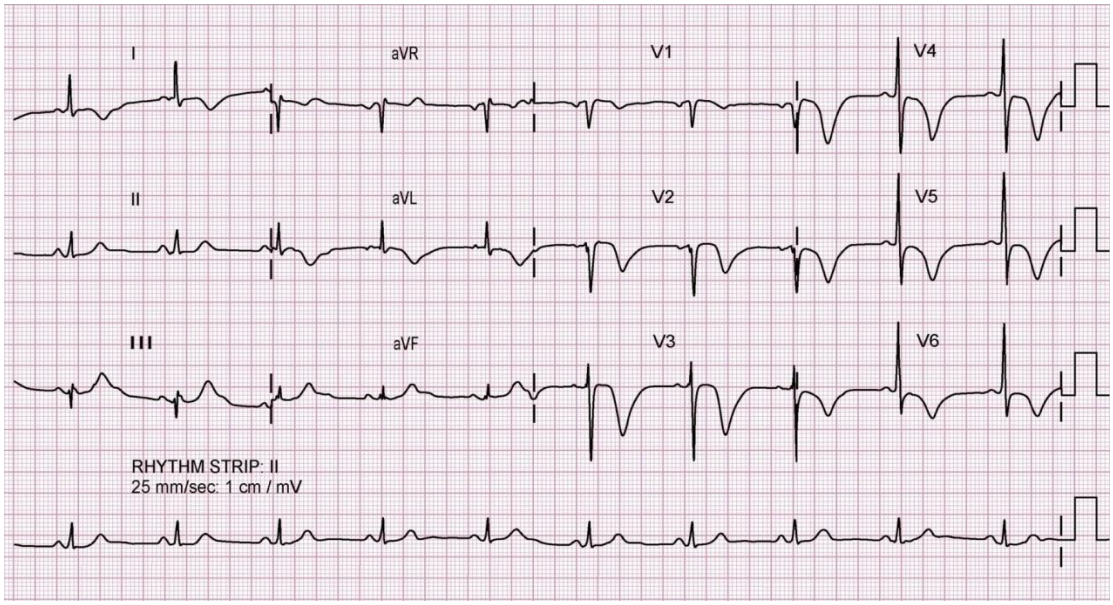
Non-ST-elevation myocardial infarction "NSTEMI"

- **Symptoms suggesting acute MI**
- **Non-specific ECG abnormalities**

- ST segment depression
- T wave inversion

- Troponin release
- Usually elevated cardiac enzymes
  - e.g. creatine kinase (CK)

NSTEMI

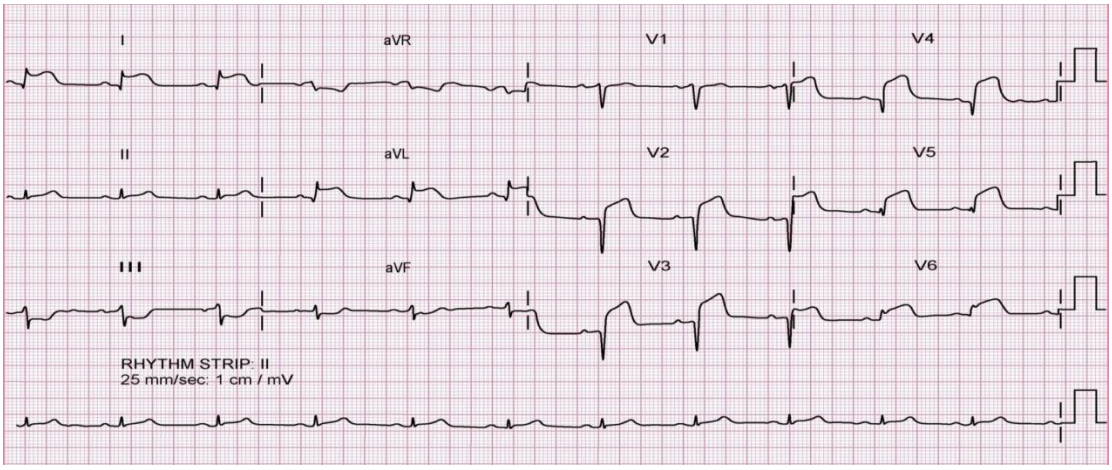


STEMI

- Symptoms suggesting acute MI
- Acute ST segment elevation
- Q waves likely to develop
- Troponin release
- Usually elevated cardiac enzymes (e.g. CK)

- ***<u>Early effective treatment may limit myocardial damage and prevent Q wave development</u>***

STEMI



General diagnosis of MI

- **The onset is usually gradual.**
- **Chest pain is often tightness, pressure, or squeezing.**
- **Radiating mostly to the left arm, the lower jaw, neck, right arm, back, and epigastrium.**
- **Levine's sign, thought to be predictive of chest pain, where the patient localizes the chest pain by clenching his fist over the sternum.**
- **Dyspnea**
- **Diaphoresis**
- **Increasing of cardio-specific enzymes – Troponin.**
- **Echocardiography :akinesia, hypokinesia.**
- **Autopsy of myocardium (thrombus ,necrosis.(**
- **ECG: Acute changes segment ST, acute left bundle- branch block.**
- **ECG: Pathological Q.**

- **New elevation of ST at 2 or more leads .**
- **In ECG Q wave MI – pathological Q and inversion T.**
- **In ECG non Q wave MI – complex QRS is normal, no pathological Q, but it is inversion T.**

- **ECHO Criteria**

- **New regional wall motion abnormalities on an Echo are also suggestive of MI .**

- **Akinesia, hypokinesia, post infarction cardiosclerosis, myocardial aneurysm**

- **, intracardiac thrombus, systolic or diastolic dysfunction of myocardium, EF , pulmonary hypertension, hypertrophy of LV, LA etc.**

Treatment

Immediate ttt for ACS "all types"

ABCDE approach

- **Aspirin 300 mg orally (crush/chew)**
- **Nitrate (GTN spray or tablet)**
- **Oxygen if appropriate ******
- **Morphine (or diamorphine)**
- **Anti-thrombotic**
  - **Aspirin**
  - **Clopidogrel or prasugrel**
  - **LMW heparin or fondaparinux**
  - **If very high risk: glycoprotein IIb/IIIa inhibitor**
- **Pain relief**
  - **Nitrate "2x1"**
  - **Morphine**
- **Oxygen if appropriate *****

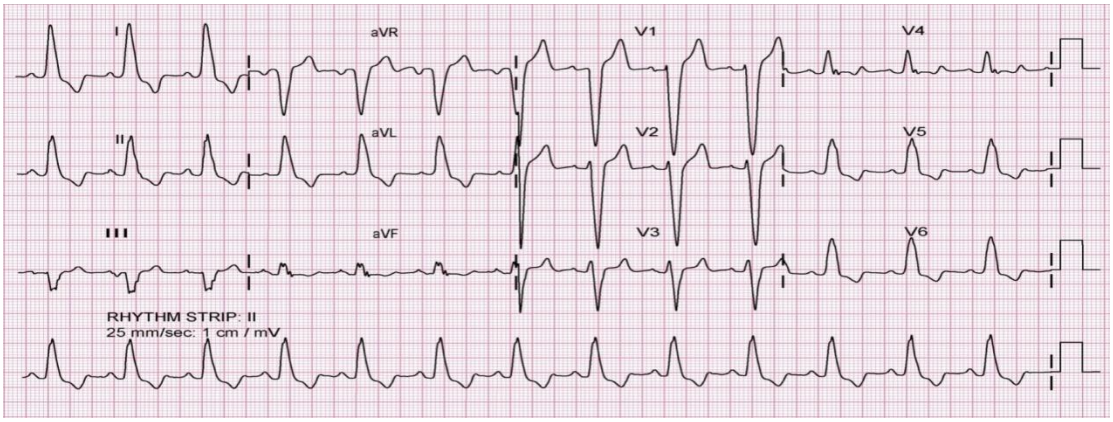
- **Myocardial protection**
  - **Beta blocker**
  - **Coronary angiography/PCI in most patients**

Emergency reperfusion therapy:

- **Percutaneous coronary intervention (PCI)**

Fibrinolytic therap Avoid delay – "Time is muscle"

LBBB



Absolute contraindications to fibrinolytic therapy

- **Previous haemorrhagic stroke**
- **Other stroke or CVA within 6 months**
- **CNS damage or neoplasm**
- **Active internal bleeding**
- **Aortic dissection**
- **Recent major surgery or trauma**
- **Known bleeding disorder**

STEMI – further management

- **Anti-thrombotic therapy**

- **Beta blocker**
- **ACE inhibitor**
- **Coronary angiography and reperfusion strategies e.g. PCI**

Brady arrhythmia

Learning outcomes

At the end of this subject you should:

- **Be able to recognize bradycardia and differentiate between the different degrees of heart block**
- **Understand the principles of treating bradycardia**
- **Understand the indications for cardiac pacing**
- **Be aware of the different methods available for cardiac pacing**
- **Know how to apply non-invasive, transcutaneous electrical pacing safely and effectively**

Normal Impulse Conduction

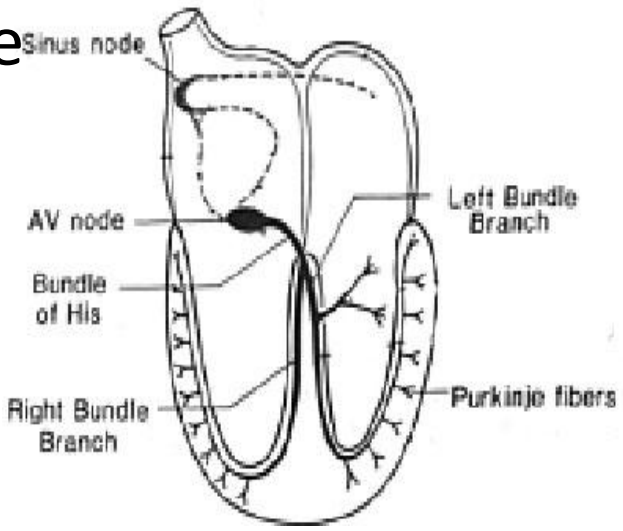
Normal Impulse Conduction

Sinoatrial node

AV node

Bundle of His

Bundle Branches



P wave = Atrial depolarization

QRS = Ventricula depolarization

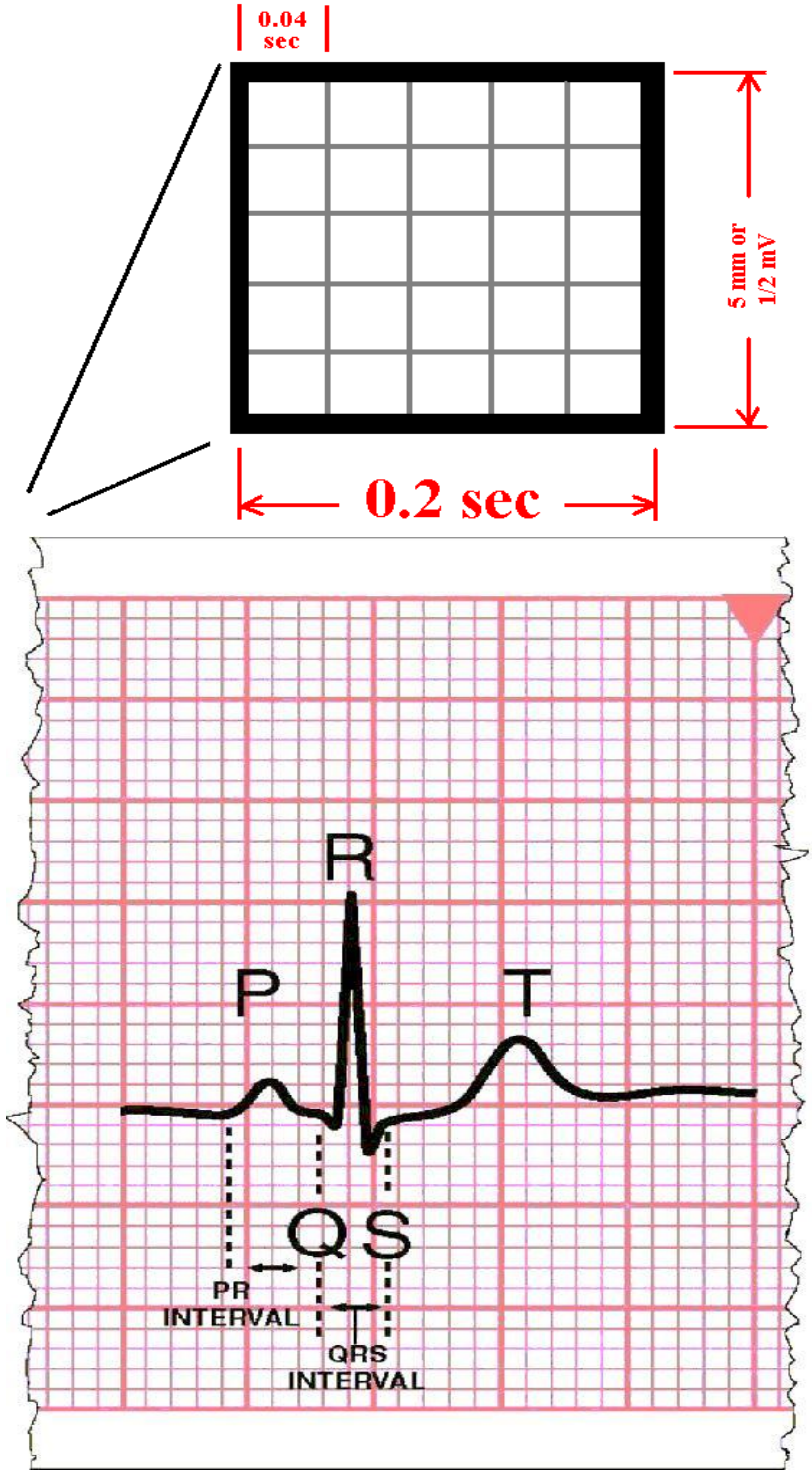
T wave =Ventricular repolarization

Pacemakers of the Heart

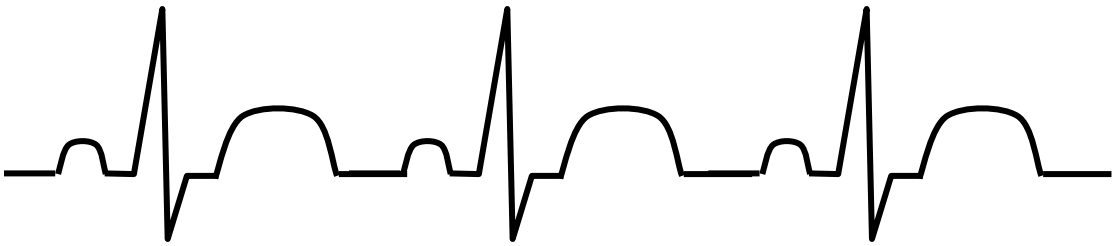
- **SA Node - Dominant pacemaker with an intrinsic rate of 60 - 100 beats/minute.**
- **AV Node - Back-up pacemaker with an intrinsic rate of 40 - 60 beats/minute.**
- **Ventricular cells - Back-up pacemaker with an intrinsic rate of 20 - 45 bpm.**

The ECG Paper

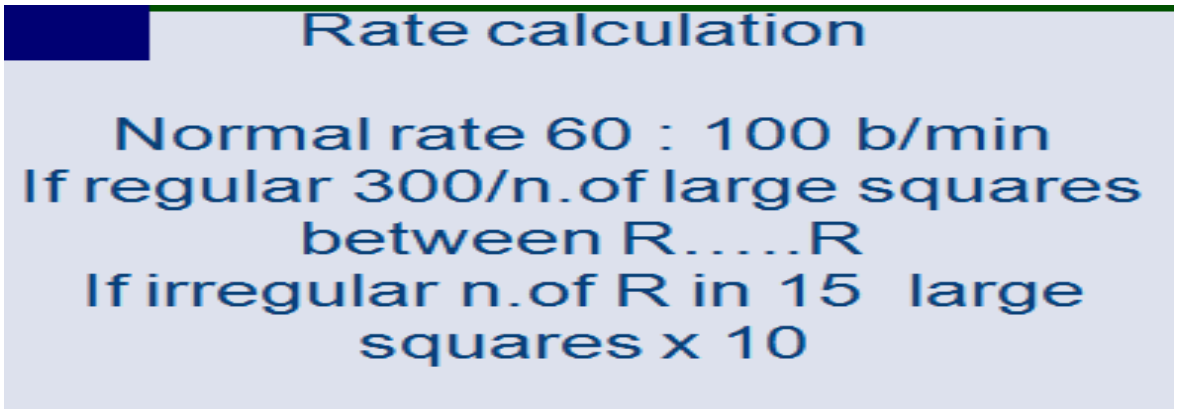
- **Horizontally**
  - **One small box - 0.04 s**
  - **One large box - 0.20 s**
- **Vertically**
  - **One large box - 0.5 Mv**



Normal sinus rhythm



- **each P wave is followed by a QRS**
- **P waves normal for the subject**
- **P wave rate 60 - 100 bpm with <10% variation**
- **rate <60 = sinus bradycardia**
- **rate >100 = sinus tachycardia**
- **variation >10% = sinus arrhythmia**



Abnormal rhythms from the Sinus Node

Sinus Tachycardia:

HR > 100 b/m

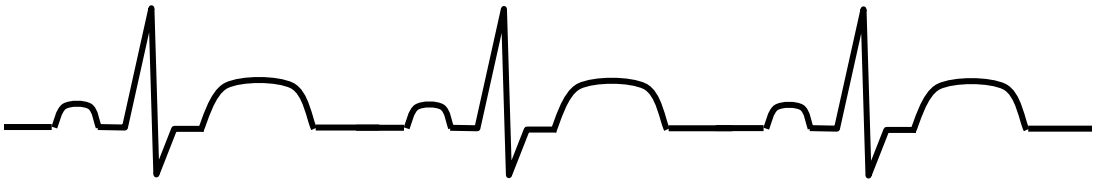
- **Causes:**
  - **Withdrawal of vagul tone & Sympathetic stimulation (exercise, fight or flight)**
  - **Fever & inflammation**
  - **Heart Failure or Cardiogenic Shock (both represent hypoperfusion states)**

- **Heart Attack (myocardial infarction or extension of infarction)**
- **Drugs (alcohol, nicotine, caffeine**

Sinus Bradycardia:

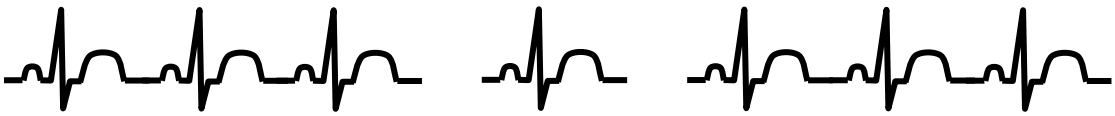
HR < 60 b/m

- **Causes:**
  - **Increased vagul tone, decreased sympathetic output, (endurance training)**
  - **Hypothyroidism**
  - **Heart Attack (common in inferior wall infarction)**
  - **Vasovagul syncope (people passing out when they get their blood drawn)**
  - **Depression**



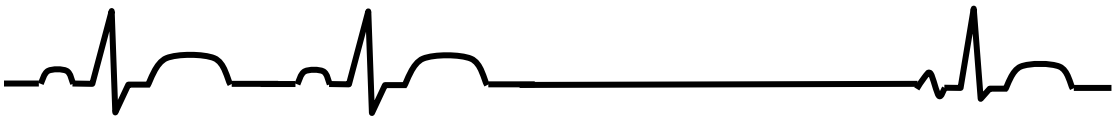
Sinus Arrhythmia Variation in HR by more than .16 seconds

- **Mechanism:**
  - **Most often: changes in vagul tone associated with respiratory reflexes**
  - **Benign variant**
- **Causes**
  - **Most often: youth and endurance training**

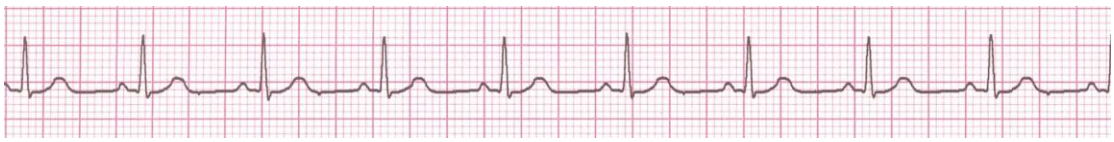


Sick Sinus Syndrome: Failure of the heart's pacemaking capabilities

- **Causes:**
  - **Idiopathic (no cause can be found)**
  - **Cardiomyopathy (disease and malformation of the cardiac muscle)**
- **Implications and Associations**
  - **Associated with Tachycardia / Bradycardia arrhythmias**
  - **Is often followed by an ectopic "escape beat" or an ectopic "rhythm"**



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**

How to read normal and abnormal rhythm strip

Is there any electrical activity? YES

2. What is the ventricular (QRS) rate?

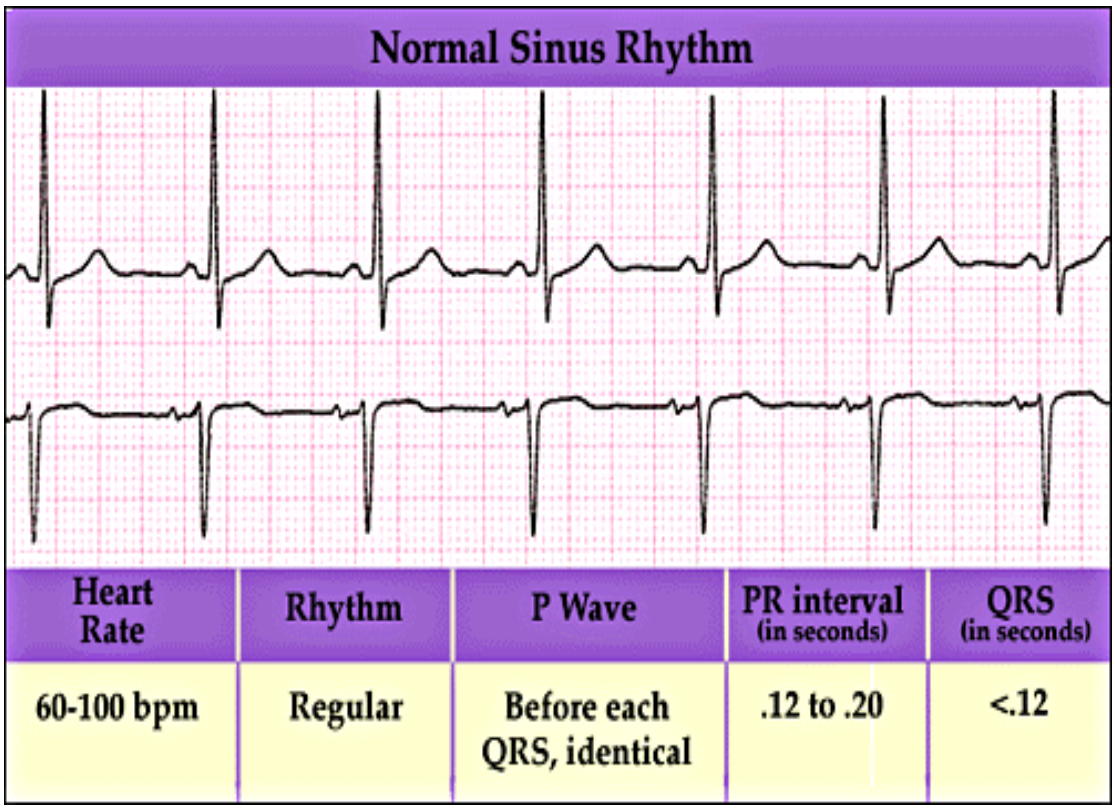
3. Is the QRS rhythm regular or irregular?

4. Is the QRS width normal or prolonged?

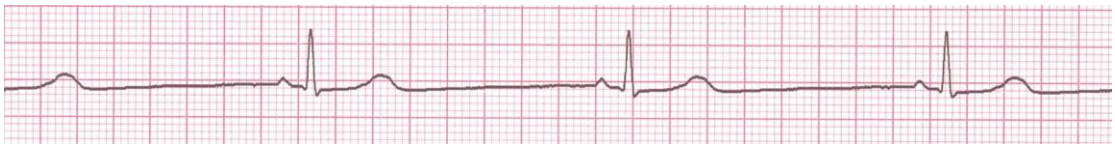
5. Is atrial activity present?

(If so what is it? Normal P waves? Other atrial activity?)

6. How is atrial activity related to ventricular activity?



Rhythm 1 sinus brady cardia



Rate? 30 bpm

Regularity? regular

P waves? normal

PR interval? 0.12 s

QRS duration? 0.10 s

Interpretation? *Sinus Bradycardia*

So , Sinus Bradycardia is :-

- **Deviation from NSR**
- **Rat < 60 bpm**
- **Etiology: SA node is depolarizing slower than normal, impulse is conducted normally (i.e. normal PR and QRS interval).**

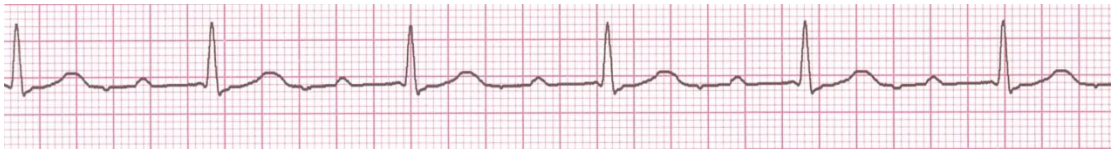
AV Nodal Blocks

- ***1st Degree AV Block***
- ***2nd Degree AV Block, Type I***
- ***2nd Degree AV Block, Type II***
- ***3rd Degree AV Block***

first degree block

- **There is a delay in conduction of the atrial impulse to the ventricles, usually at the level of the AVN.**
- **This results in prolongation of the PR interval to > 0.2 s. QRS complex follows each P wave, and the PR interval remains constant.**

Rhythm 2 first degree block



Rate? 60 bpm

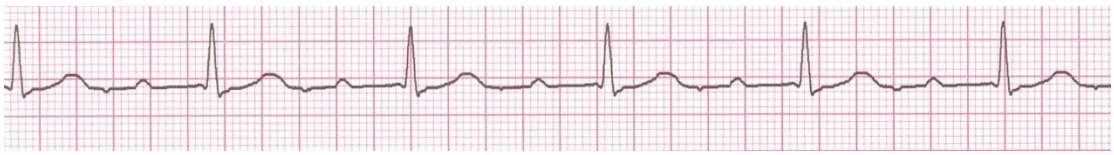
Regularity? regular

P waves? normal

PR interval? 0.36 s

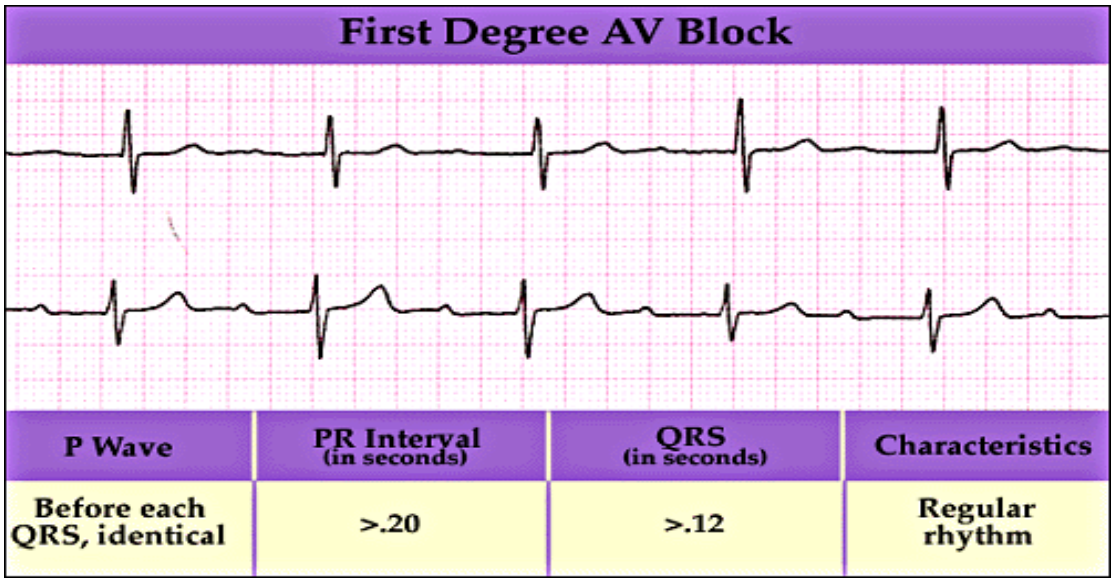
QRS duration? 0.08 s

Interpretation? *1st Degree AV Blockz*



So, 1st Degree AV Block is:-

- **Deviation from NSR**
  - **PR Interval> 0.20**
- **Etiology: Prolonged conduction delay in the AV node or Bundle of His.**



First Degree AV Block

P Wave	PR Interval (in seconds)	QRS (in seconds)	Characteristics
Before each QRS, identical	>.20	>.12	Regular rhythm

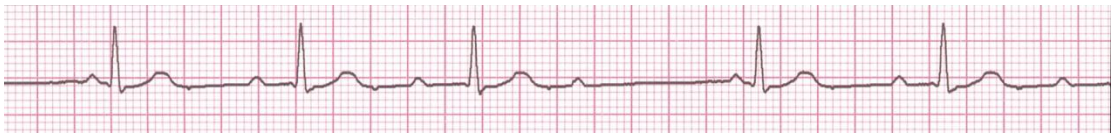
second degree block

- **There is intermittent failure of conduction between the atria and ventricles. Some P waves are not followed by a QRS complex.**

Mobitz type I block (Wenckebach phenomenon)

- **The initial PR interval is normal but progressively lengthens with each successive beat until eventually atrioventricular transmission is blocked completely and the P wave is not followed by a QRScomplex.**
- **The PR interval then returns to normal, and the cycle repeats.**

Rhythm 3 2nd Degree AV Block, Mobitz Type I



Rate? 50 bpm

Regularity? regularly irregular

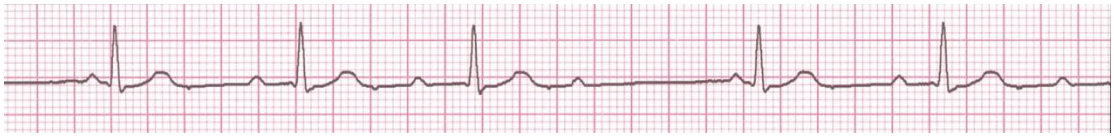
P waves? nl, but 4th no QRS

PR interval? lengthens

QRS duration? 0.08 s

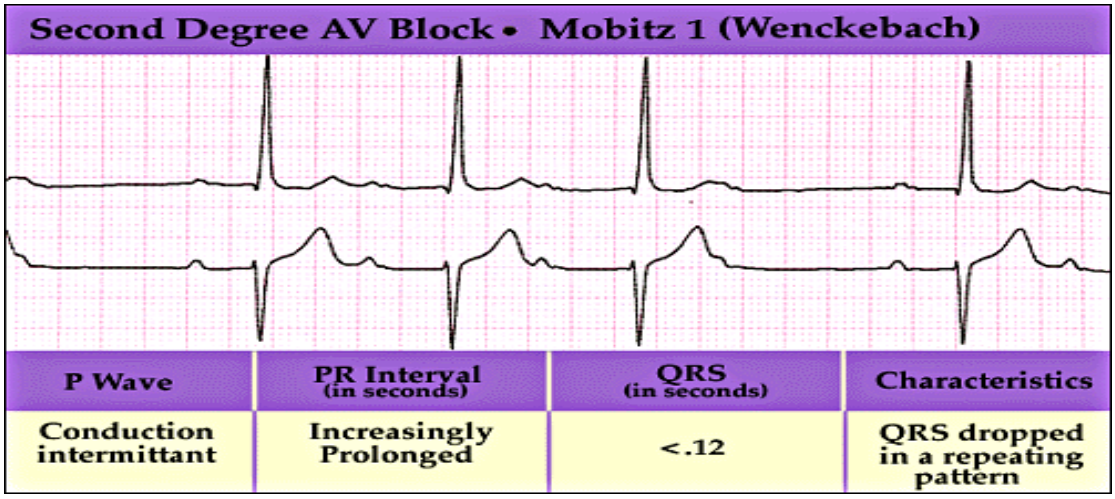
Interpretation? *2nd Degree AV Block, Type I*

So 2nd Degree AV Block, Mobitz Type I is :-



- ***Deviation from NSR***
  - ***PR interval progressively lengthens, then the impulse is completely blocked (P wave not followed by QRS).***

- ***Etiology: Each successive atrial impulse encounters a longer and longer delay in the AV node until one impulse (usually the 3rd or 4th) fails to make it through the AV node.***



2nd degree heart block Mobitz type II

less common but is more likely to produce symptoms. There is intermittent failure of conduction of P waves. The PR interval is constant, though it may be normal or prolonged. The block is often at the level of the bundle branches and is therefore associated with wide QRS complexes.

Progress to complete third degree atrioventricular block.

Rhythm 4 2nd degree heart block Mobitz type II

Rate? 40 bpm

Regularity? regular

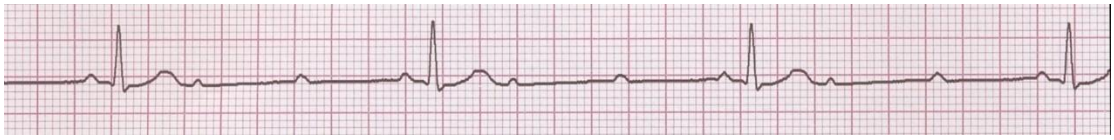
P waves? nl, 2 of 3 no QRS

PR interval? 0.14 s

QRS duration? 0.08 s

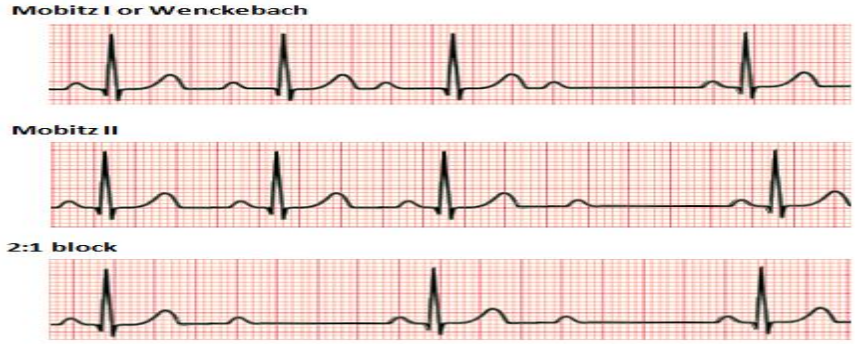
Interpretation? *2nd Degree AV Block, Type II*

So 2nd Degree AV Block, Type II is :-

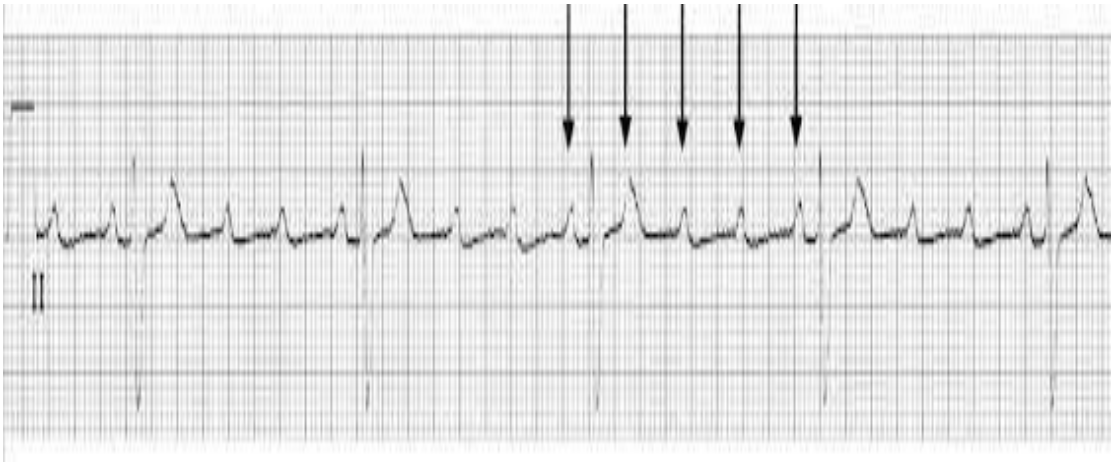


- ***Deviation from NSR***
  - ***Occasional P waves are completely blocked (P wave not followed by QRS).***
- ***Etiology: Conduction is all or nothing (no prolongation of PR interval); typically block occurs in the Bundle of His.***

2;1 AV Block



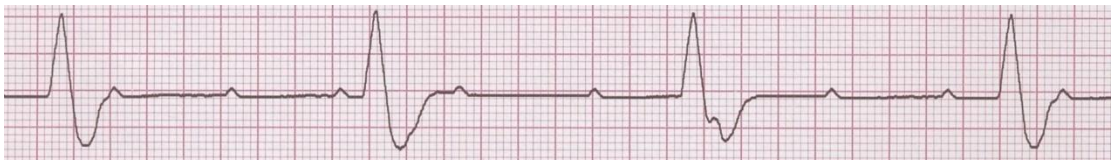
A V Block 2nd degree fixed ratio



Third degree AV block

- *There is complete failure of conduction between the atria and ventricles, with complete independence of atrial and ventricular contractions. The P waves bear no relation to the QRS complexes and usually proceed at a faster rate.(A V dissociation)*

Rhythm 3 *Third degree AV block*



Rate? 40 bpm

Regularity? regular

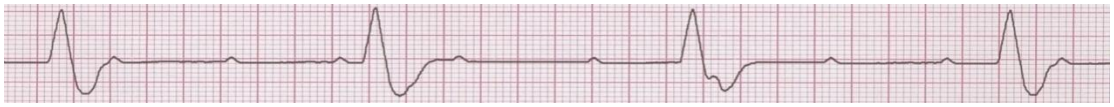
P waves? no relation to QRS

PR interval? none

QRS duration? wide (> 0.12 s)

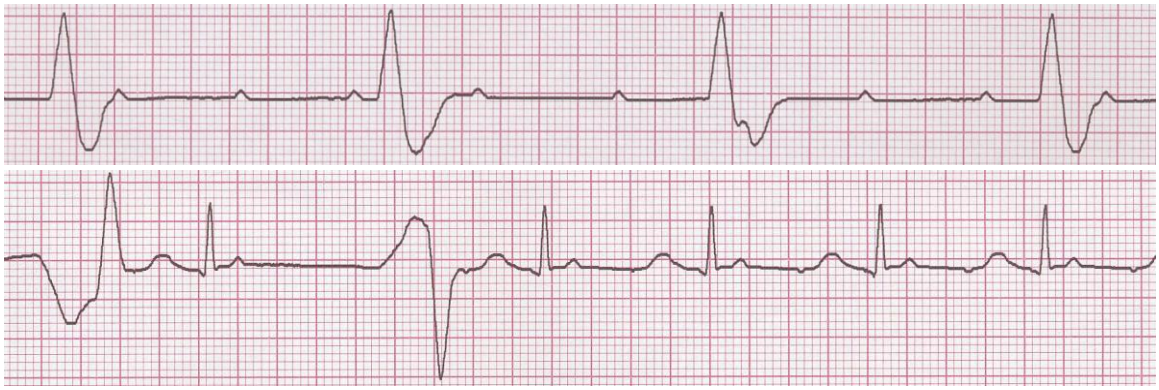
Interpretation? *3rd Degree AV Block*

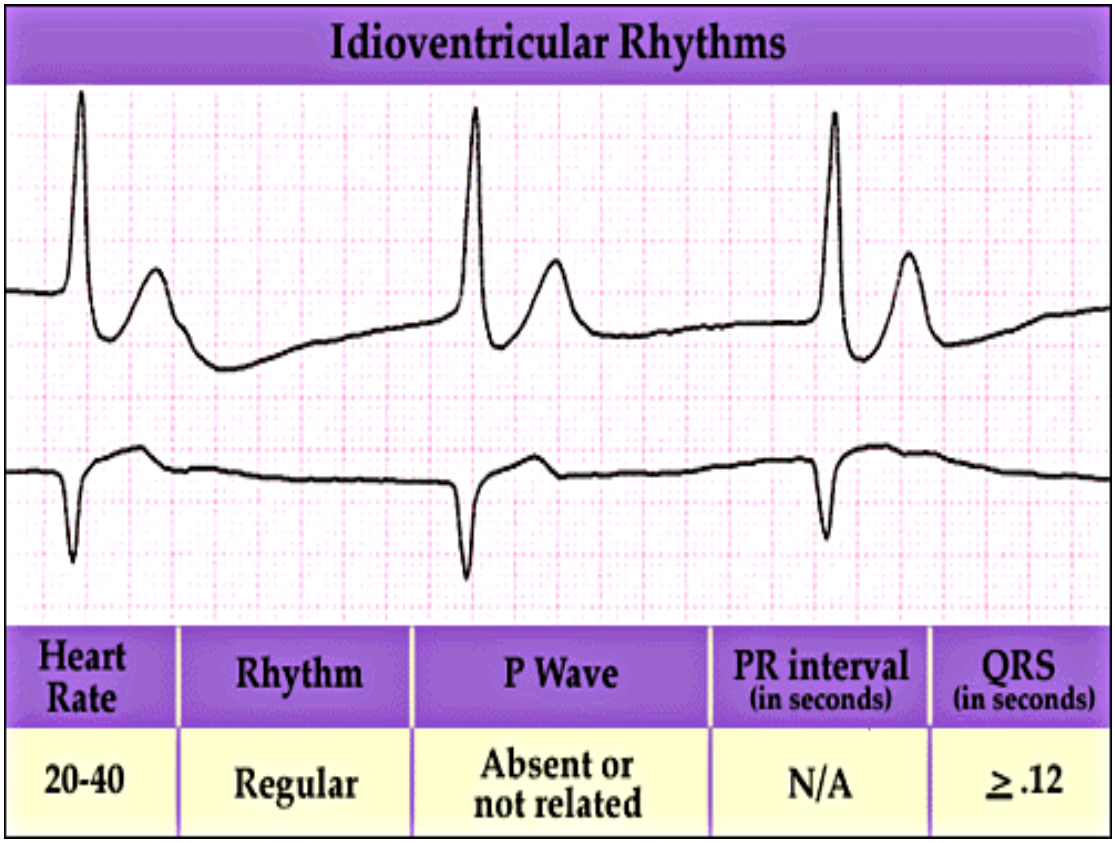
So, 3rd Degree AV Block



- *Deviation from NSR*
  - *The P waves are completely blocked in the AV junction; QRS complexes originate independently from below the junction.*
- *Etiology: There is complete block of conduction in the AV junction, so the atria and ventricles form impulses independently of each other. Without impulses from the atria, the ventricles own intrinsic pacemaker kicks in at around 30 - 45 beats/minute.*
- **Remember**

When an impulse originates in a ventricle, conduction through the ventricles will be inefficient and the QRS will be wide and bizarre.

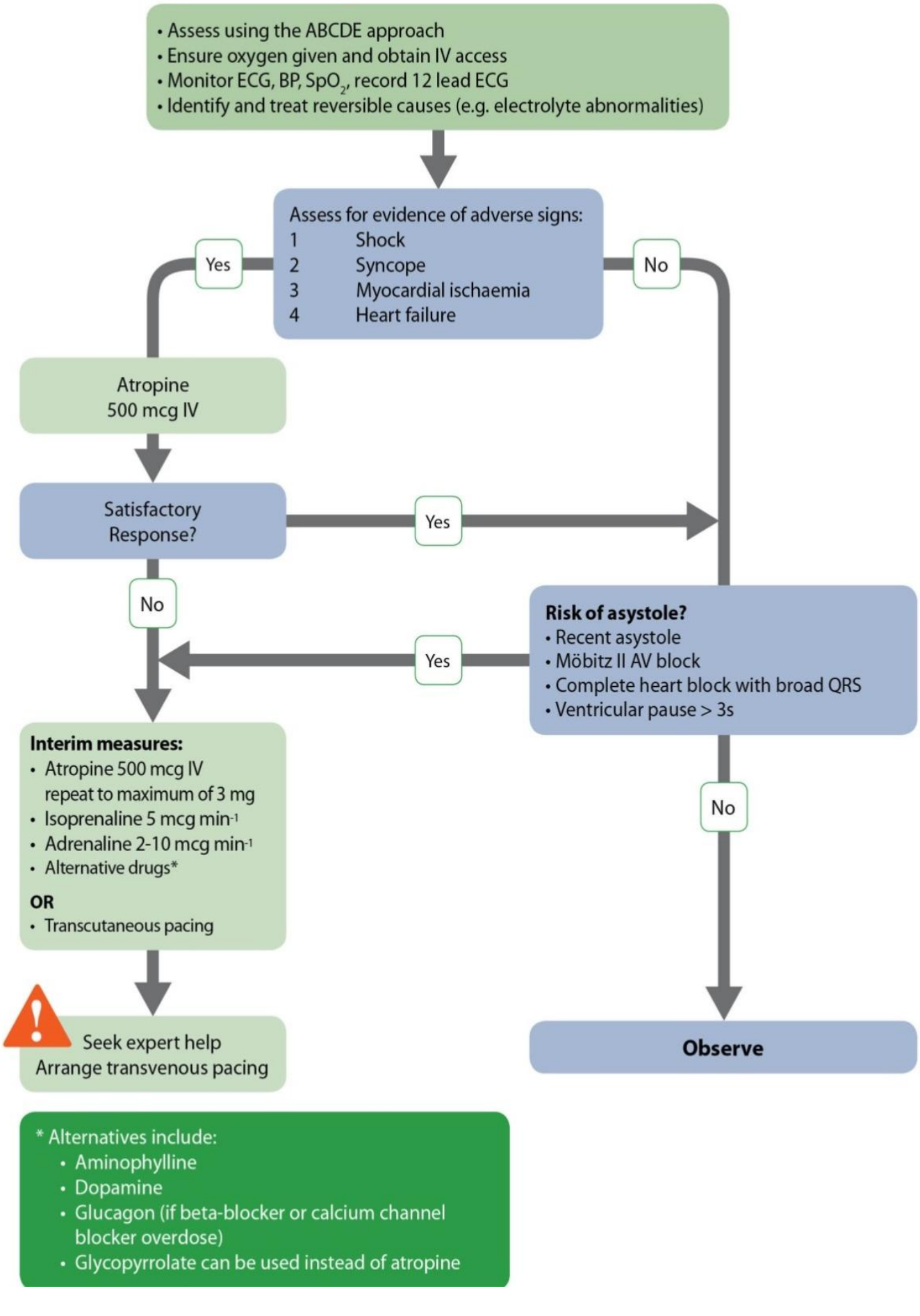




Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
20-40	Regular	Absent or not related	N/A	≥ .12

Bradycardia algorithm

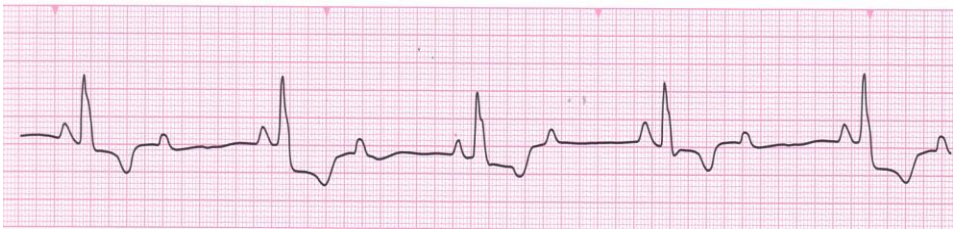
Includes rates inappropriately slow for haemodynamic state


• Assess using the ABCDE approach
• Ensure oxygen given and obtain IV access
• Monitor ECG, BP, SpO_2, record 12 lead ECG
• Identify and treat reversible causes (e.g. electrolyte abnormalities)
Assess for evidence of adverse signs:
1 Shock
2 Syncope
3 Myocardial ischaemia
4 Heart failure
Yes
No
Atropine
500 mcg IV
Satisfactory Response?
Yes
No
Risk of asystole?
• Recent asystole
• Möbitz II AV block
• Complete heart block with broad QRS
• Ventricular pause > 3s
Yes
No
Interim measures:
• Atropine 500 mcg IV repeat to maximum of 3 mg
• Isoprenaline 5 mcg min^{-1}
• Adrenaline 2-10 mcg min^{-1}
• Alternative drugs*
OR
• Transcutaneous pacing
Seek expert help
Arrange transvenous pacing
Observe
* Alternatives include:
• Aminophylline
• Dopamine
• Glucagon (if beta-blocker or calcium channel blocker overdose)
• Glycopyrrolate can be used instead of atropine

Managenent of brady cardia

Case study

- ***Clinical setting and history***
  - ***60-year-old man referred to admissions unit by GP***
  - ***Long-term history of heart disease***
  - ***Feeling light-headed and breathless***



Clinical course

- ***ABCDE***
  - ***A : Clear***
  - ***B : Spontaneous breathing, rate 18 min^{-1}***
  - ***C : Looks pale, P 45 min^{-1}, BP 90/50 mmHg, CRT 3 s***

Initial rhythm?

- ***D : Alert, glucose 4.5 mmol l^{-1}***
- ***E : Nil of note***

What action will you take for management

1- Atropine

Indication

- ***Symptomatic bradycardia***

Contraindication

- ***Do not give to patients who have had a cardiac transplant***

Dose

- ***500 mcg IV, repeated every 3 - 5 min to maximum of 3 mg***

Actions

- ***Blocks vagus nerve***
- ***Increases sinus rate***
- ***Increases atrioventricular conduction***

Side effects

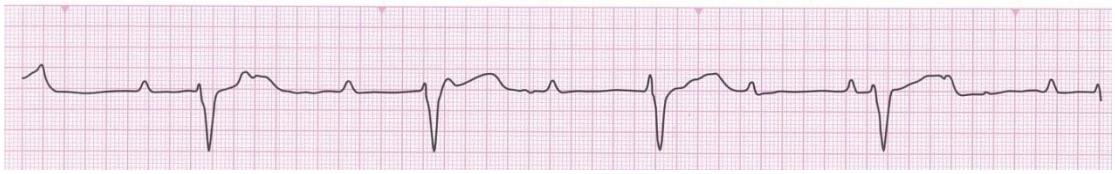
- ***Blurred vision, dry mouth, urinary retention***
- ***Confusion***

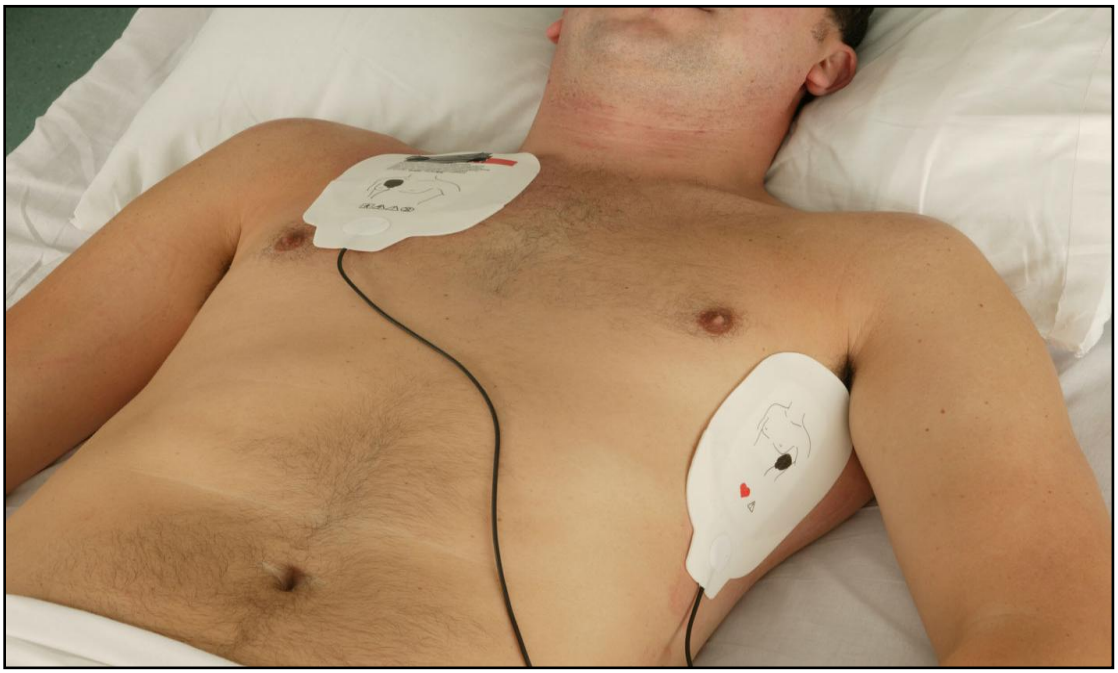
No response to atropine

- ***Patient becomes more breathless, cold, clammy and mildly confused***
- ***Change in rhythm***
- ***ABCDE***
  - ***A : Clear***
  - ***B : Spontaneous breathing, rate 24 min^{-1}***

    widespread crackles on auscultation
  - ***C : Looks pale, HR 35 min^{-1}, BP 80/50 mmHg, CRT 4 s***
  - ***D : Responding to verbal stimulation***
  - ***E : Nil of note***

What will you do now?





2- Pacing

NOT

NOT READY YET

3- Adrenaline

Infusion of 2-10 mcg min^{-1} titrated to response

OR 4- Isoprenaline infusion 5 mcg min^{-1} as starting dose

OR 5-Dopamine infusion 2-5 mcg kg^{-1} min^{-1}

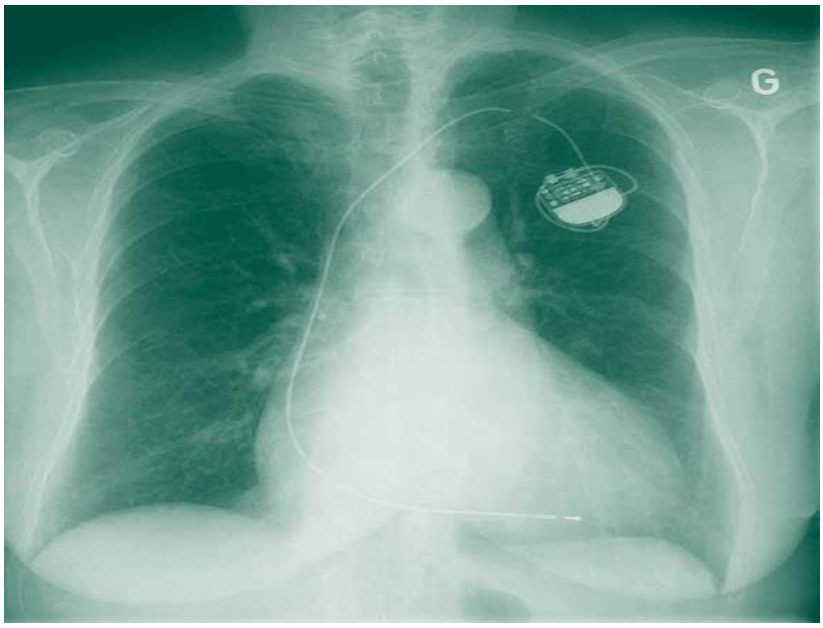
6-Consider need for expert help

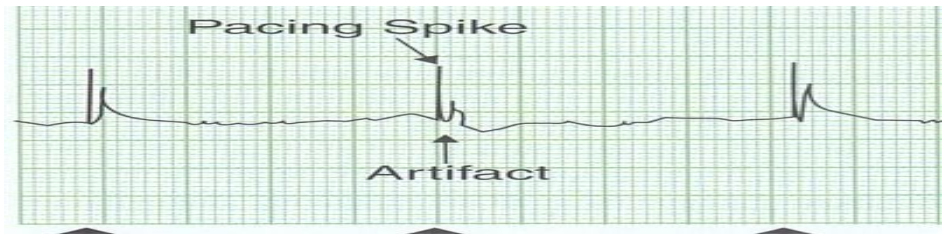
7-Prepare for transcutaneous pacing

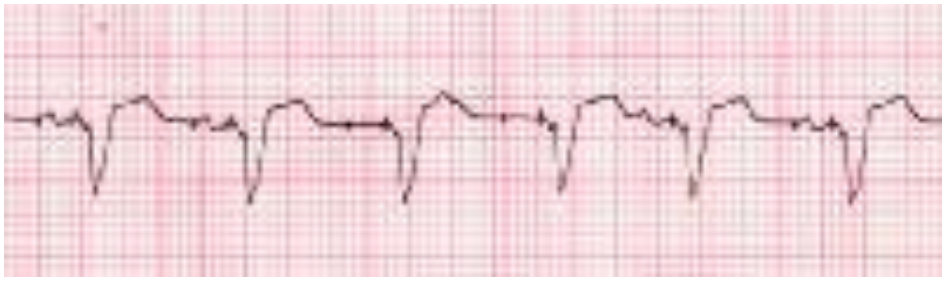
8- Consider percussion pacing as interim measure

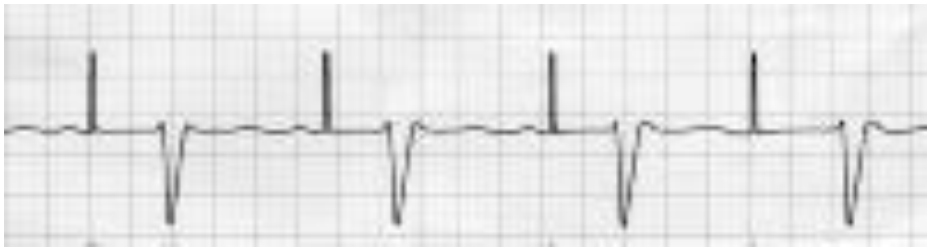
***9*-Confirm electrical capture and mechanical response once transcutaneous pacing has started**

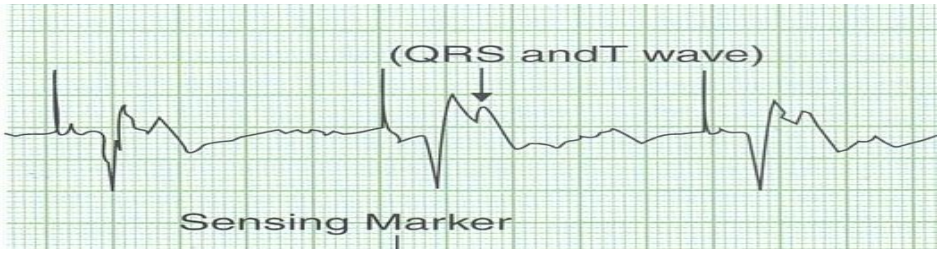
Pace maker










(QRS andT wave)
Sensing Marker