Pathophysiology-Arrhythmia

Faculty of Pharmaceutical Sciences

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- If the problem was from SA node is sinus arrhythmia

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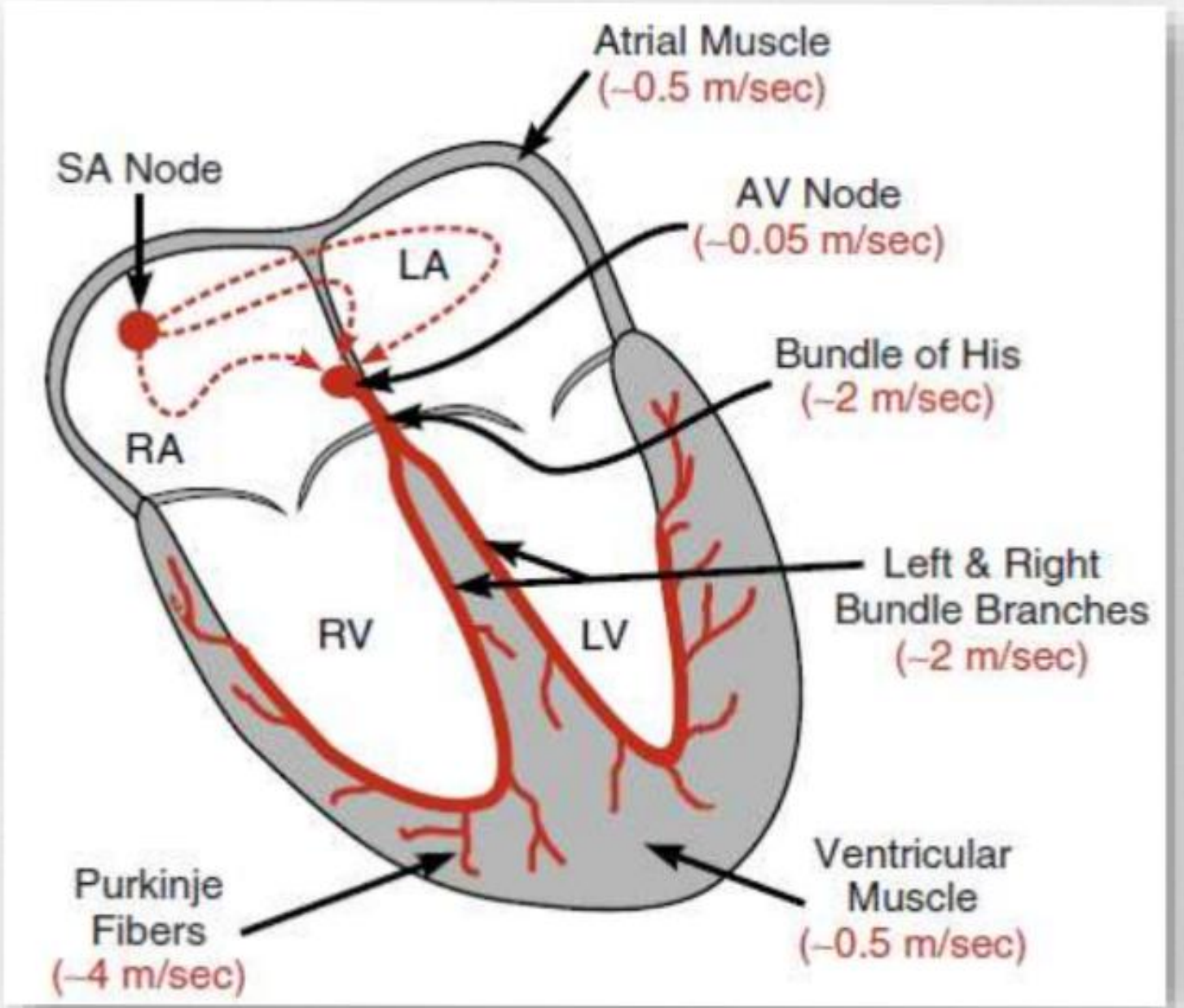
-if the arrhythmias originate from AV node which is the junction between atria and ventricles, it is called atrial arrhythmias.

- if the arrhythmias originate from the ventricle, it is ventricular arrhythmias.

- any arrhythmias originate above the ventricles, it is called supraventricular arrhythmia SVA as sinus and atrial arrhythmias

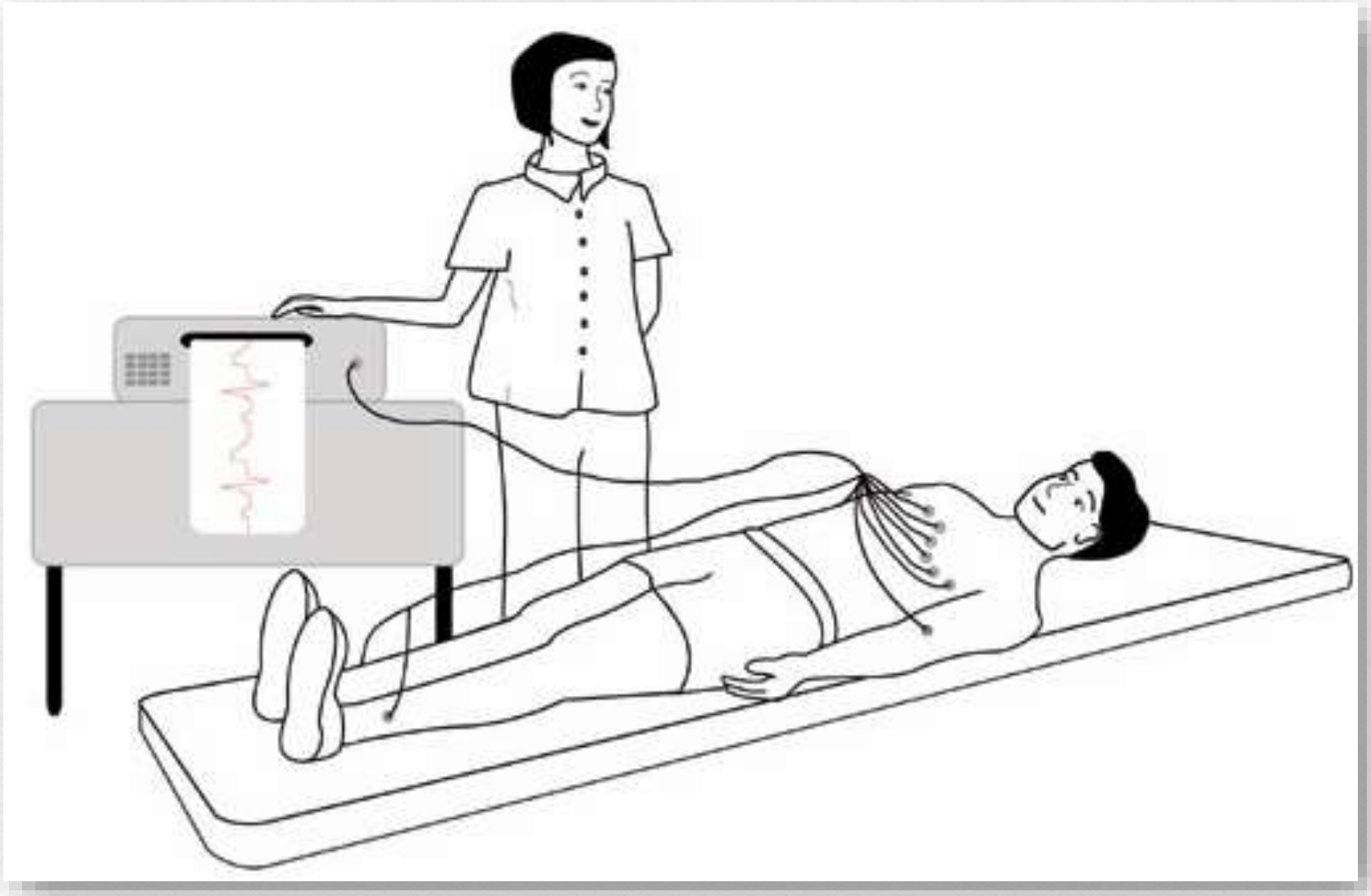


- Abnormal rhythm on ECG=Arrythmia.

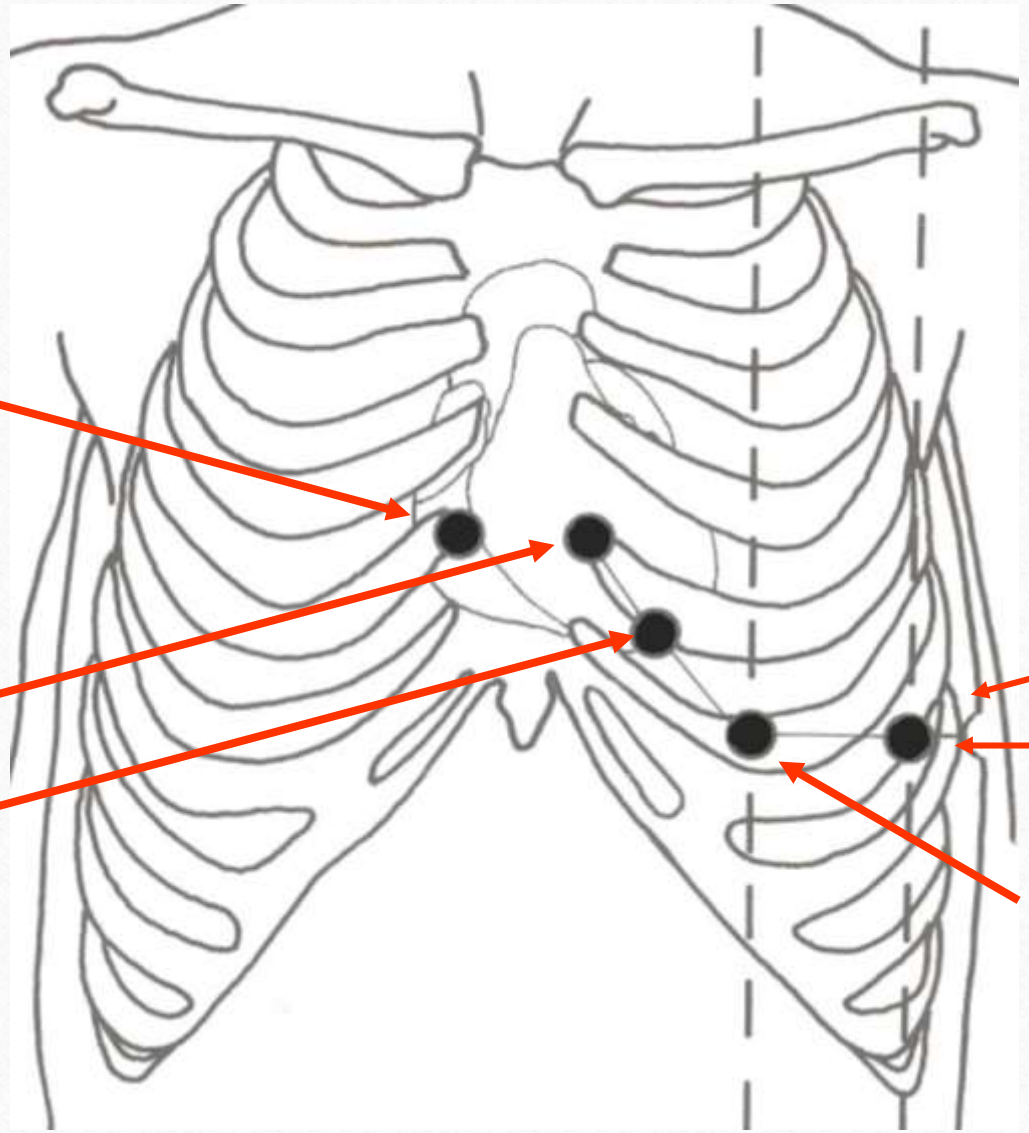
- Normal HR=60-100, if the action potential was generated. by SA node. While if the action potential was generated by other than SA node, HR will be less than 60 (Bradycardia) as all the heart components are conductive.

- Bradycardia and/or tachycardia can be physiologically normal or pathophysiological abnormal.

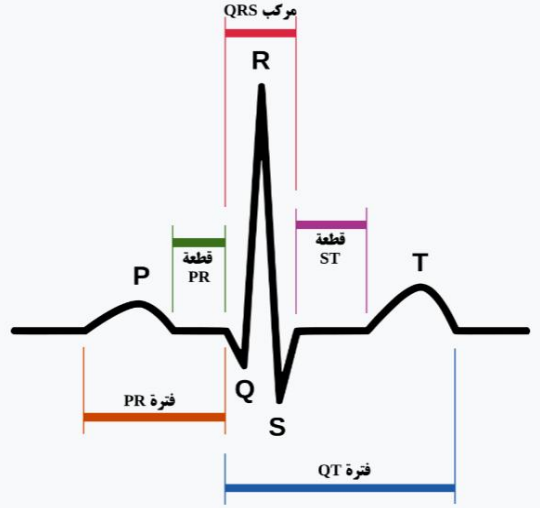
- Heart rate should be from 60-100 bpm.
If HR less than 60 is bradyarrhythmia, if it is more than 100 is tachyarrhythmias (from 100-150 simple tachyarrhythmias, 150-250 paroxysmal as suddenly starts and ends, 250-350 is called atrial flutter, higher than 350 is fibrillation. If it is in the atria, it is atrial fibrillation but, in the ventricles, it is ventricular fibrillation. If HR is between 40-60 is mild bradyarrhythmia, from 40-20 is moderate bradyarrhythmia, from 20-0 is severe bradyarrhythmia.



Placement of Precordial Electrodes



normal ECG



تخطيط كهربائية القلب في النظم الجيبي الطبيعي.

if the time is low from the normal → tachycardia and if the time high from the normal time → bradycardia

Interval	Average time (sec)	Range time (sec)	Events in the heart during interval
P wave			Atrial depolarization
PR interval (from p-wave to R)	0.18	0.12 to 0.2	Atrial depolarization and conduction through AV node
QRS duration	0.08	To 0.10	Ventricular depolarization and atrial re-polarization
QT interval	0.40	To 0.43	Ventricular depolarization
ST interval(QT minus QRS)	0.32		Ventricular re-polarization

Heart blocks: arrhythmia occur in AV node → blocking somewhere → blocking in conductive pathway → conductive pathways not running in normal level.

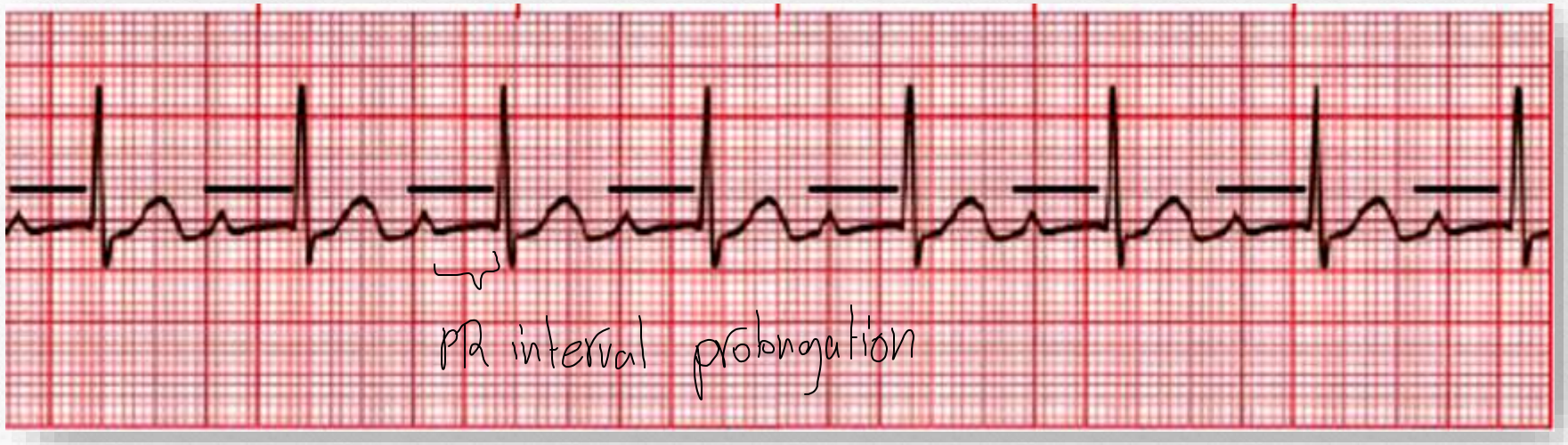
I. Block at the level of AV node: (bradycardia)

↳ give the patient parasympathatic antagonist, sympathatic agonist.

block in certain stage in ECG

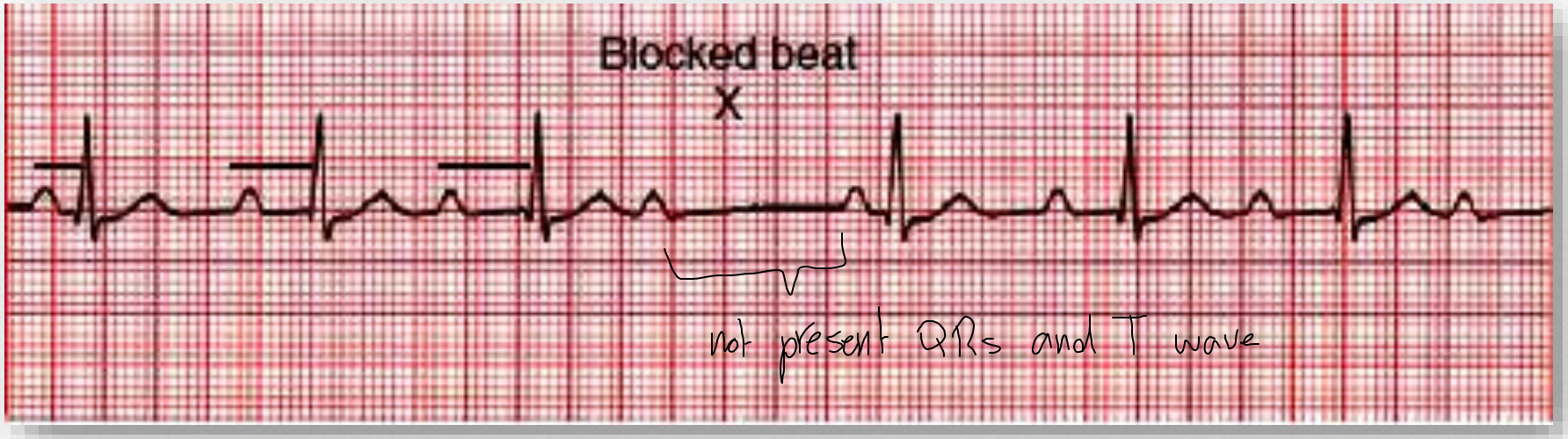
A. First degree heart block: every atrial depolarization is followed by conduction to ventricle but delay. ECG changes prolongation of PR interval to more than 0.22 second.

or prolongation of RR

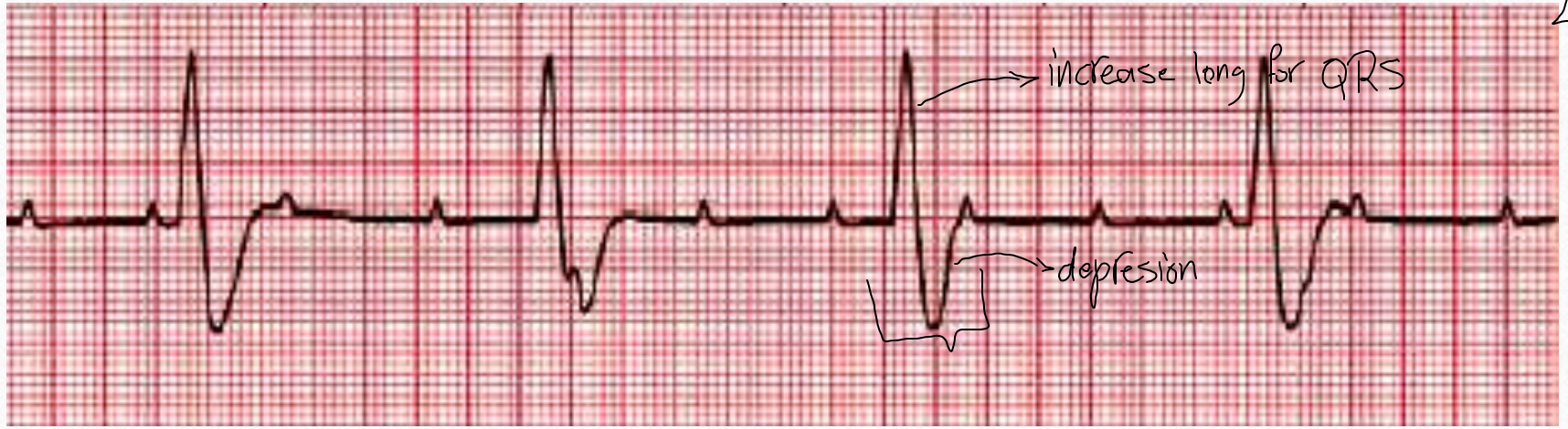


B. Second degree heart block: some P waves conducted but other not. ECG changes every second or third P wave conducted to the ventricles.

atrium or P wave not conducted to the ventricle



C. Third degree heart block (complete heart block):



→ the atrium works by itself and the ventricle works by itself

Rate: Atrial: 60–100 bpm; ventricular: 40–60 bpm
Rhythm: Usually regular, but atria and ventricles act independently. It occurs when all atrial activity fails to conduct to the ventricle so the Bundle of His will be responsible form generation of impulses. into ventricle.

SA node generation action potential for atrium.

→ ischemia , beta blocker (treatment for tachyarrhythmia) → (high doses)

Caused by: 1 Acute myocardial infarction, 2 calcify aortic stenosis, 3 cardiomyopathy, 4 drugs (digoxin), 5 increase of potassium.

↳ hyperpolarization

↳ treatment for tachyarrhythmia (activation for parasympathetic system)
↳ high doses

Block below AV node: A. block at Bundle of His, B. Block at the branches (Right or Left branch).

(خارج تدخل فيه)

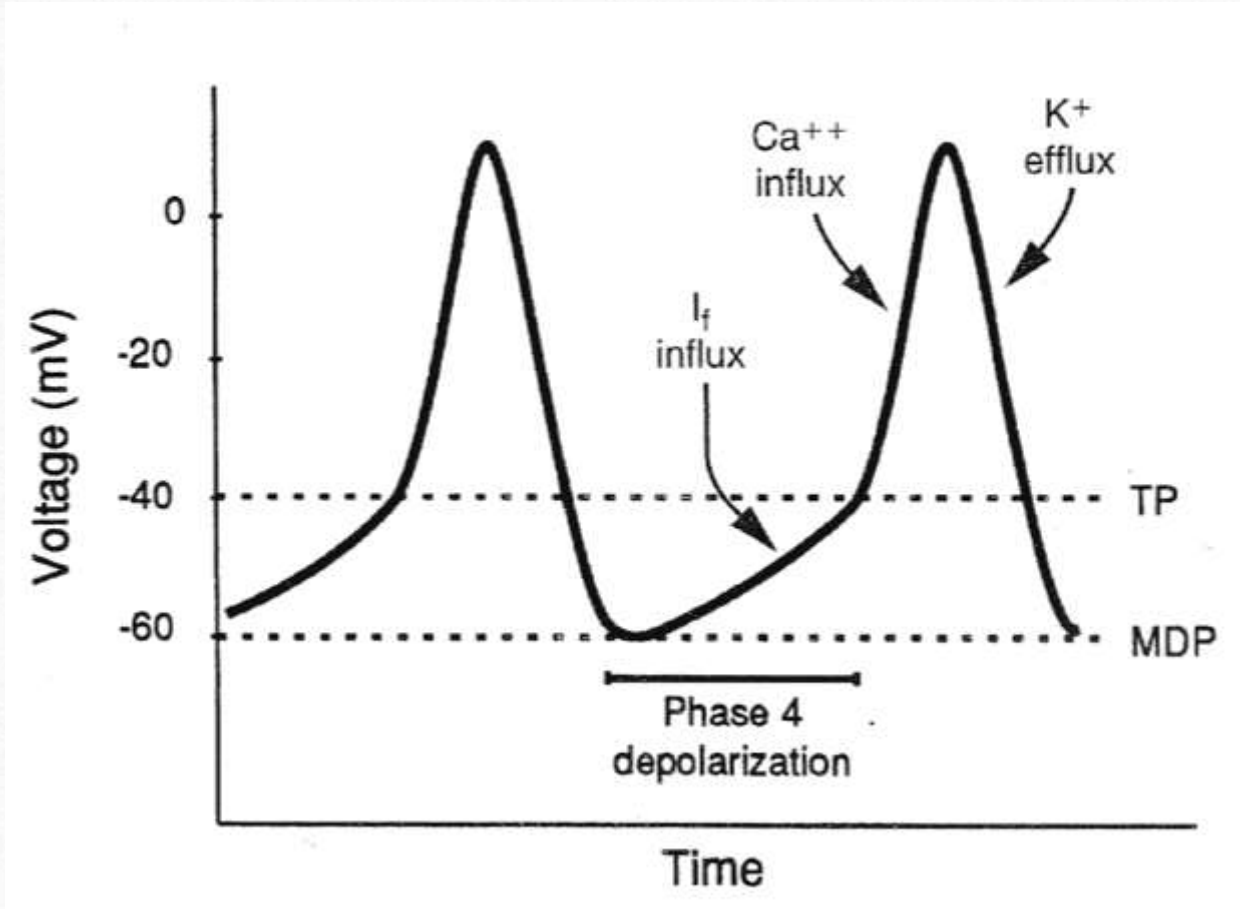
❑ Sinus rhythm:

It is caused by the changes of number of impulses emitted form SA node. Heart rates more than 100/min is called (tachycardia), while less than 60/min is called (bradycardia).

It is usually of two types:

- sinus bradycardia
- sinus tachycardia

Normal Pacemaker Activity in the SA Node



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1. Sinus bradycardia:

- Abnormally slow heart rhythm.
- May result from:

  - Abnormal impulse formation (Sinus bradycardia).
  - Abnormal conduction of impulses (AV conduction block).

1. Sinus bradycardia:

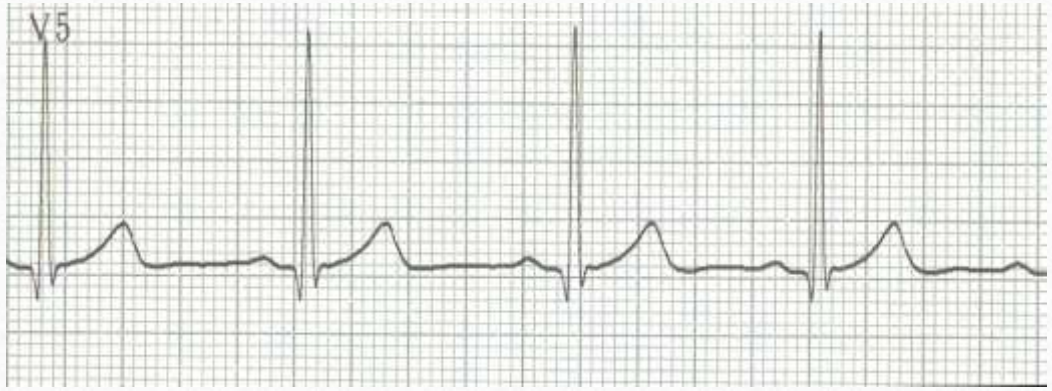
- Sinus node disorders.

- Sinus node (and AV Node) Function is profoundly influenced by autonomic nervous system tone.

  - Increase in sympathetic tone or circulating catecholamines increases sinus rate. direct

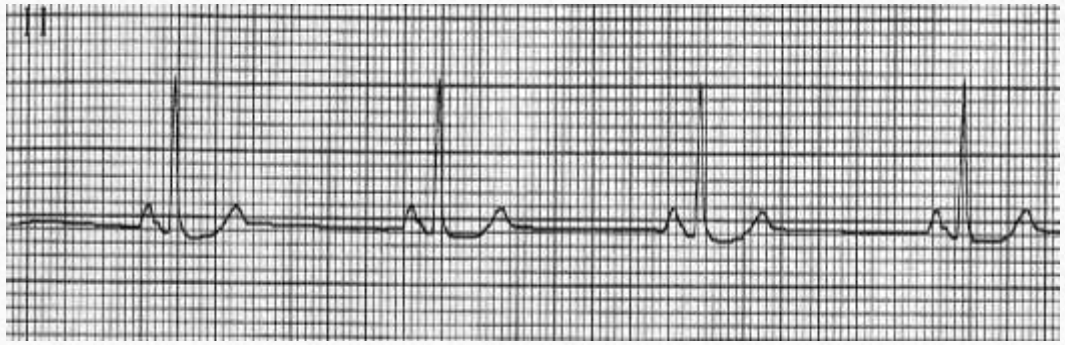
  - Increase in parasympathetic tone slows sinus rate. inverse

Normal Sinus Rhythm: 60 – 100 beats per minute (bpm)



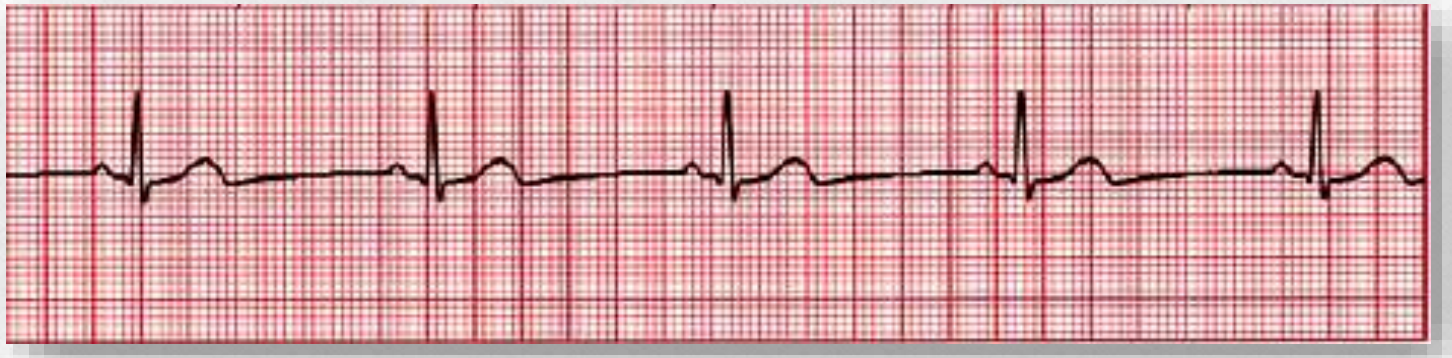
Sinus cycle length=920 ms
Heart rate = 65 bpm

Sinus Bradycardia: < 60 bpm



Sinus cycle length=1400 ms
Heart rate = 43 bpm

1. Sinus bradycardia:



Causes:

low tempreture (normal, abnormal) ← low thyroid hormone (normal, abnormal) →

A. Extrinsic causes: hypothermia, hypothyroidism, and raised intra cranial pressure, drugs (beta-blockers, digitalis, and anti-arrhythmic drugs).

← يستجيب الجسم لهذه الزيادة بإنقاص ضغط الدم bradycardia لذلك يصير عنا

→ tachyarrhythmia

B. Intrinsic causes: acute ischemia, infarction of SA node.

ECG changes: Prolonged R-R interval.

2. Sinus tachycardia

- ✓ **Abnormally rapid heart rhythm.**

- ✓ **May result from:**
  - **Abnormal impulse formation :** three causes?
    1. **Automaticity.**
    2. **Triggered activity.**
  - **Abnormal impulse conduction:**
    3. **Re-entry.**

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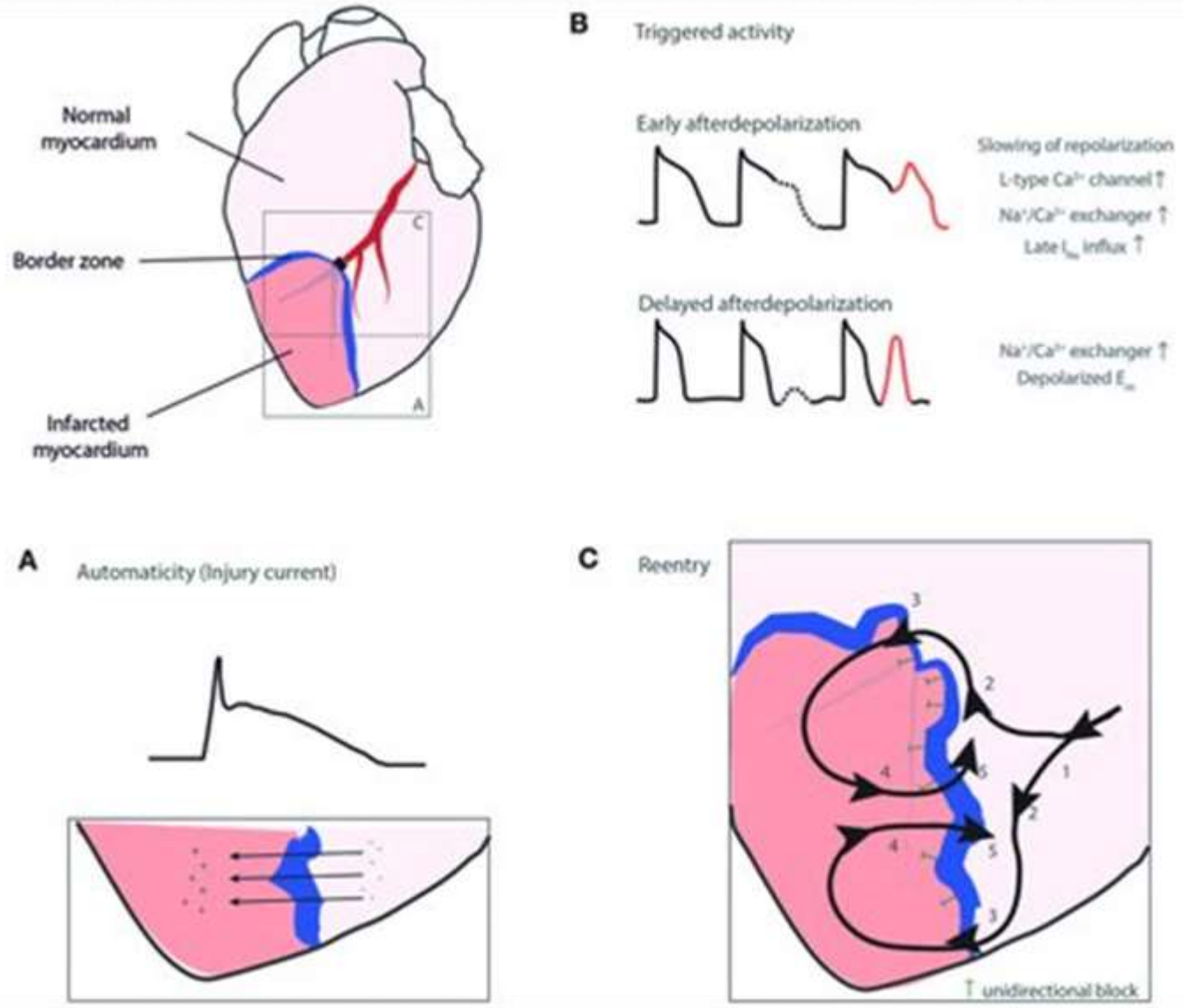
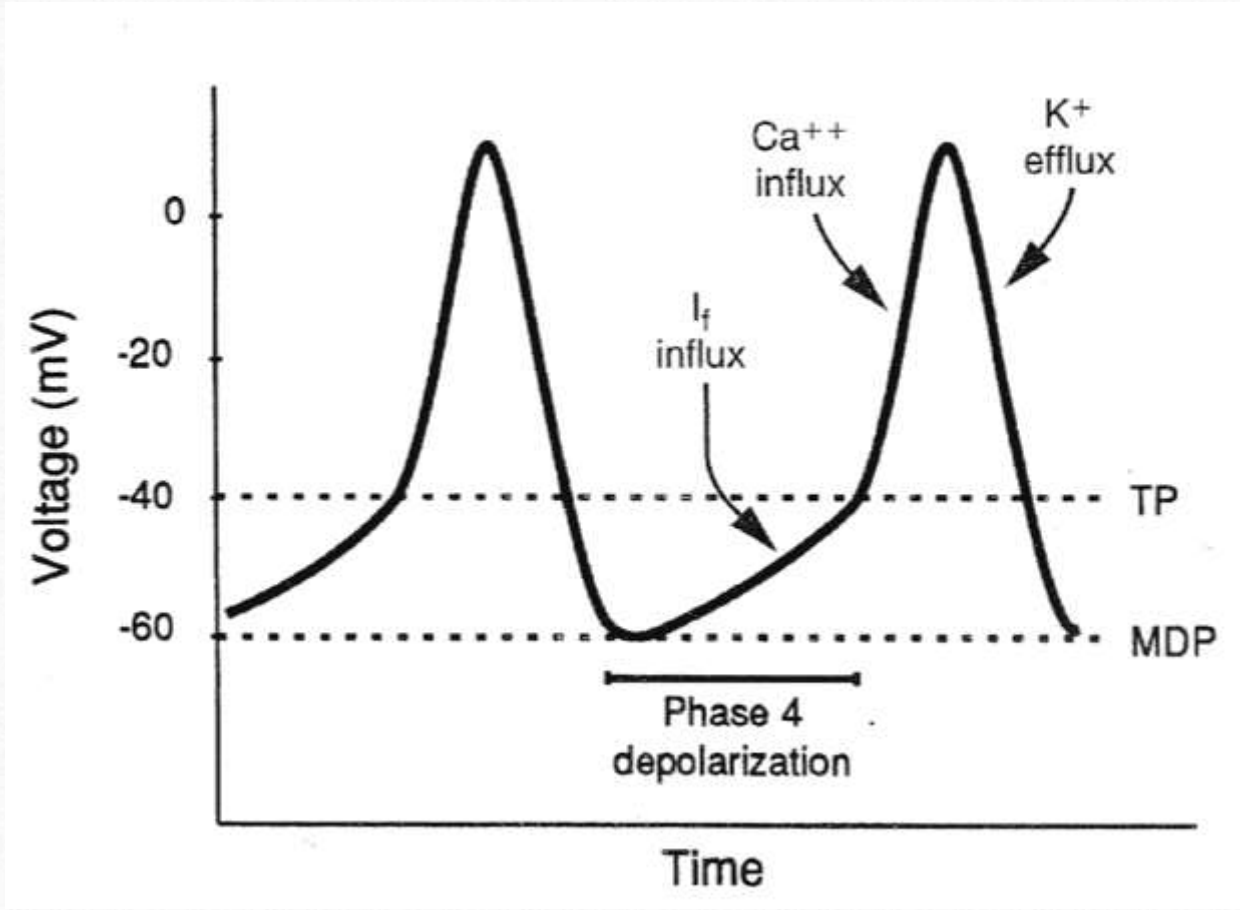


https://www.researchgate.net/publication/337023955_Ventricular_Arrhythmias_in_First_Acute_Myocardial_Infarction_Epidemiology_Mechanisms_and_Interventions_in_Large_Animal_Models/figures?lo=1

Normal Pacemaker Activity in the SA Node



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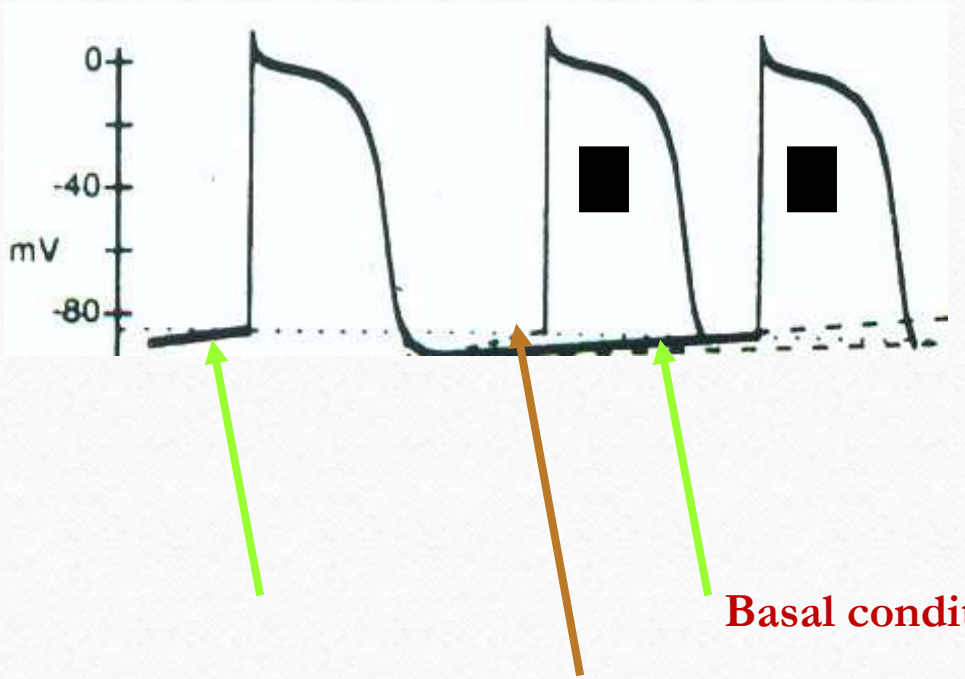
2. Sinus tachycardia

Enhanced Normal Automaticity



Increased slope of phase 4 depolarization

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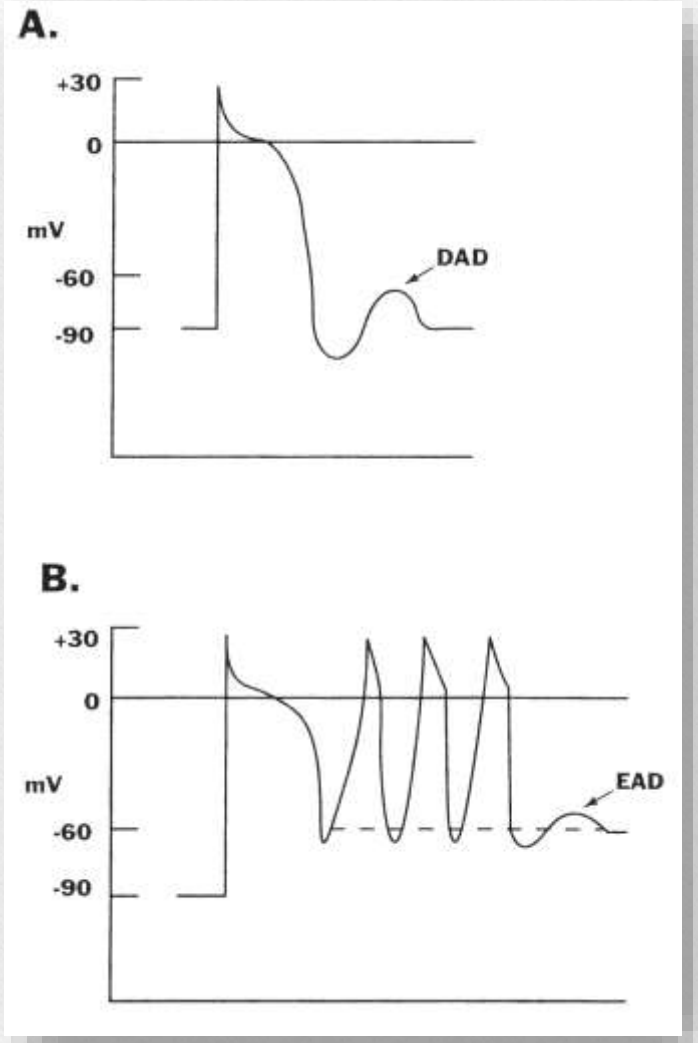
2. Sinus tachycardia

Triggered activity



Delayed Afterdepolarization: (typical of digitalis toxicity) Arises after repolarization is complete.

Early Afterdepolarization: (associated with LQT Syndrome). Arises during phase 2 or 3 of repolarization.

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2. Sinus tachycardia

Reentry

- **Requisites:**

≥ 2 pathways for impulse conduction that can be joined proximally and distally.

- **Initiation of reentry requires :**

1. Unidirectional block in one pathway.
2. Slow conduction in the alternate path.
3. Recovery of excitability at the original site of block.

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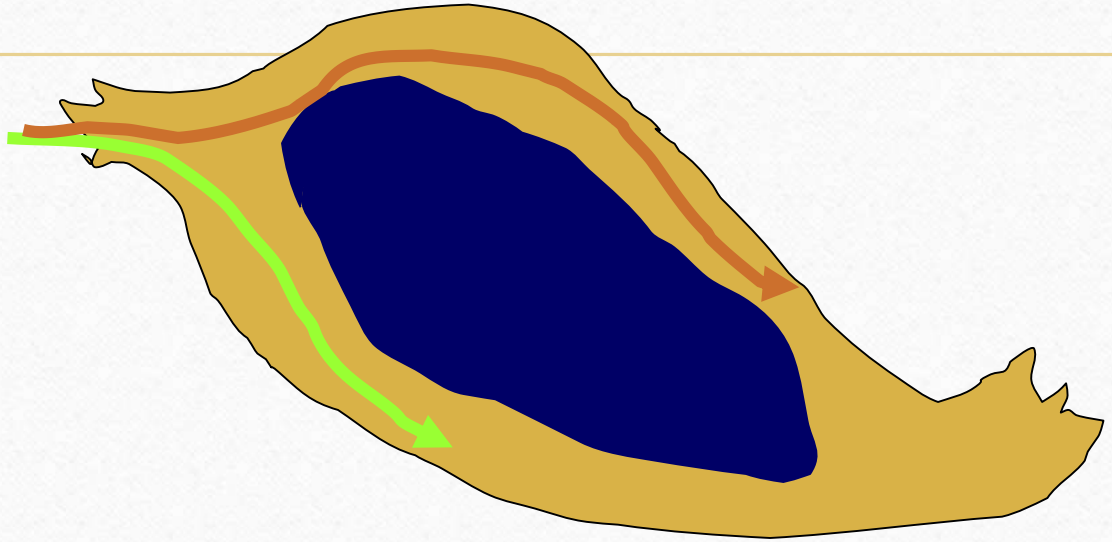
Requirements for Reentry



Basal state – conduction over both pathways – wavefronts collide

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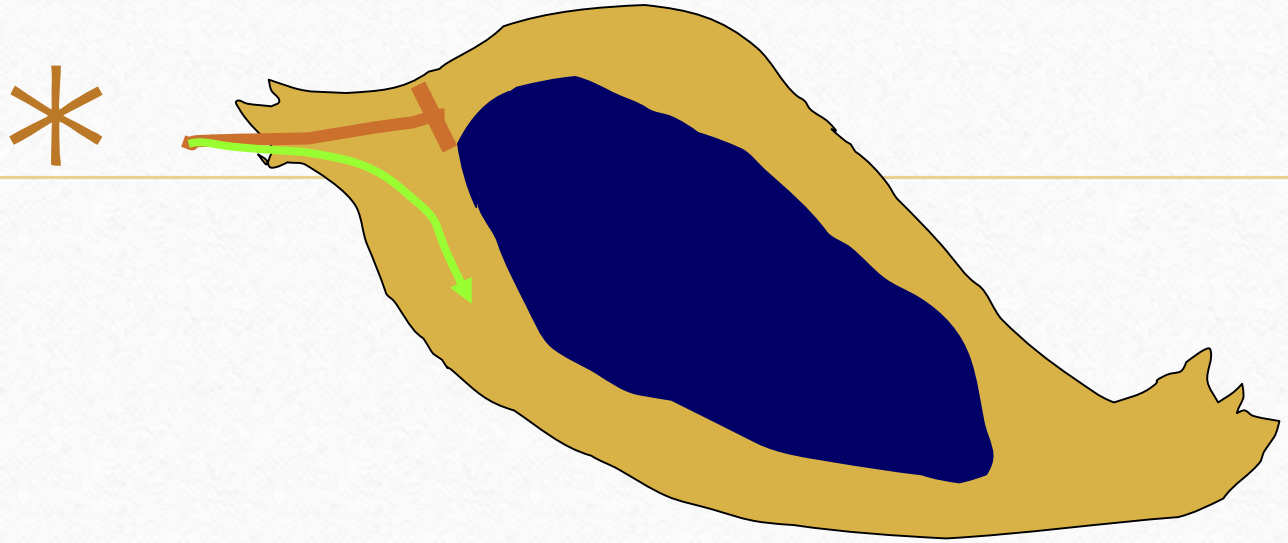
Initiation of Reentry



Premature impulse (*) finds one pathway (blue) refractory, hence conduction blocks. The alternate pathway (green) is excitable, and able to conduct.

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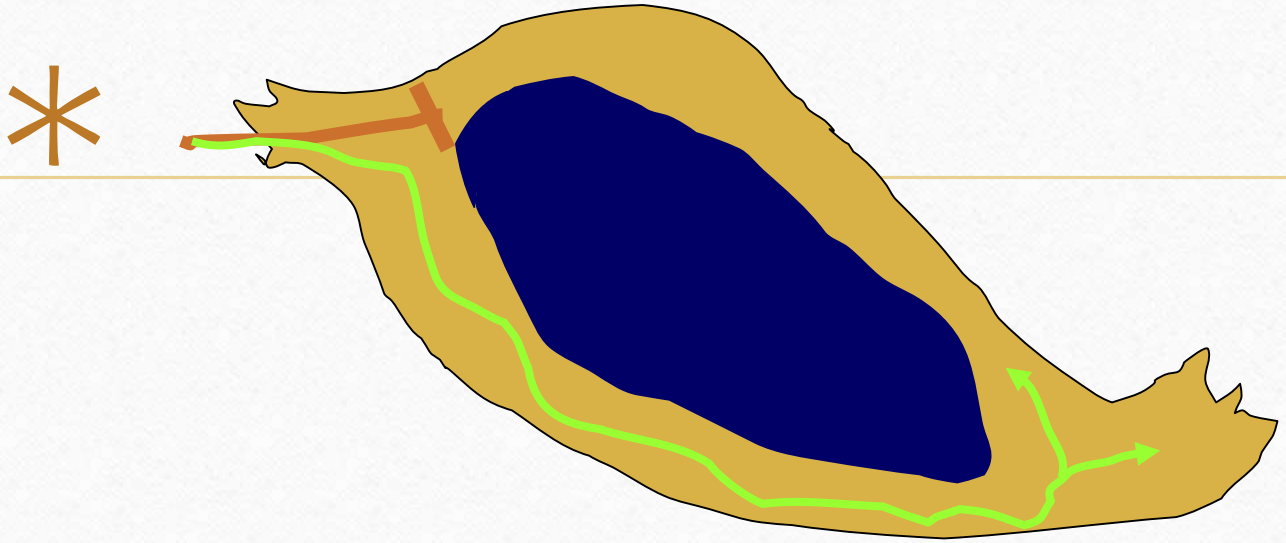
Initiation of Reentry



Although the green is able to conduct, because it was activated prematurely the tissue was still in its relative refractory period. Therefore, conduction over this pathway occurs more slowly than normal.

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Initiation of Reentry

If conduction over green is adequately slow, enough time may elapse for the original site of blue conduction block (*) to recover excitability. If this occurs, the green impulse may establish REENTRY.

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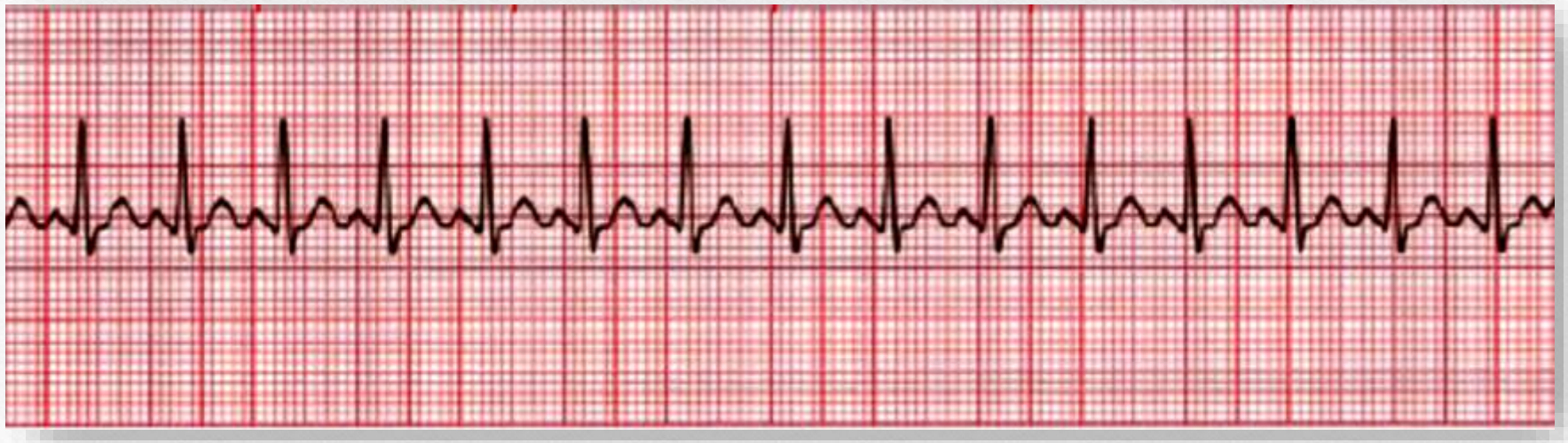
2. Sinus tachycardia: causes A. acute causes: exercise, emotion, pain, fever, acute heart failure, B. chronic causes: pregnancy, anemia, hyperthyroidism, excess catecholamine. ECG: short R-R interval.



❖ Ectopic beat (extra-systoles, premature beat):

ما إله علاج بس البعد عن الأسباب حتى ما يصير tachyarrhythmia

A premature contraction is contraction of heart before the time that normal contraction would have been expected. Most premature contraction result from ectopic foci in the heart, which emits abnormal impulses at odd time during cardiac rhythm.

Possible causes of ectopic foci are:

❶The local area of ischemia.

❷Small calcified plaques at different points in the heart press against the adjacent cardiac muscle so some fibers are irritated.

❸Toxic irritation of the AV node, Purkinje system, or myocardium caused by drugs, nicotine, or caffeine. If an irritable ectopic focus discharges once, the result is an ectopic beat. If the ectopic foci discharge repetitively at a rate higher than that of the SA node, it produces rapid, irregular tachycardia.

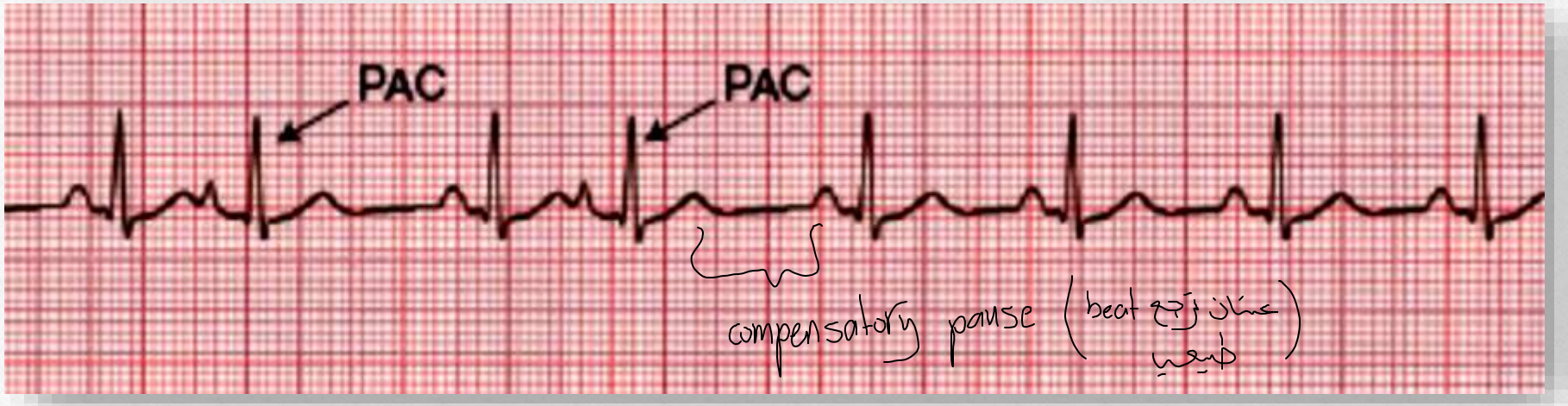
It could be: ① premature beat ② compensatory pause

1. Atrial ectopic: The ECG changes are: not reach to 250 (paroxysmal tachycardia)

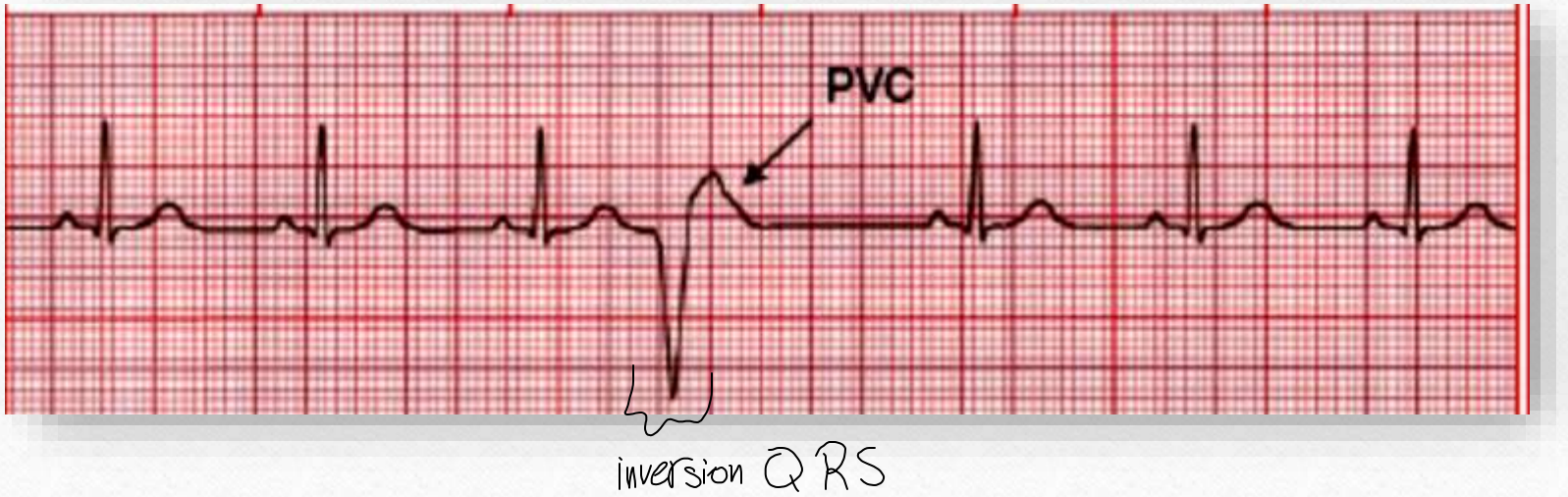
❶ The P wave of this beat occurs too soon in the heart cycle,

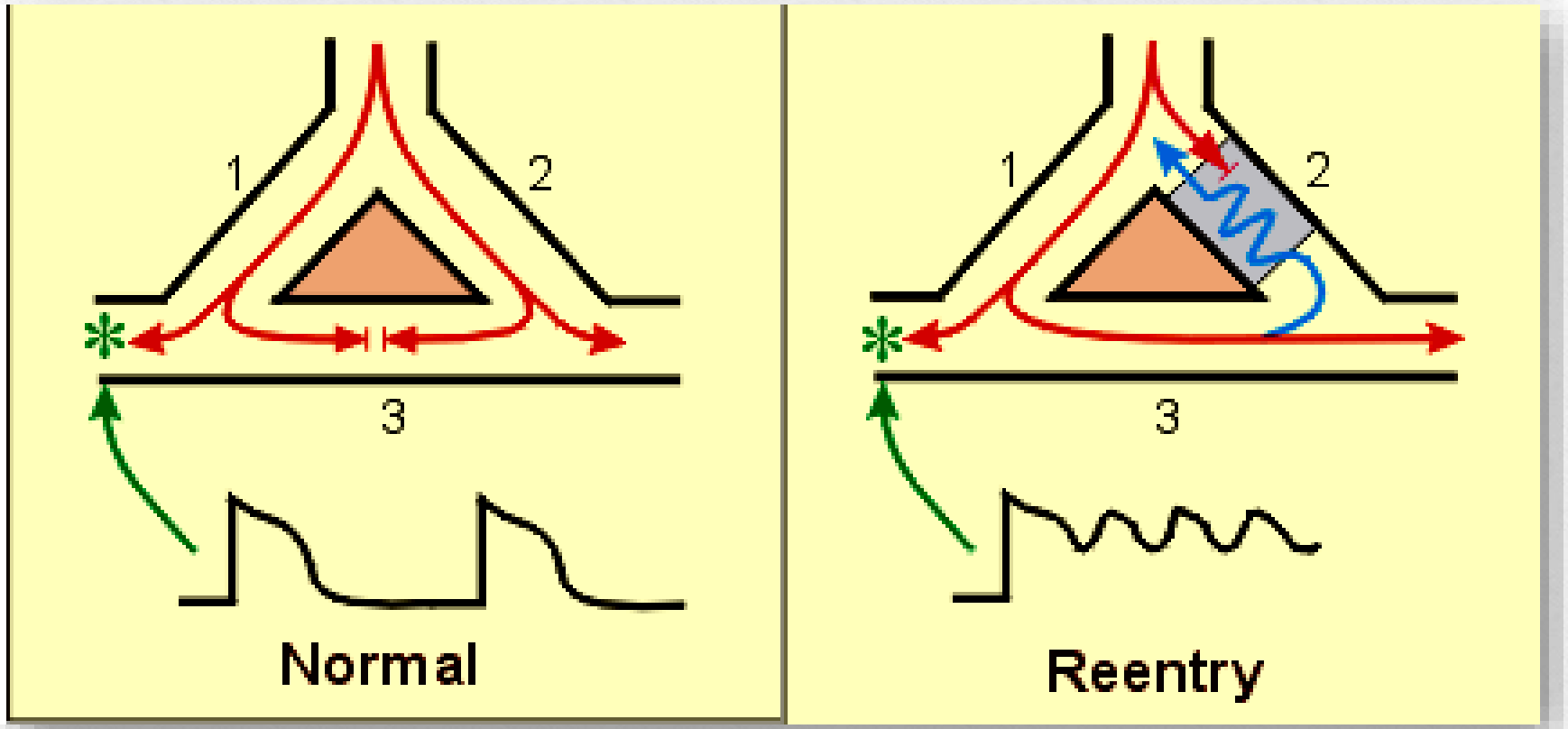
❷ The P-R interval is shortened, indicating that the ectopic origin of the beat is near the A-V node → not regular, if beat regular → tachycardia

❸ The interval between the premature contraction and next succeeding contraction is slightly prolonged, which is called (compensatory pause).



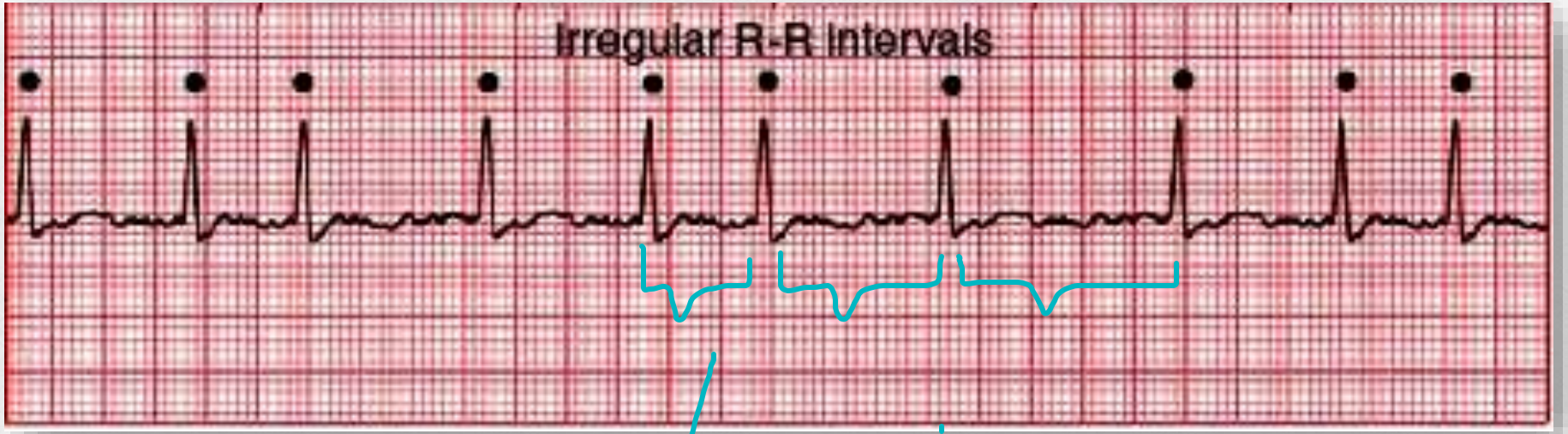
2. ventricular ectopic:





2. Re-entry: when the tachycardia is initiated by an ectopic beat but sustained by a closed-loop or re-entry circuit. Most tachyarrhythmias are due to re-entry.

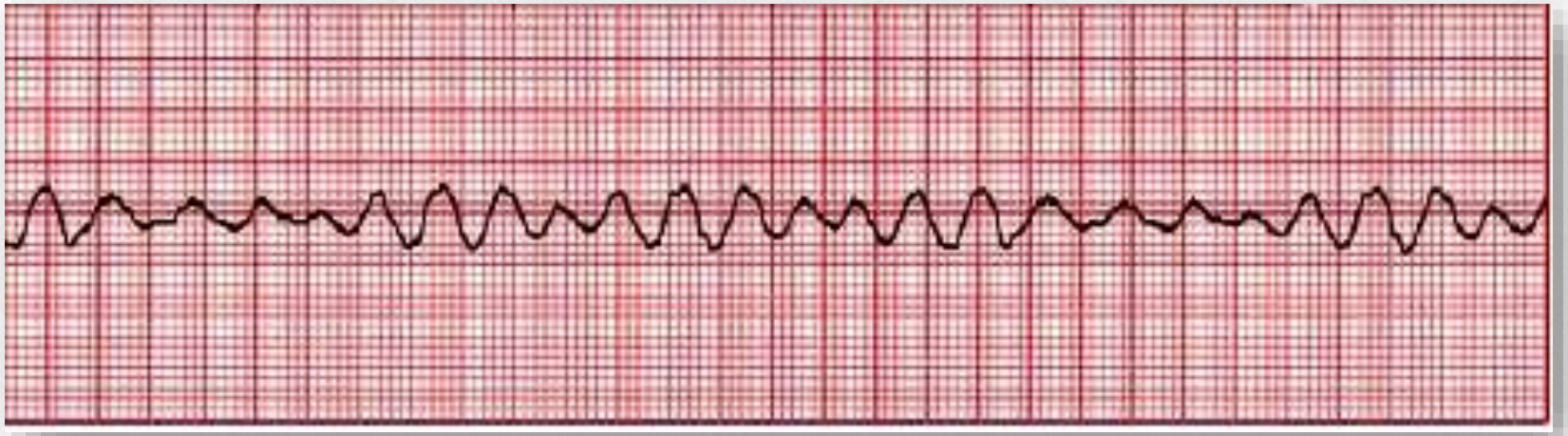
A. Atrial fibrillation: ECG: normal but irregular QRS, there are no P waves but the baseline may show irregular fibrillation waves.



B. Ventricular fibrillation: → lead to death → just apear in ECG P-wave.

The effects of ventricular fibrillation: The fibrillating ventricles, like the fibrillating atria, look like a quivering "bag of worms". The fibrillating ventricles cannot pump blood effectively and circulation of the blood stops. Therefore, in the absence of emergency treatment, ventricular fibrillation that last more than a few minutes is fatal. The most common cause of sudden death in patients with myocardial infarction is ventricular fibrillation. The ventricular fibrillation can often be stopped and converted to normal sinus rhythm by mean of electrical shock. The ECG changes: it shows waves of varying frequency and amplitude.

B. Ventricular fibrillation:



↳ Just appears p-wave

❑ Anti-arrhythmic drugs:

Classification of anti-arrhythmic drugs (Vaughan-Williams classification):

tachyarrhythmia → stop sympathatic, ↓ velocity, ↓ conduction rate, ↑ parasympathatic

Goal of therapy:

a. Therapy aims ❶to restore normal pacemaker activity ❷ modify impaired conduction that leads to arrhythmias. Conduction velocity depends on the size of the inward current during upstroke of the action potential (↑inward current→↑ the velocity of conductance)

b. Therapeutic effects are achieved by:

❶sodium- or calcium-channel blockade, ❷prolongation of the effective refractory period (it is slightly longer than an absolute refractory period), ❸blockade of sympathetic effects on the heart. Many anti-arrhythmic drugs affect depolarized tissue (ectopic foci) to a greater extent than they affect normally polarized tissue.

❑ Anti-arrhythmic Drugs:

Treatment of tachy-arrhythmias:

Class I:

a. Quinidine
b. Disopyramide
c. Lidocaine [Xylocaine]
d. Flecainide
e. propafenone

Class II:

Class II drugs are β-adrenoceptor antagonists, including propranolol, which act by reducing sympathetic stimulation. They inhibit phase 4 depolarization, depress automaticity; prolong AV conduction, and decrease heart rate (except for agents that have sympathomimetic activity) and contractility.

Major drugs:

a. Propranolol [Inderal],
b. Atenolol,
c. Metoprolol → beta adrenergic blocker
d. Bisoprolol
e. Sotalol → شائع في الأردن

Class III:

Class III drugs prolong action potential duration and effective refractory period. These drugs act by interfering with outward K currents or slow inward Na currents.

- Amiodarone [Cordarone]: give as IV form, as degoxin → degoxin فعاليته اقل من

a. Amiodarone is structurally related to thyroxine. It increases refractoriness, and it also depresses sinus node automaticity and slows conduction.

Class IV drugs:

Mechanism

a. Class IV drugs selectively block L-type calcium channels.

b. These drugs prolong nodal conduction and effective refractory period and have predominate actions in nodal tissues

- Verapamil [Calan,Isoptin]: → block calcium channel blocker

a. Verapamil is a phenylalkylamine that blocks both activated and inactivated slow calcium channels.

Other anti-arrhythimic drugs:

- ✓ **Digoxin:** can control ventricular response in atrial flutter or fibrillation.
  - Digoxin toxicity
- Extracardiac manifestations
- a. anorexia, nausea, vomiting
- b. Diarrhoea
  - Cardiac manifestations

a. Bradycardia

b. Multiple ventricular ectopics

c. Ventricular bigeminy (premature ventricular contraction)

❑ Treatment of Brady-arrhythmia:

1. Atropine → muscaranic antagonist

a. Atropine blocks the effects of acetylcholine. It elevates sinus rate and AV nodal and sinoatrial (SA) conduction velocity, and it decreases refractory period.

b. Atropine is used to treat bradyarrhythmias that accompany MI.

2. Isoproterenol [Isuprel]

a. Isoproterenol stimulates β-adrenoceptors and increases heart rate and contractility.

b. Isoproterenol is used to maintain adequate heart rate and cardiac output in patients with AV block.

Thank You