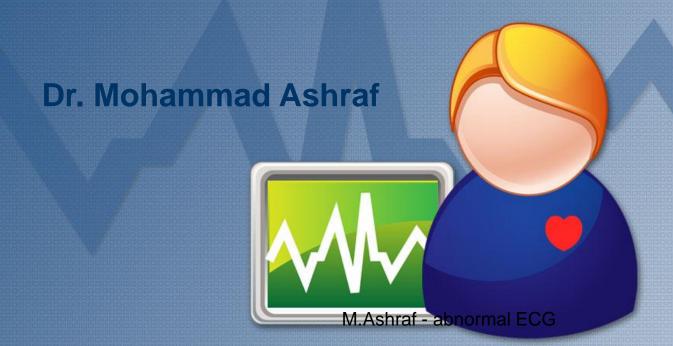


Heart function alterations Arrhythmia, abnormal ECG



Cardiovascular Physiology



Arrhythmias

- Arrhythmias are abnormalities of electrical rhythm that result from abnormal impulse formation, impulse conduction, or both.
- Clinically, it is important to:
 - Identify common arrhythmias and their clinical presentation.
 - Understand underlying pathophysiology.
 - Determine if/what treatment is necessary.

abnormal **ECG** abnormal abnormal rhythm conduction ectopic bundle abnormal sinus sinoatrial A-V nodal focus branch rhythm block block (heart block block) sinus sinus respiratory bradycar sinus tachycardi arrythmia 2nd ist degree 3rd degree degree active passive atrial flutter ventricular A-V nodal fibrillation paroxysmal atrial extrasystole rhythm tachycardia fibrillation M.Ashraf - abnormal ECG fppt.com

I. Abnormal sinus rhythm

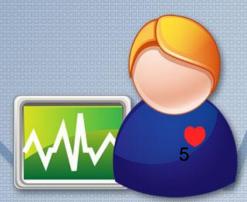


Sinus tachycardia

• The impulses arise from SAN but at a rate faster than 100 per min.

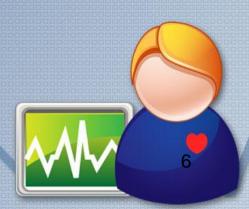
• causes:

- o Physiological: as in exercise, emotions.
- o Pathological: as fever, thyrotoxicosis & anemia.
- Patients may report heart palpitations.



• ECG: normal rapid waves.





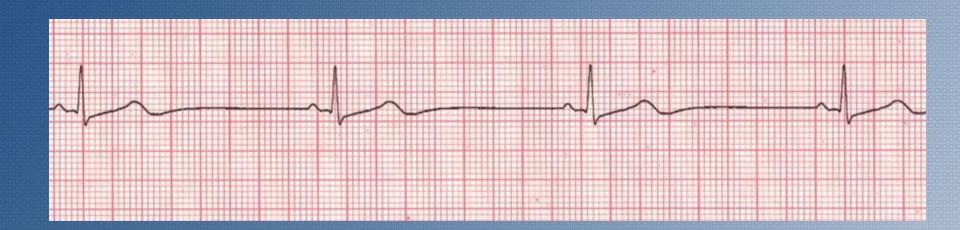
Sinus bradycardia

• The impulses arise from SAN but at a rate slower than 60 per min.

• causes:

- o Physiological: as in athletics, during sleep.
- Pathological: as hypothermia, myxoedema & ↑
 intracranial tension & sick sinus syndrome (often develop in elderly).
- \circ **Drugs**: use of β-blockers and nondihydropyridine calcium channel blockers.

• **ECG**: normal slow waves

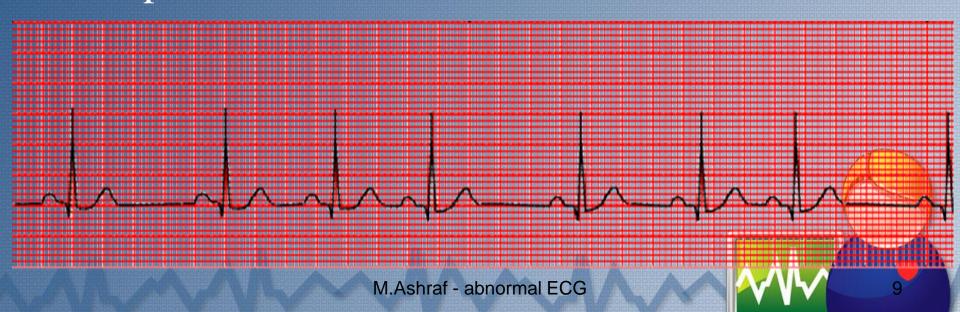


- Usually asymptomatic.
- Treatment:
 - If asymptomatic, no treatment is required.
 - o If bradycardia causes symptoms of low COP (eg, syncope, altered mental status), treat with anticholinergics (eg, atropine). Pacemaker insertion if condition is chronic.

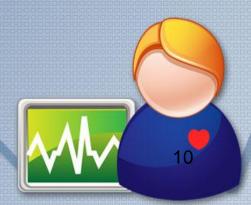
Respiratory sinus arrhythmia

- Normally, HR increases during inspiration and decreases during expiration.
- ECG: normal with increased HR during inspiration.

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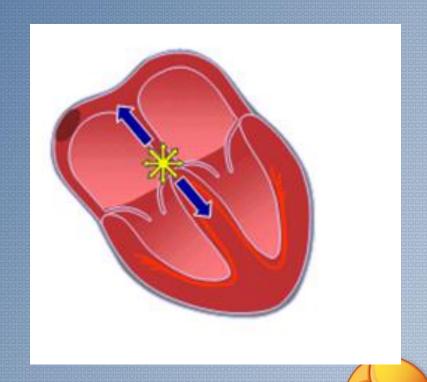


II. Ectopic rhythm



Passive ectopic A-V nodal rhythm

- When the SAN stops, the impulses will arise from the AVN.
- The rhythm is regular but slower.



• <u>ECG</u>:

- ✓ Inverted P wave (may be before or after QRS and may be absent).
- ✓ Shortened P-R interval.
- ✓ Normal QRS & T waves.





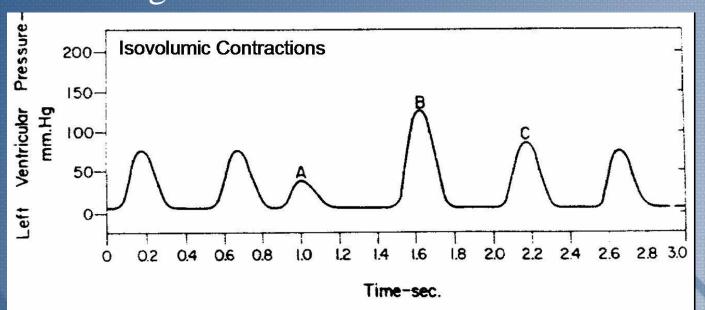
Active ectopic Extrasystole (premature beat)

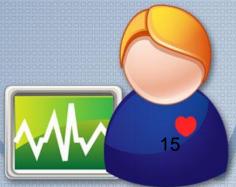
- This is a premature cardiac contraction produced by discharge from ectopic focus during diastole i.e. occurring before the next expected normal beat.
- As the next SA nodal impulse will fall during the absolute refractory period of the extrasystole, extrasystole is usually followed by a compensatory pause.

Usually asymptomatic but some patients report palpitations or feel a "skipped beat."



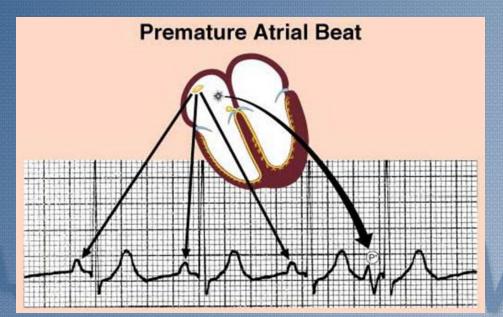
- The contraction produced by the extrasystole is weaker than normal contractions because the time available for ventricular filling is decreased.
- The contraction produced by the SAN after the compensatory pause is stronger than other contractions because the time available for ventricular filling is increased.

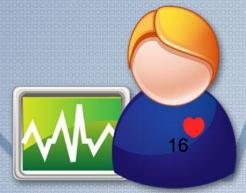




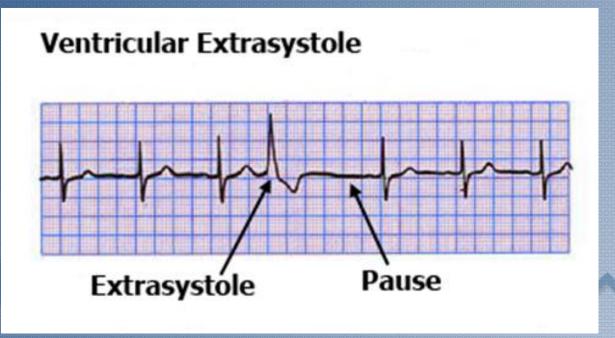
Types of extrasystole

- 1. Atrial extrasystole: (the ectopic focus is in the atrium)
 - ✓ The whole impulse is premature (short T-P line)
 - ✓ P wave is abnormal (inverted or notched).
 - ✓ QRS complex is normal.
 - ✓ Followed by *incomplete* compensatory pause.

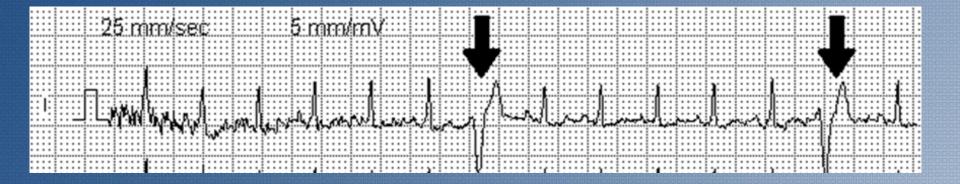


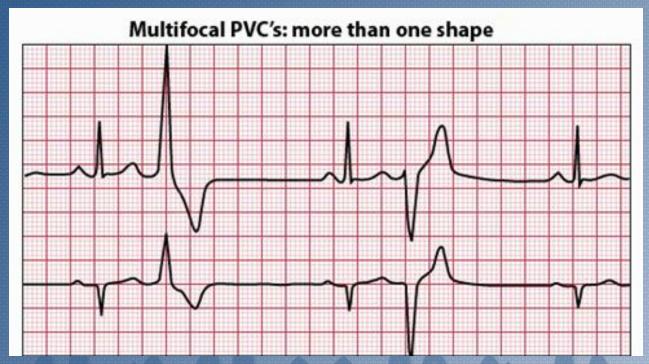


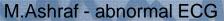
- 2. Ventricular extrasystole: (the ectopic focus is in the ventricle)
 - ✓ The whole impulse is premature (short T-P line)
 - ✓ P wave is absent.
 - ✓ QRS complex is abnormal shaped (bizarre shaped), prolonged duration.
 - ✓ Inverted T wave.
 - ✓ Followed by compensatory pause.

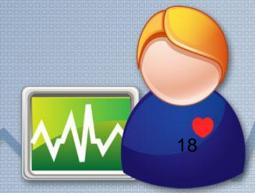






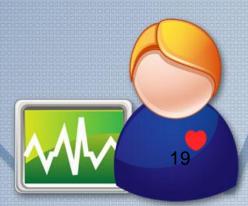






Treatment

- ⇒ Reassurance: PVCs alone are not life threatening and do not increase the risk of fatal arrhythmias in individuals without underlying heart disease.
- ⇒ Avoidance of triggers of ectopic foci (eg, caffeine, nicotine, Anxiety and lack of sleep)
- ⇒Electrolyte repletion may be helpful.

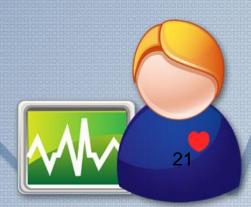


Paroxysmal tachycardia

⇒This condition is characterized by sudden attacks of increased HR (> 200 beats / min.) that last for seconds, minutes, hours or even a day then the attack end suddenly i.e. in paroxysms.

⇒Patients may report palpitations with/without precipitants (eg, stress, caffeine, alcohol).

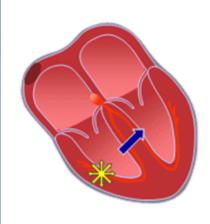
- ⇒Paroxysmal tachycardia may be:
- ✓ Supraventricular tachycardia.
 - o Atrial.
 - A-V nodal re-entrant tachycardia (AVNRT) (the most common)
- ✓ Ventricular tachycardia: serious condition as it indicates a considerable damage in the ventricles and may lead to fatal ventricular fibrillation.

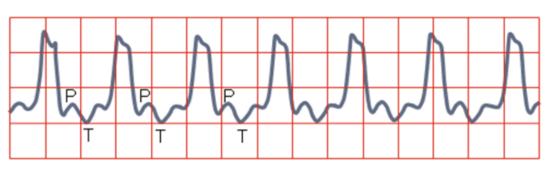


⇒ECG: as extrasystole but the waves are repeated all over the paroxysm. retrograde P waves (via a reentry circuit) may be inverted or hidden

VENTRICULAR TACHYCARDIA

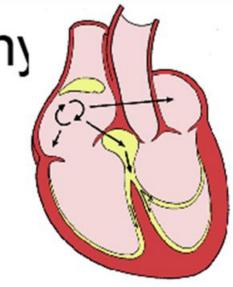
Impulses originate at ventricular pacemaker

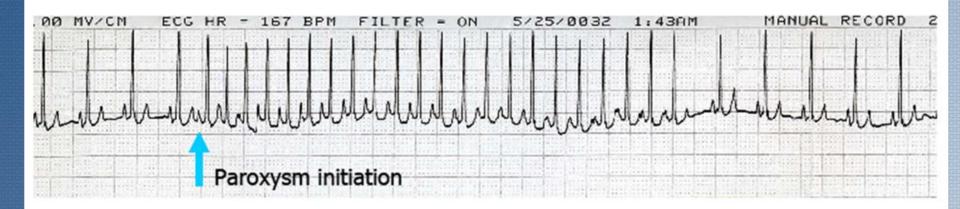




Wide ventricular complexes. Rate > 120/min

Supraventricular tachy



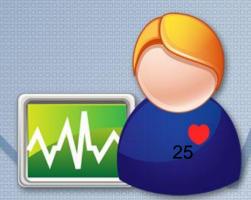


- *AKA **Paroxysmal** atrial tachycardia
- *Paroxysm = abrupt change

Treatment

- Paroxysmal Supraventricular tachycardia can be terminated by:
 - o pressure on the eyeball (oculo-cardiac reflex) or on the carotid sinus.
 - If that is not successful, IV adenosine (also slows AV node conduction) is a rapid therapy.
- For more long-term control: β-blockers, nondihydropyridine CCBs, ablation, and class IA or IC antiarrhythmics.

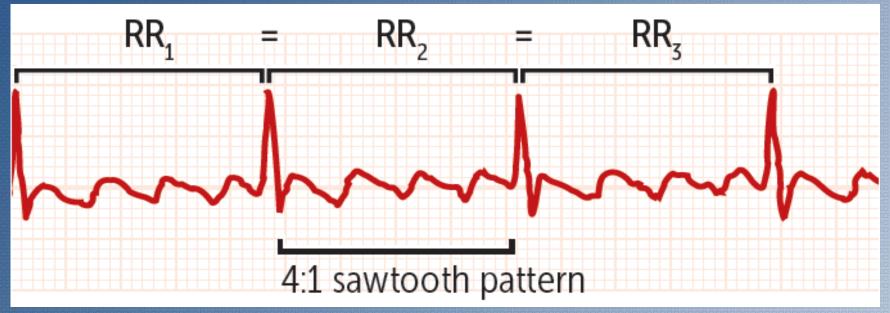
Atrial flutter & atrial fibrillation



	Atrial flutter	atrial fibrillation
Atrial rhythm	200 – 400 Regular Atrium contracts as one unit	400 – 600 Irregular No effective atrial contractions
Ventricular rhythm	Regular	Irregular irregularity
ECG	Multiple P waves (saw tooth appearance) Normal & regular QRS & T waves	Absent P waves or replaced by f waves. Normal but irregular QRS & T waves.
Pulse deficit	Absent	present

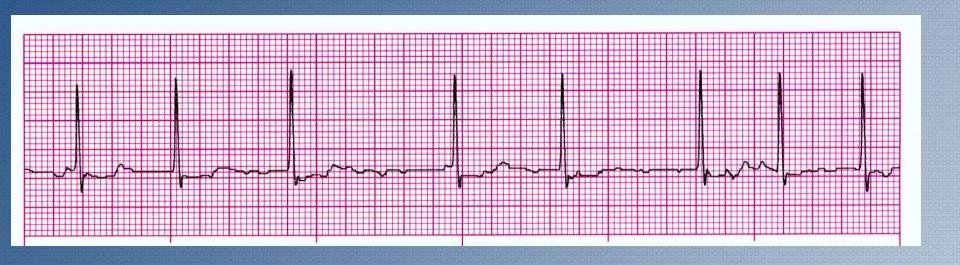
Flutter is a transient disease either return to sinus rhythm or converted into fibrillation.

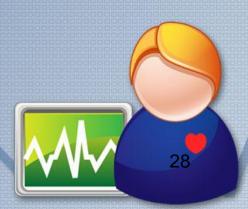
Atrial flutter





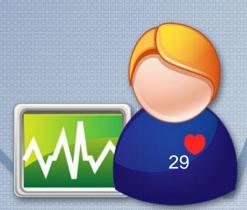
Atrial fibrillation

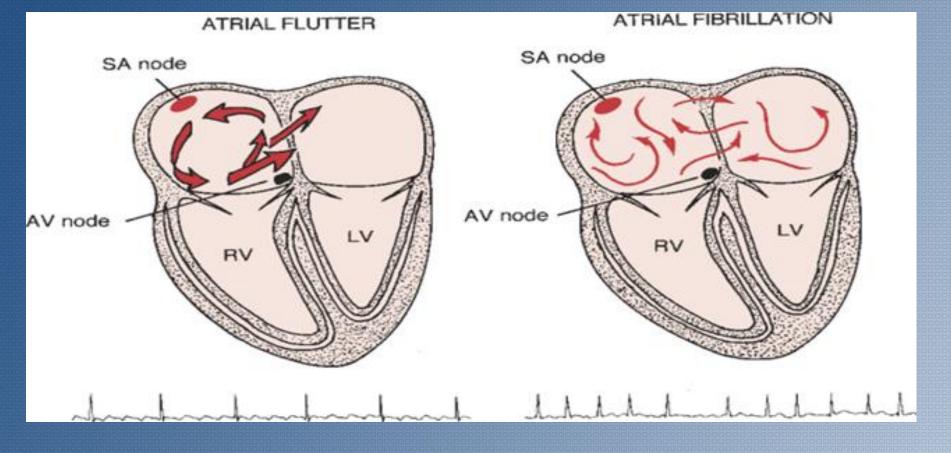




Most common risk factors of atrial fibrillation are:

- ⇒ Often associated with right or left atrial enlargement eg, from mitral stenosis.
- ⇒ Hypertension The most important risk factor in the United States.
- ⇒ Coronary artery disease.
- ⇒ Congestive heart failure.
- ⇒ Excessive alcohol intake.
- ⇒ Thyrotoxicosis.





These make the formation of multiple re-entry circuits more likely to occur due to:

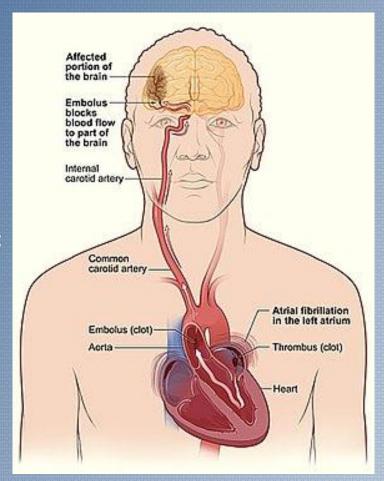
- i. Elongation of the pathway by atrial dilatation.
- ii. Delay in conduction by ischemia.
- iii.Shortening of absolute refractory period by drugs.

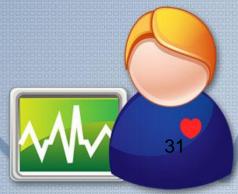
Problems of Atrial fibrillation:

- ⇒Impaired CO due to rapid ventricular rate.
- ⇒Can lead to thromboembolic events, particularly stroke.

• Treatment:

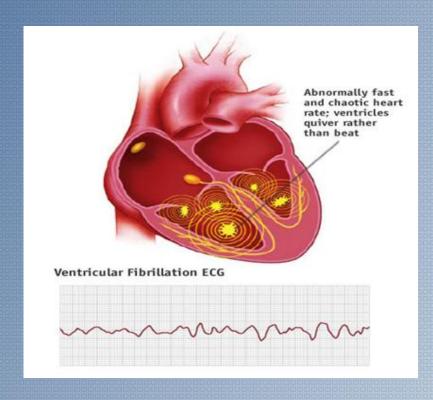
anticoagulation, rate control, rhythm control, and/or cardioversion. Definitive treatment is catheter ablation.

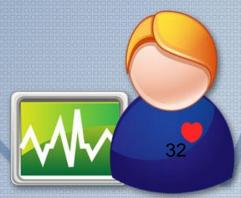




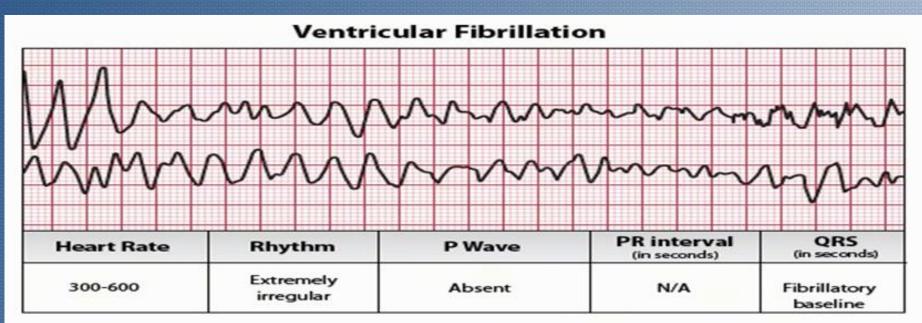
Ventricular fibrillation

- ⇒ Fatal if not treated promptly by immediate CPR and defibrillation.
- ⇒The ventricular muscle contract in irregular ineffective way.
- ⇒ Mostly, VF develops from untreated sustained VT in patients with underlying heart disease.
- ⇒ In a minority of cases, idiopathic VF can destrato abnormal ECG





⇒**ECG**: A completely irregular inconstant rhythm with no identifiable waves.



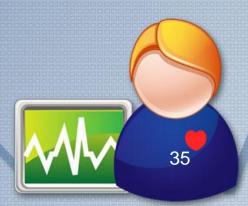
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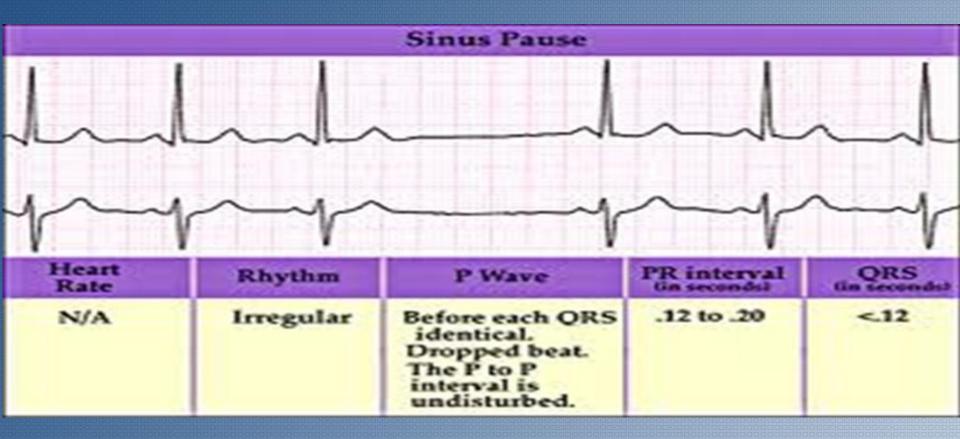
Abnormal conduction

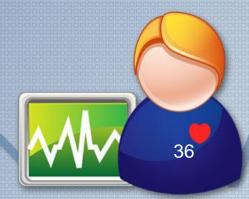


Sinoatrial block

- The impulse fails to escape from the SAN.
- The whole beat is missed.
- ➤ If the condition is permanent the heart picks up a new rhythm from the AVN.
- If the condition is temporary as in sick sinus syndrome the sinus rhythm returns again.





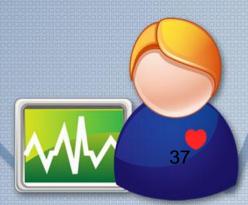


A-V nodal block

⇒A delay or failure in conduction through the AV node.

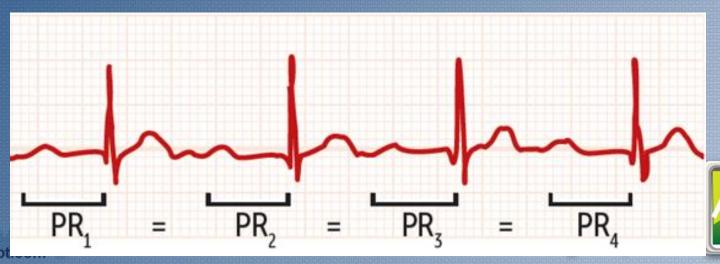
⇒has three types (or degrees):

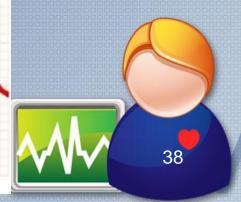
- ≥1st degree heart block.
- ≥2nd degree AV block.
- ≥3rd degree (complete) AV block



1st degree AV block

- ⇒ There is only prolongation of the P-R interval (above 0.20 second) but still all impulses from the SAN reach the ventricles.
- ⇒ Benign and asymptomatic. No treatment required except that of underlying cause to prevent progression to more advanced degrees.

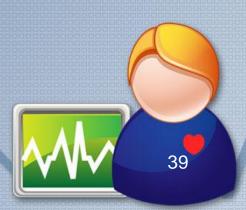




Causes of AV block

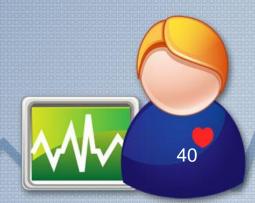
Reversible transient causes:

- High vagal tone.
- Transient ischemia.
- Drugs: β-blockers, nondihydropyridine CCBs, antiarrhythmics.
- Lyme disease.
- Commonly due to myocarditis.



Permanent structural cause:

- Myocardial infarction.
- Amyloidosis and sarcoidosis.
- Aging due to degeneration of the AV node conduction pathways.



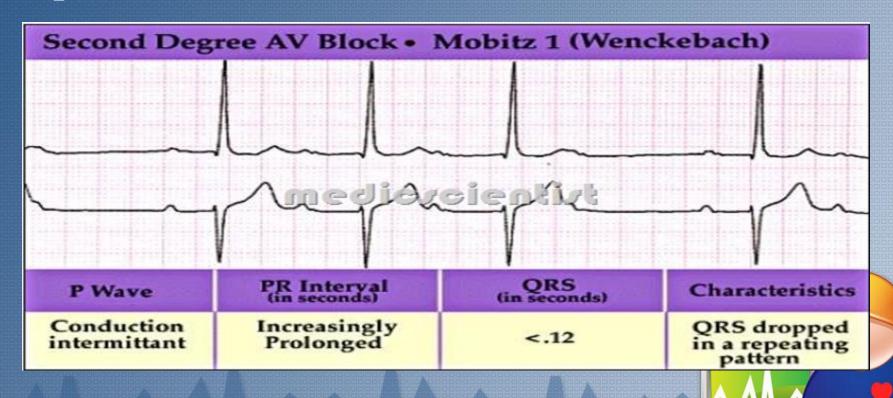
2nd degree heart block

- ⇒Intermittent failure of AV conduction that results in a P wave not followed by QRS complex.
- ⇒subdivided into Mobitz type I & Mobitz type II:

a) Mobitz type I "Wenckebach's phenomenon"

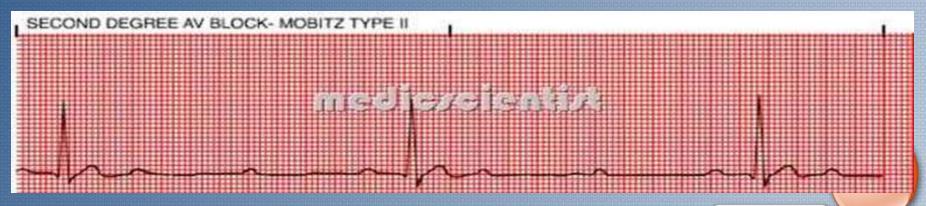
⇒<u>ECG</u>: there is progressive lengthening of the P-R interval for several successive beats until a P wave not followed by QRS complex.

- ⇒ Variable RR interval with a pattern (regularly irregular).
- ⇒Usually asymptomatic but may Needs pacemaker insertion.



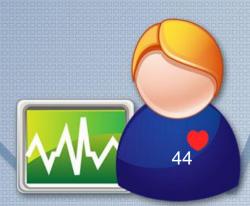
Mobitz type II:

- ⇒ ECG: the atrial rate is normal & regular. Every 2, 3 or 4 consecutive P waves are followed by one QRS complex (2:1 or 3:1 or 4:1 rhythm)
- ⇒ May progress to 3rd-degree block without warning.
- ⇒ Often treated with pacemaker even if asymptomatic.

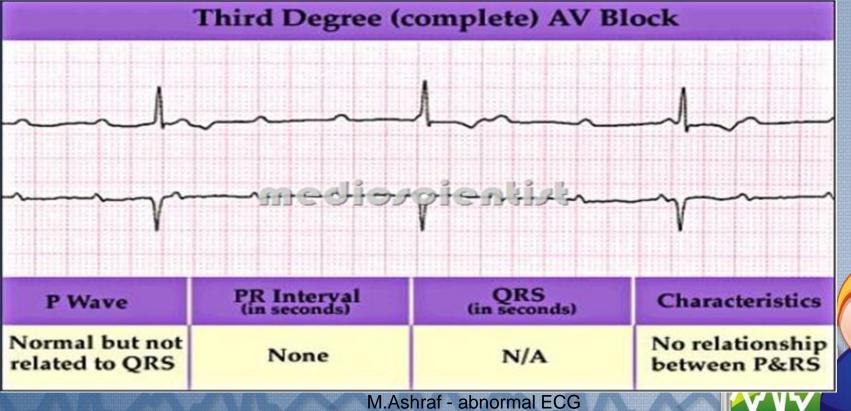


3rd degree heart block

- ⇒Complete block of AV nodal conduction → no impulses pass from atria to ventricles.
- ⇒The atria will beat according to sinus rhythm (70 b / min) while the ventricles beat according to idioventricular rhythm (25-40 b / min).



ECG: complete dissociation between P-wave and QRS complexes. But the P waves alone are regular = 70 / min and the QRS complexes are regular = $25 / \min$

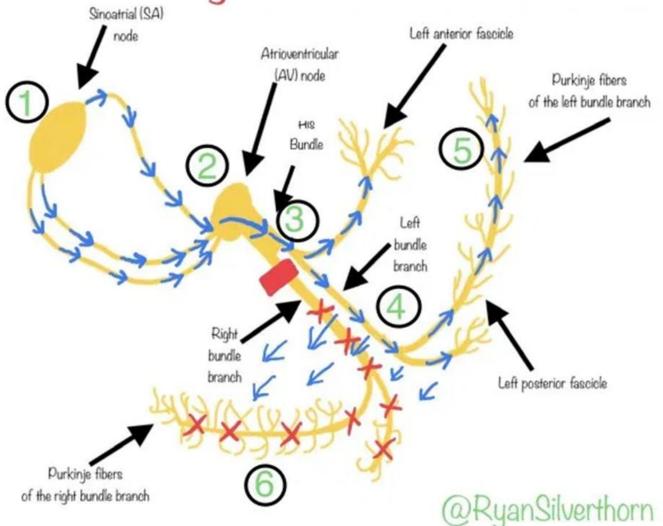


Bundle Branch Block (BBB)

- ⇒It may be right or left bundle branch block.
- ⇒The impulse passes normally in the intact bundle and then reaches the other ventricle through the ventricular muscle.

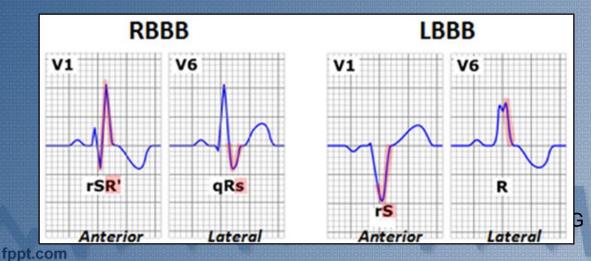


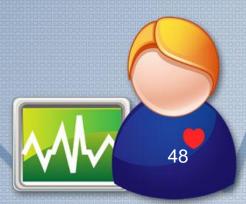
Right Bundle Branch Block

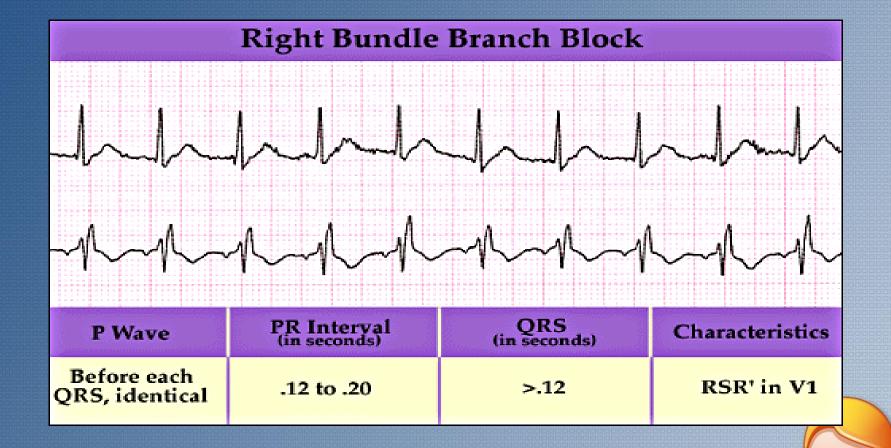


\Rightarrow **ECG**:

- ✓ HR is normal.
- ✓ P wave is normal.
- ✓ QRS is widened with:
 - ⇒In Rt BBB it is M-shaped RSR' complex ("bunny ears") in V1.
 - ⇒In Left BBB: Prominent S wave (no R wave) in V1. Wide and notched R wave in V6.
- ✓ Inverted T wave.







Classification of arrhythmias according to heart rate

A. Arrhythmias with heart rate less than 60 b/m (bradyarrhythmias):

sinus bradycardia, 2nd or 3rd degree AV block,

B. Arrhythmias with heart rate more than 100 b/m (tachyarrhythmias):

sinus tachycardia, paroxysmal tachycardia, atrial flutter and atrial fibrillation.

C. Arrhythmias with normal heart rate: extrasystole, 1st degree AV block, BBB & respiratory sinus arrythmia.

ECG changes in myocardial lesions

After few hours: elevation of ST segment in leads overlying the infarction & ST depression in leads of the opposite side of the heart.

After few days:

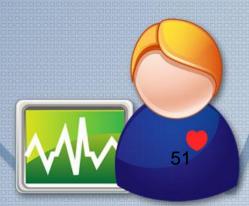
- ⇒ ST segment still elevated.
- ⇒ T wave becomes inverted.
- ⇒ Appearance of deep Q (pathological Q).

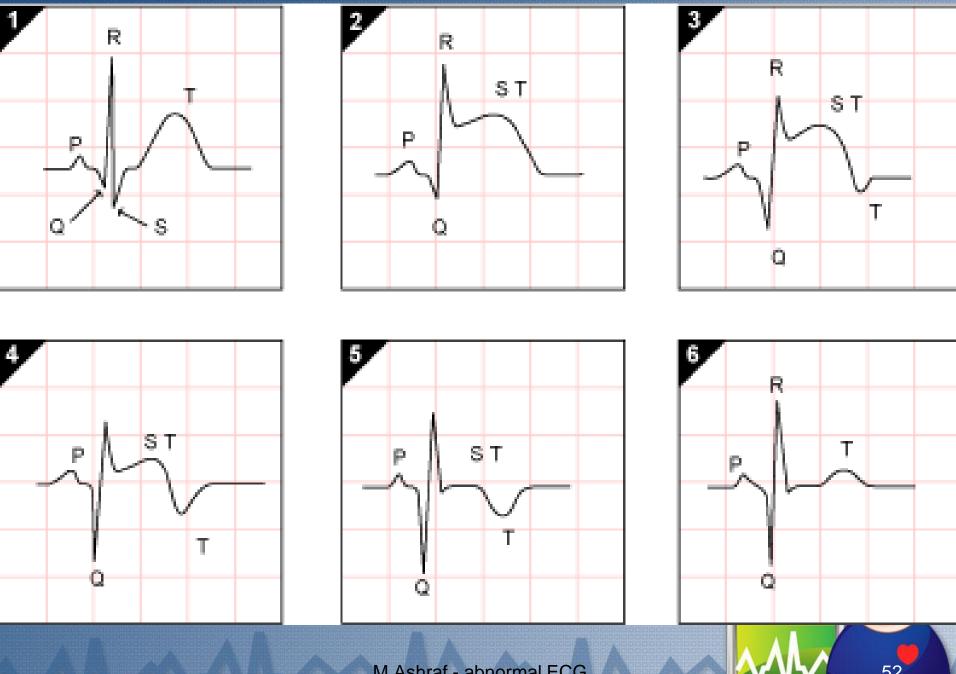
After few weeks:

- ⇒ ST segment returns to normal.
- ⇒ T wave and Q wave changes persist.

After few months:

- \Rightarrow T wave \rightarrow returns to normal.
- ⇒ Deep Q persists.







Last slide It is over

Any questions?

