Normal Ryhythm
- Normal Heart Rate (60 - 100 beat/min)
- orgeno of electrical impulses → SA node
- Normal conductive patway
- Normal velocity

لو صار فيها مشكلة ← بتعيق وصول electrical impulses من مكان ثاني ← خلال بـ Heart Rate بكون أقل من ٦٠

Pathophysiology-Arrhythmia

Faculty of Pharmaceutical Sciences

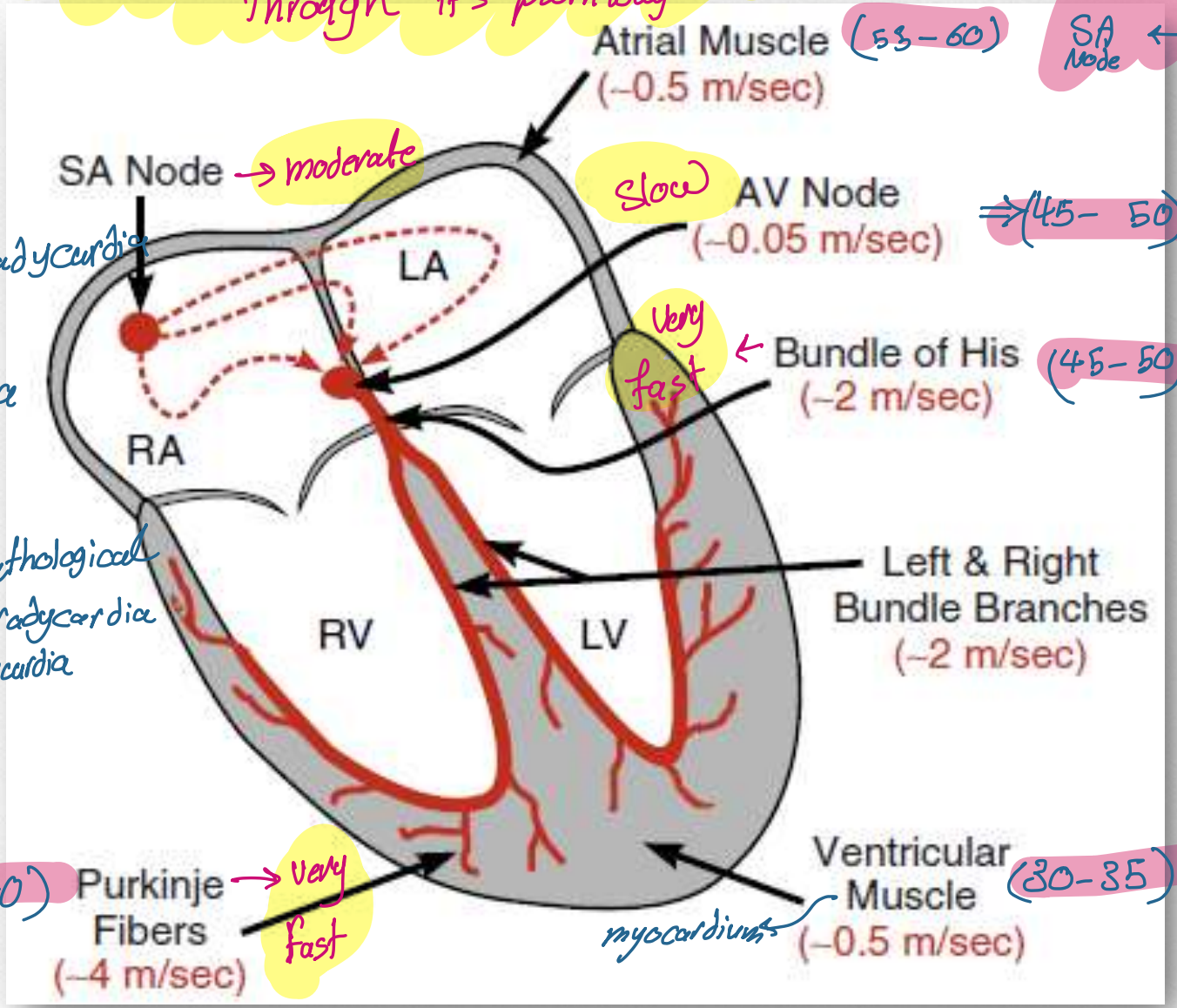
Dr. Amjaad Zuhier Alrosan, Dr. Abdelrahim Alqudah

Velocity to Conduct the action potential through it's pathway

Normal Heart Rate
SA Node ← (60-100) Beat/min

⇒ Normal ⇒ Physology
* sleep → Normal Bradycardia
* exercise → Normal tachycardia
Pregnancy

⇒ Abnormal ⇒ Pathology
* Hypothyrodism → Pathological Bradycardia
* Hyperthyrodism → tachycardia



Tecycardia * اعلى من 100

100 – 150 Simple

* 150 – 250 Proximal فجائي

* 250 – 350 Atrial fluter النوع الأسوء

> 350 fibrilation

atrial ←

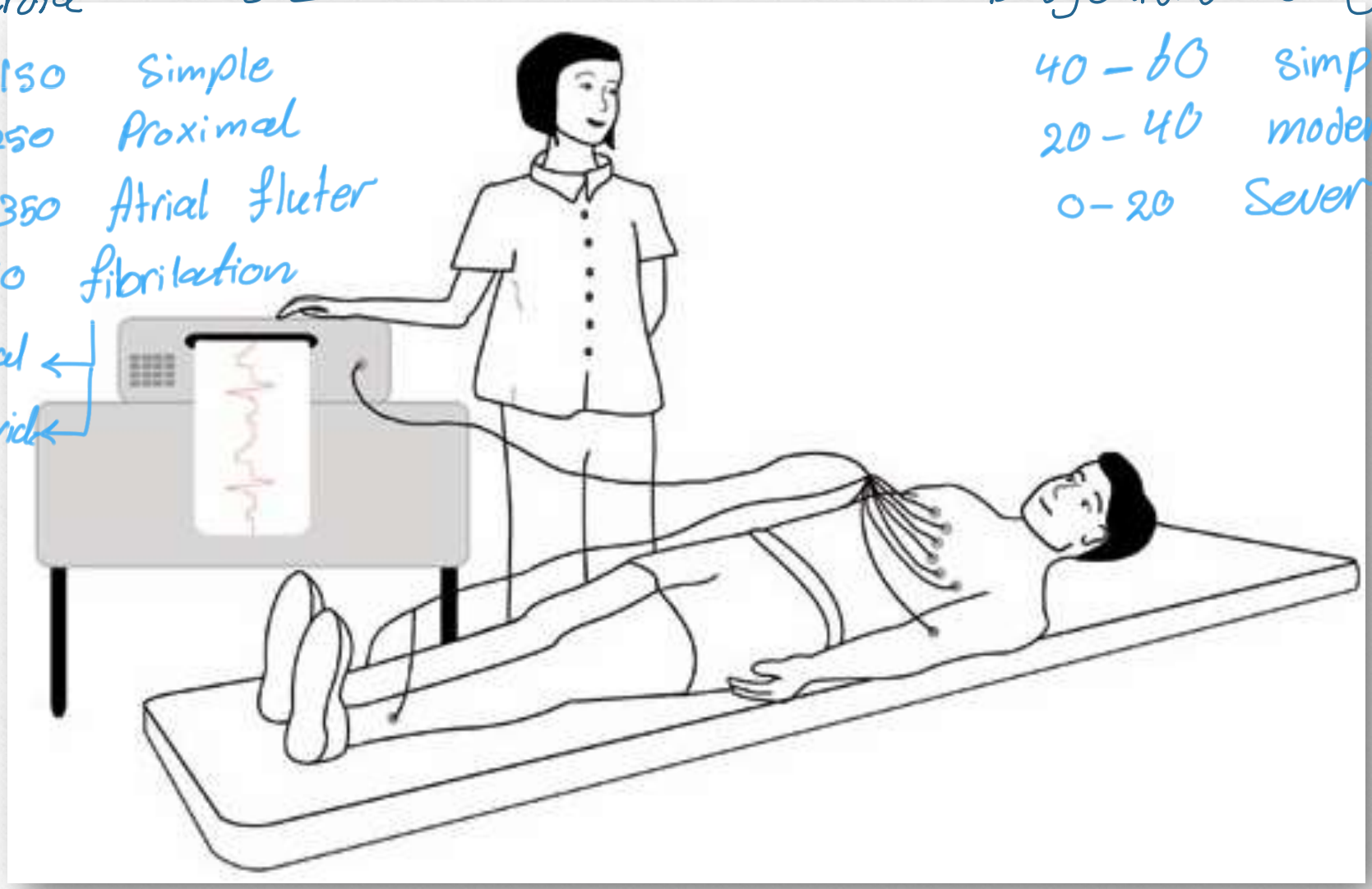
Ventricle ← الأسوء

Bradycardia * اقل من 60

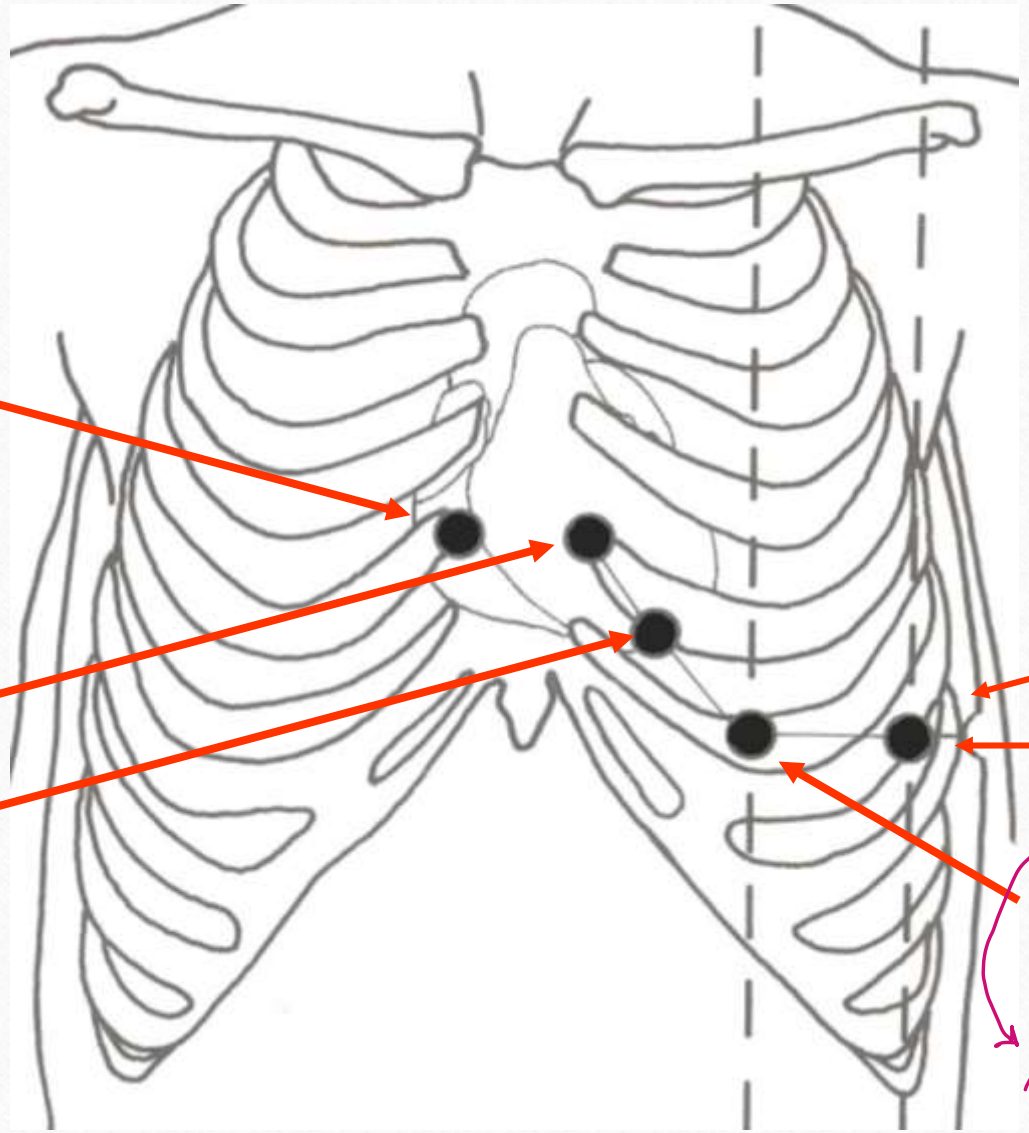
40 – 60 simple

20 – 40 moderate

0 – 20 Severe



Placement of Precordial Electrodes



Arrhythemia أنواع الـ

SA node → Sinus arrhythmia

AV node → Atrial arrythmia

Atrium أو حولين منطقة الـ

Ventricular → ventricular arrythmia

Above the Ventricle → Supraventricular arrythmia

Mechanisms اللي بتصير فيها الـ Arrythmia

1 Increas Automestiy

عضلة القلب قادرة على انتاج action potential من تلقاء نفسها

Beat normal بس سريعة

عدد النبضات زيادة عن الـ Normal

Sympathatic pathway ← متى بتصير؟ - لما احفز

Ca^+ agonest → contraction بتزيد

في وقت قصير بين النبضات بس لسا في وقت

هاد أسوء من اللي قبله

2 Triggered Automestiy

بتأثر على الـ Beat نفسها

في حالات Iscemia

SA Node بتفكر انه ما في Blood flow فبتزيد الـ contraction

بصير هاد النوع كنوع من رد الفعل

في صنفين نوعين:

① early after Depolarization (EAD) الأخطر

- بعد ما يصير ventecular depolarization

P wave → QRS قبل ما تنتهي → second Beat

② delyed after Depolarization (DAD)

- بنتهي الـ depolarization & Repolarization بس متأخر بعدها على طول

second Beat

Refractory period

In tachycardia

أقصى

delaying بقل ومدة ال

بسبب تحفيز ال

Sympathatic

لما بدي أعالجه

بثبط ال Sympathetic

وبحفز ال Parasympathatic

In Bardy cardia

الأول

delaying بزيد ال

بسبب تحفيز ال Parasympathatic

لما بدي اعالجه بثبط ال

Para sympathatic

وأحفز ال Sympathatic

كل ما زاد ال time ⇐ يعني زاد ال Delay ← Brady والعكس

Interval	Average time (sec)	Range time (sec)	Events in the heart during interval
P wave			Atrial depolarization
PR interval	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

لو قلت عنها ← يعني عندي tachy

لو زادت عنها يعني عندي Brady

سؤال الامتحان : بيجي تعريف وتعين نوع الـ Heart Blocks

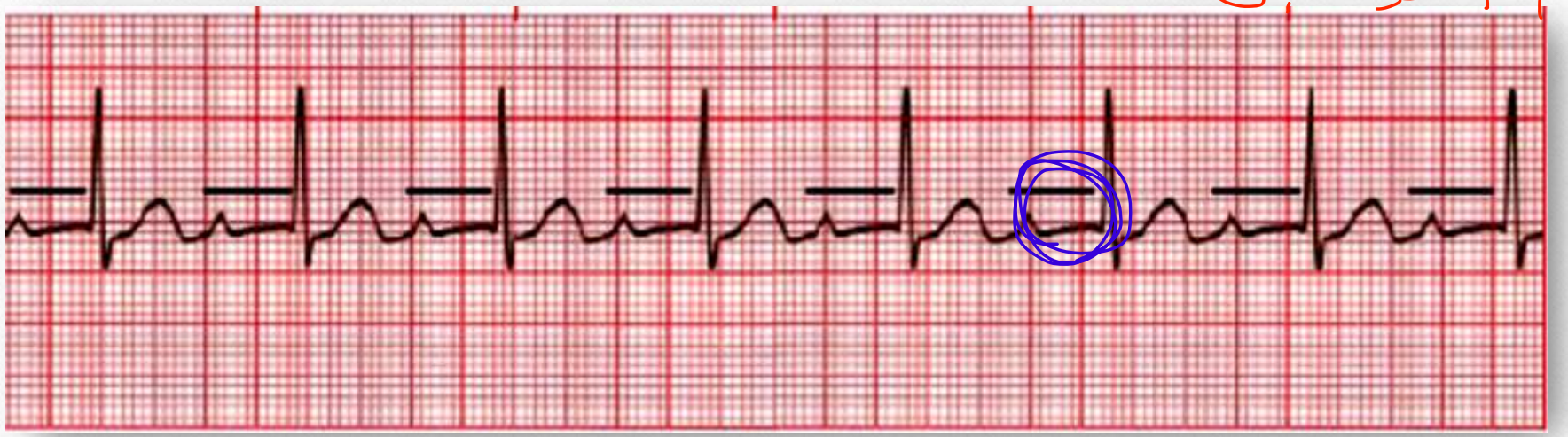
Heart blocks:

I. Block at the level of AV node:

Vagus nerve عن طريق ← بزيد Para وبقلل Sympath
عندي دوا اسمه Digaxin بستخدمه عشان تقلل من tachycardia

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.

كلهم ظاهرين بس PR طويلة



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

→ QRS مش موجودة → T-wave لانه ال ventricle ماصارله بالتالي مافي

not conductive

عشان يكمل ويعمل Depolarization

Repolarization

When take High dose of B-Blocker

Antagonist to sympathatic

⇒ sympathatic

بأثر على Cardic muscle

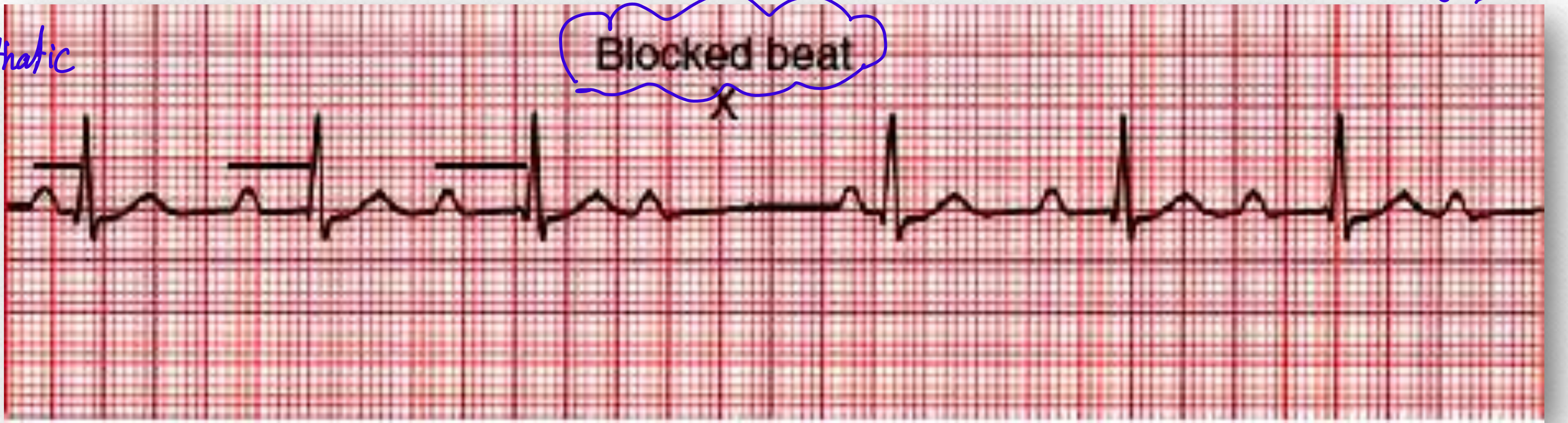
بزيد Contractility

فَ لما اعطي B-Blockers

بوقف ال sympathatic

فَ بوقف مشي ال contraction

in ventricles



→ Para

مابأثر على عضلة القلب

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

High dose of calcium
⇒ Plateau بتزيد مدة الـ
وبالتالي بزيد الـ delay
لدرجة بفصل الـ conduction
بين الـ atrial & ventricular

Heart Rate كنا حكي انه
Normal (60-100)
تمام؟!
atrial ⇐ بعاي الحالة ال
ما بشتغل بشكل متوافق مع
Ventricle
two Rates فبينشأ معي

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.

Caused by: Acute myocardial infarction, calcify aortic stenosis, cardiomyopathy, drugs (digoxin), increase of potassium.

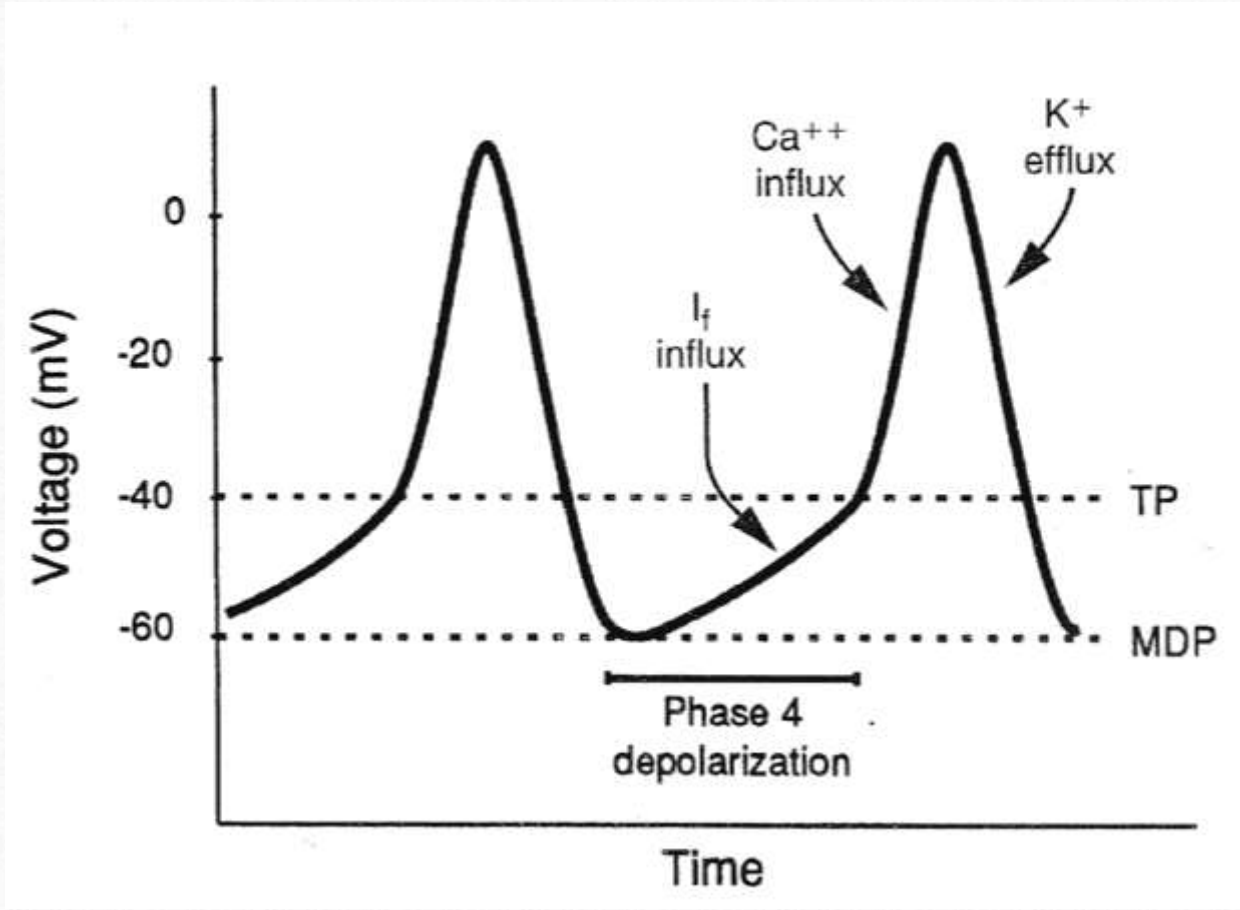
Block below AV node: A. block at Bundle of His, B. Block at the branches (Right or Left branch).

❑ Sinus rhythm: ⟹ SA Node failure

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:

Normal Pacemaker Activity in the SA Node



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1. Sinus bradycardia:

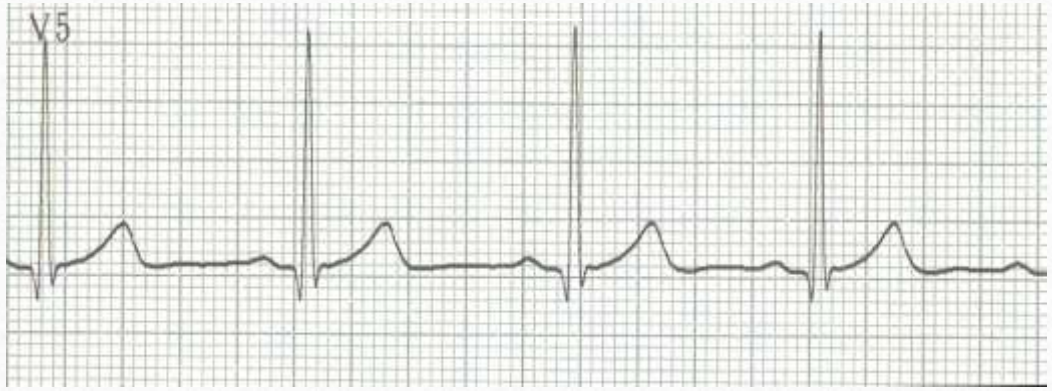
- Abnormally slow heart rhythm.
- May result from:
  - Abnormal impulse formation (Sinus bradycardia).
  - Abnormal conduction of impulses (AV conduction block).

1. Sinus bradycardia:

- Sinus node disorders.

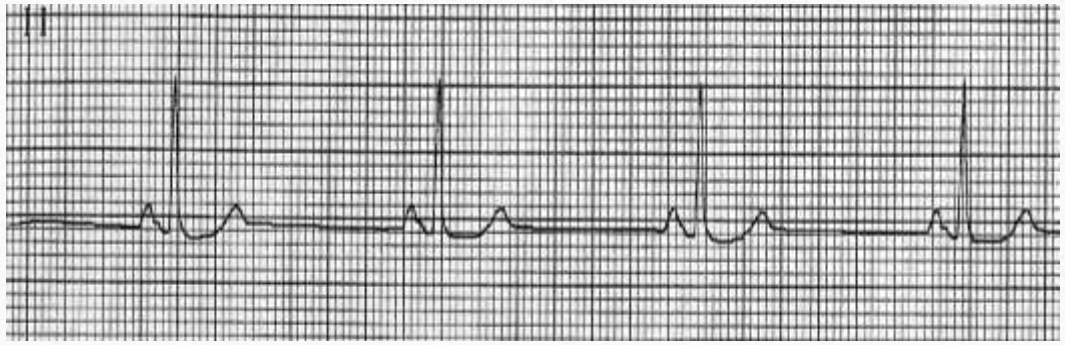
- Sinus node (and AV Node) Function is profoundly influenced by autonomic nervous system tone.
  - Increase in sympathetic tone or circulating catecholamines increases sinus rate.
  - Increase in parasympathetic tone slows sinus rate.

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:

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

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 :**
    - **Automaticity.**
    - **Triggered activity.**
  - **Abnormal impulse conduction:**
    - **Re-entry.**

شرحناهم بالأول

Block بتهيي لانه عندي ischemia او

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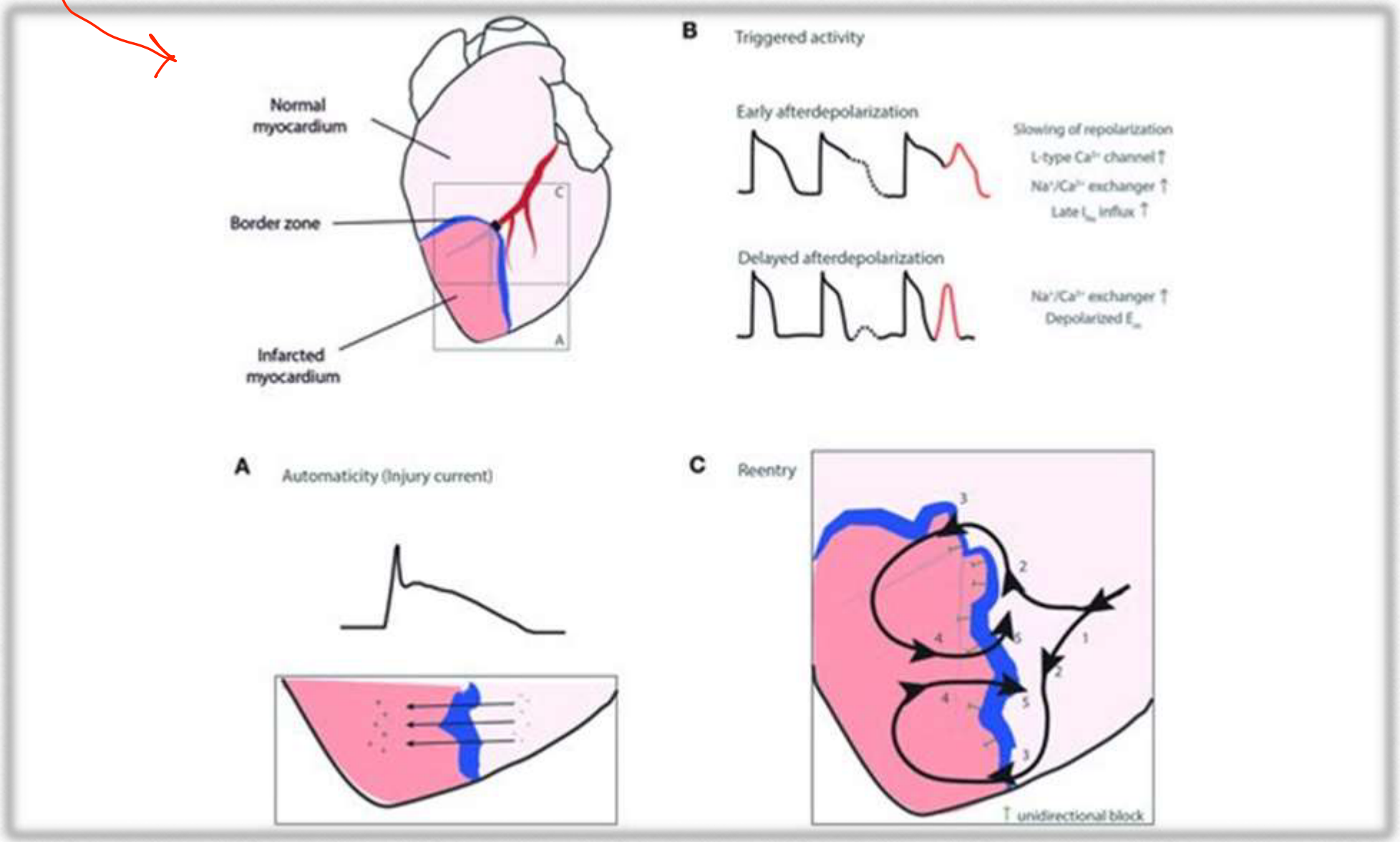
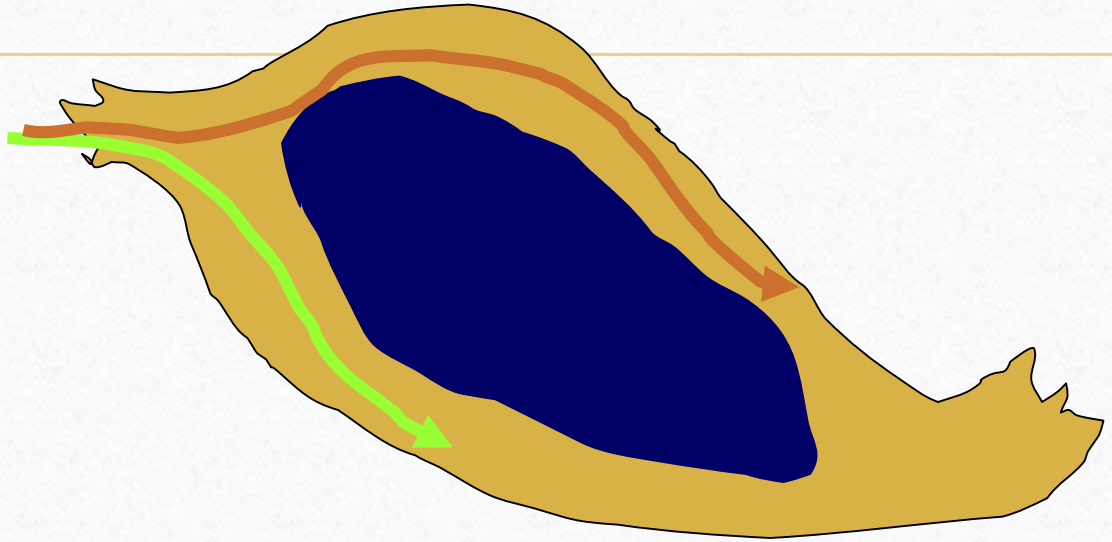


https://www.researchgate.net/publication/337023955_Ventricular_Arrhythmias_in_First_Acute_Myocardial_Infarction_Epidemiology_Mechanisms_and_Interventions_in_Large_Animal_Models/figures?lo=1

Requirements for Reentry



Basal state – conduction over both pathways – wavefronts collide

الوضع الطبيعي

فيه موجات

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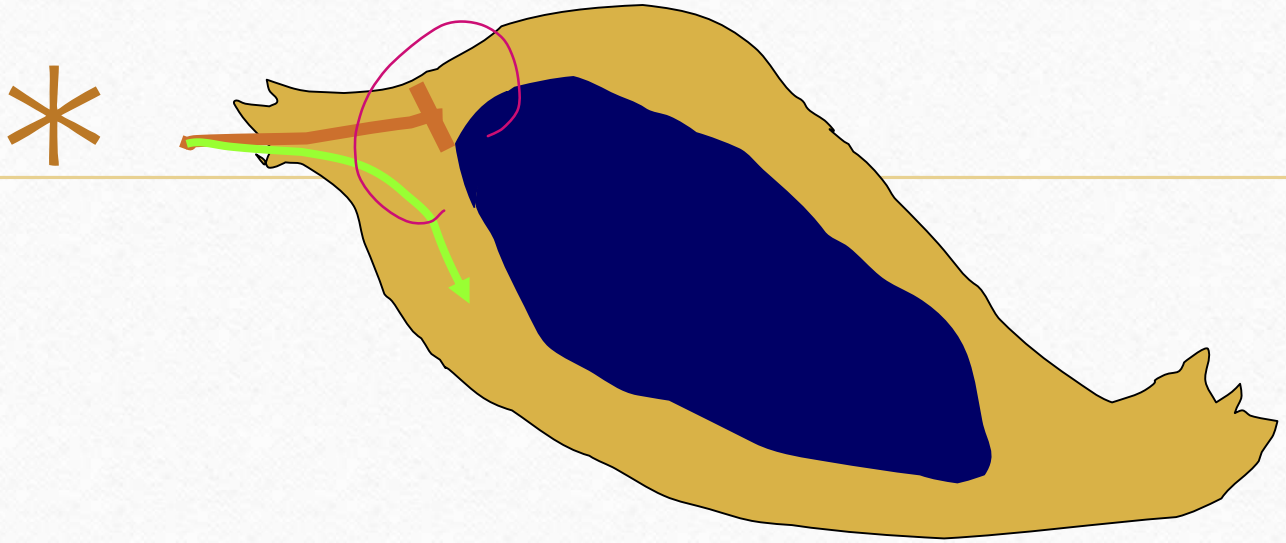
Initiation of Reentry



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

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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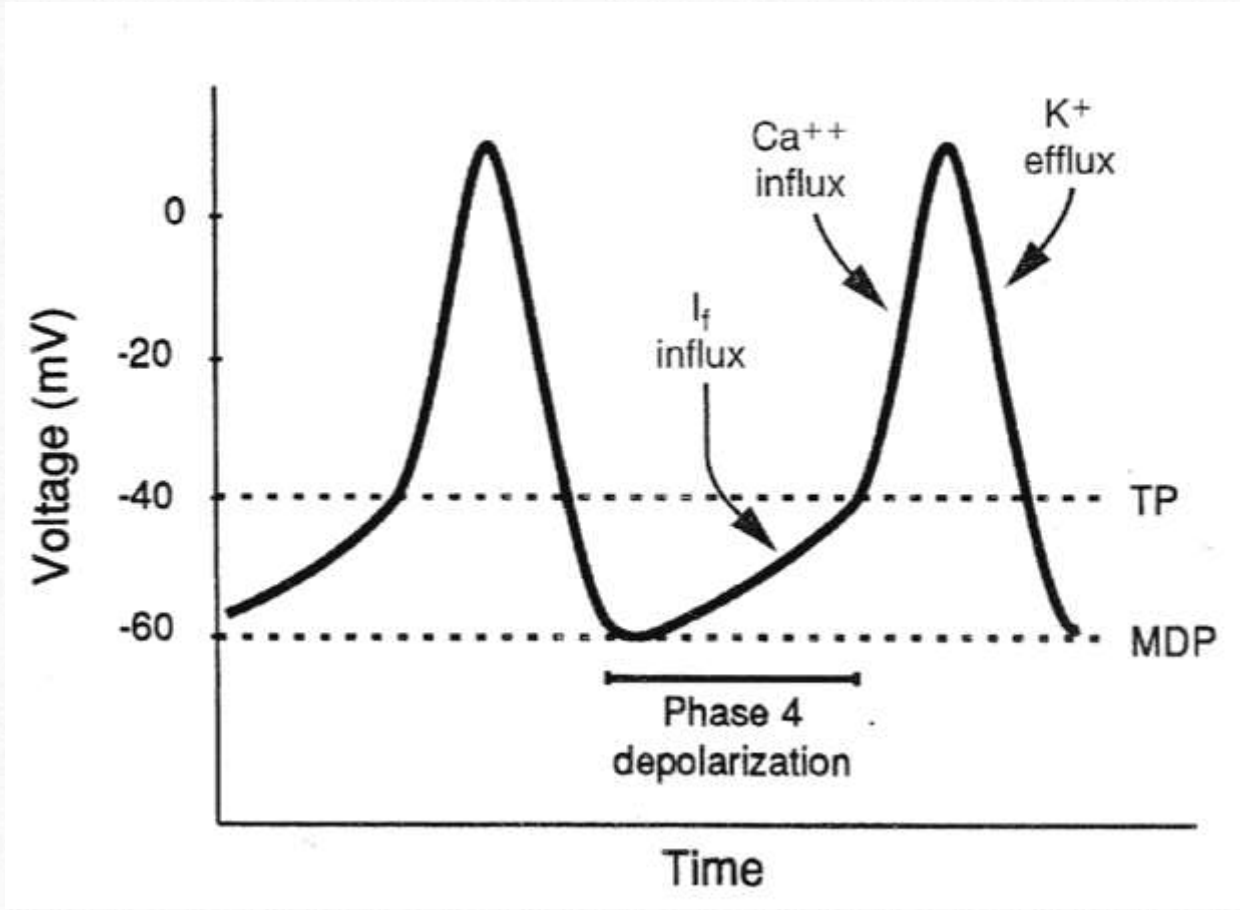
Normal Pacemaker Activity in the SA Node



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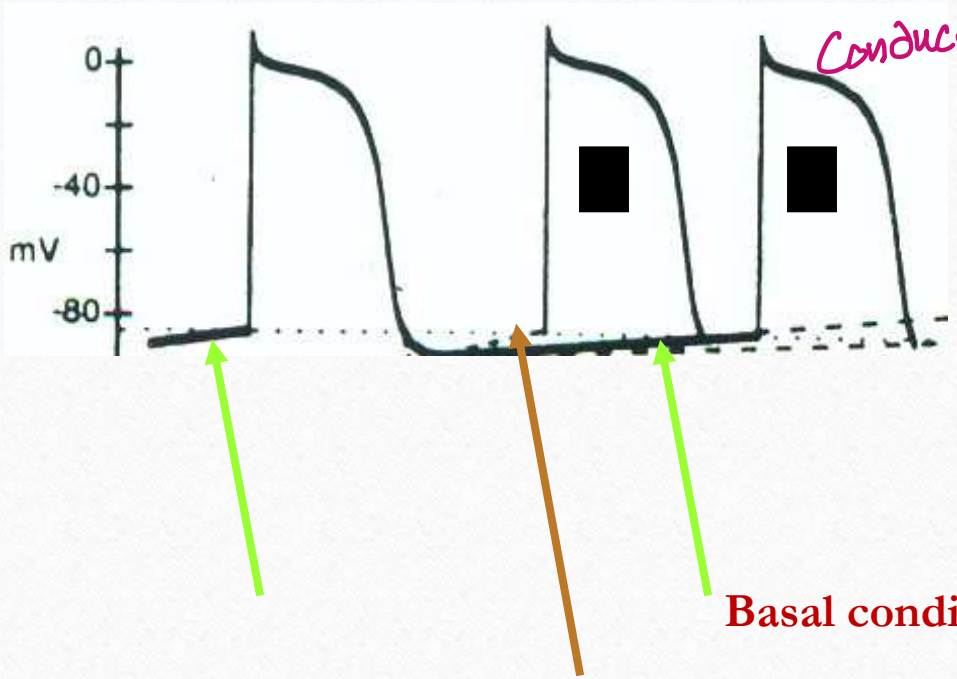
2. Sinus tachycardia

Enhanced Normal Automaticity

المشكلة بال SA Node بتزيد HR اعلى من 100

المشكلة بال AV Node بتعكل من الconduction



Increased slope of phase 4 depolarization

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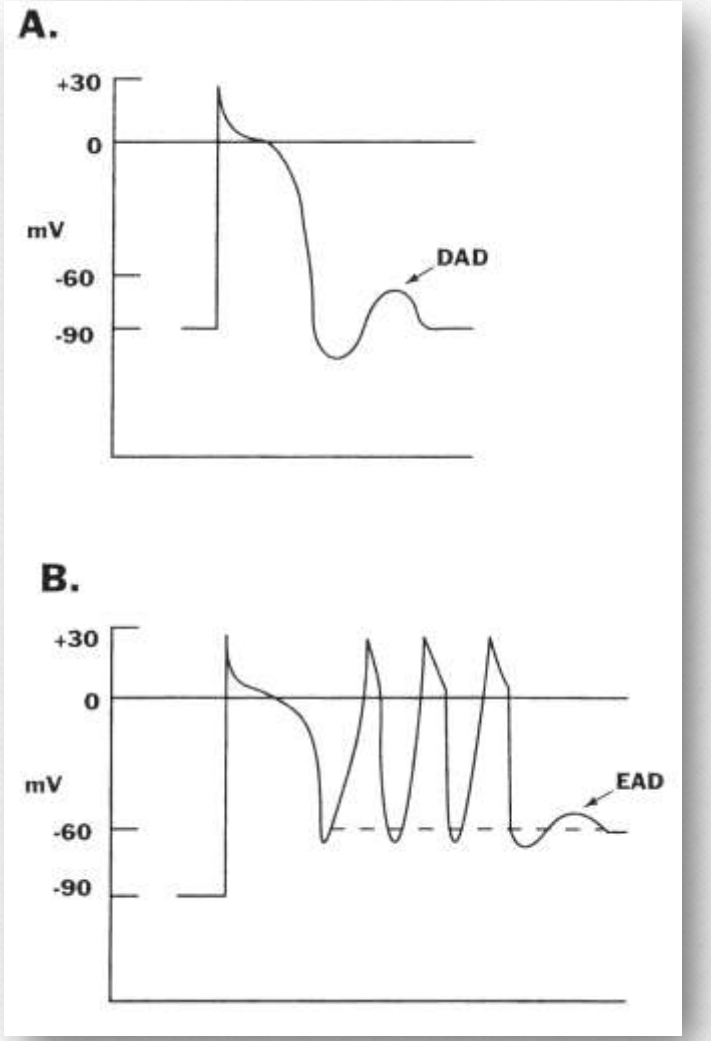
2. Sinus tachycardia

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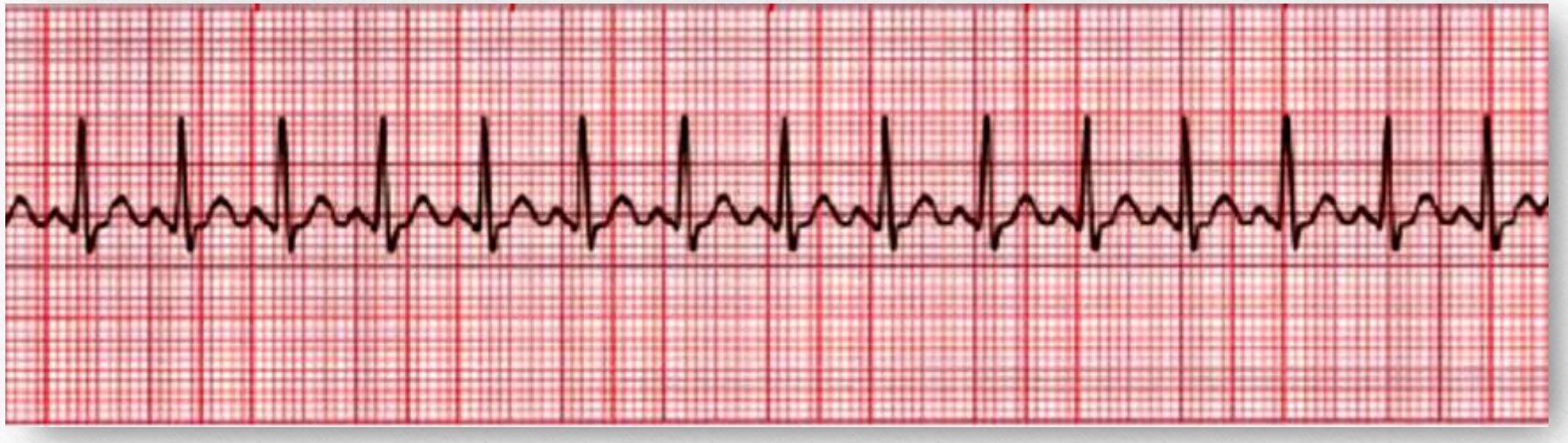
2. Sinus tachycardia: causes A. acute causes: exercise, emotion, pain, fever, acute heart failure, B. chronic causes: pregnancy, anemia, hyperthyroidism, excess catecholamine. ECG: short R-R interval.



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

عندي نبضة عني ناضجة بتصير فجأة

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:

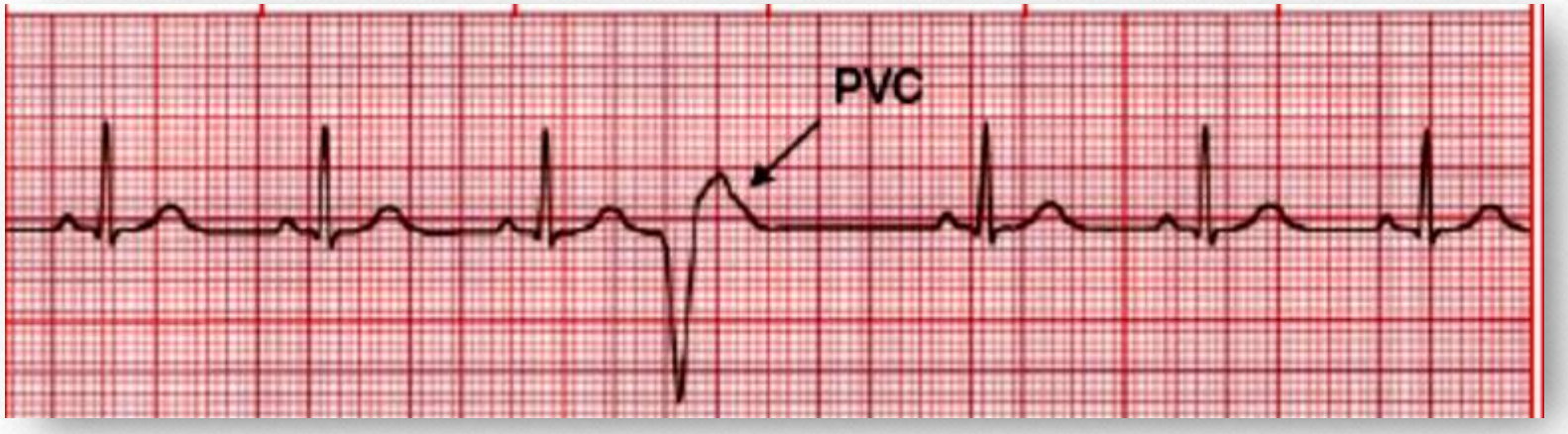
1. Atrial ectopic: The ECG changes are:

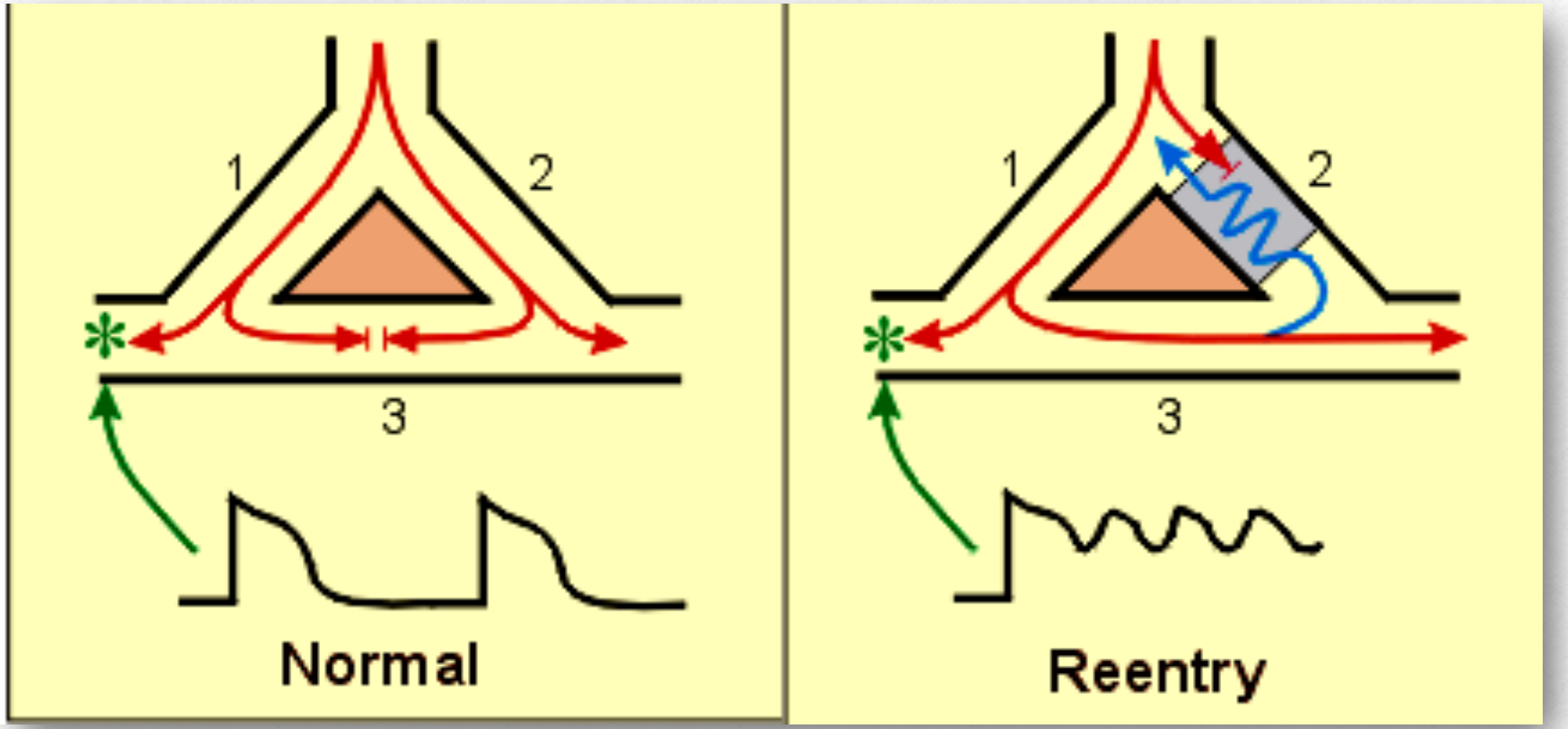
❶The P wave of this beat occurs too soon in the heart cycle,

❷ The P-R interval is shortened, indicating that the ectopic origin of the beat is near the A-V node

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

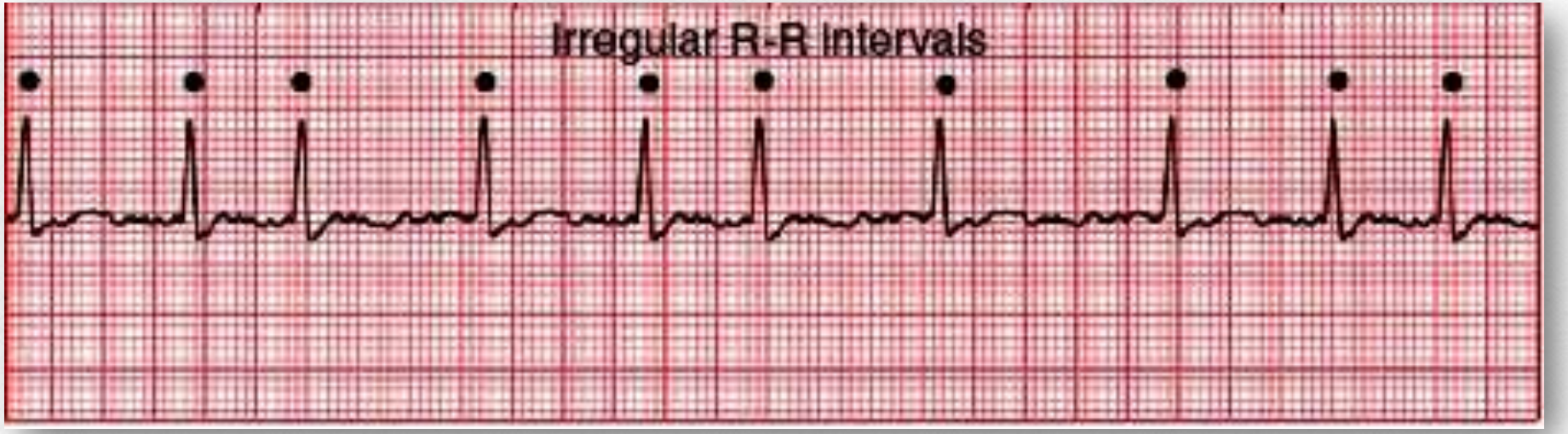
2. ventricular ectopic:





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.

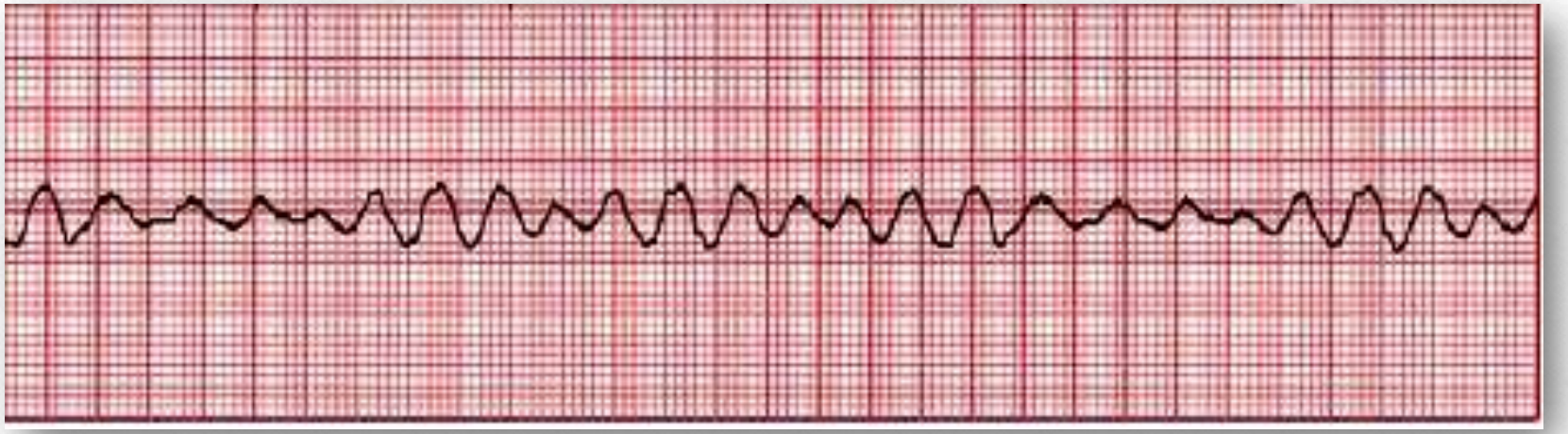
A. Atrial fibrillation: ECG: normal but irregular QRS, there are no P waves but the baseline may show irregular fibrillation waves.



B. Ventricular fibrillation:

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:



❑ Anti-arrhythmic drugs:

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

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
d. Bisoprolol
e. Sotalol

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]:

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

Class IV drugs:

Calcium channels Blockers

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]:

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

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.

agonest

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