تفريغ علم وظائف الأعضاء المرضي

اسم الموضوع:

Arrhythmia

إعداد الصيدلاني/ة:

لؤي المجدلاوي

Soul Academy

لجان الدُّفعات

بسم الله الرحمن الرحيم

* يتحدث الشابتر عن عدم انتظام نبضات القلب و الاسباب لها وعلاجاتها ورسماتها ال ECG

* التركيز على تعريفات ال Arrhythmia والعلاجات لها

* هذا الشابتر تم ترجمته اى مادة السكند وهو أول شابتر بالسكند

Pathophysiology-Arrhythmia

Faculty of Pharmaceutical Sciences

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اللهم صل على سيدنا محمد

Normal

HB = (60-100) SA node

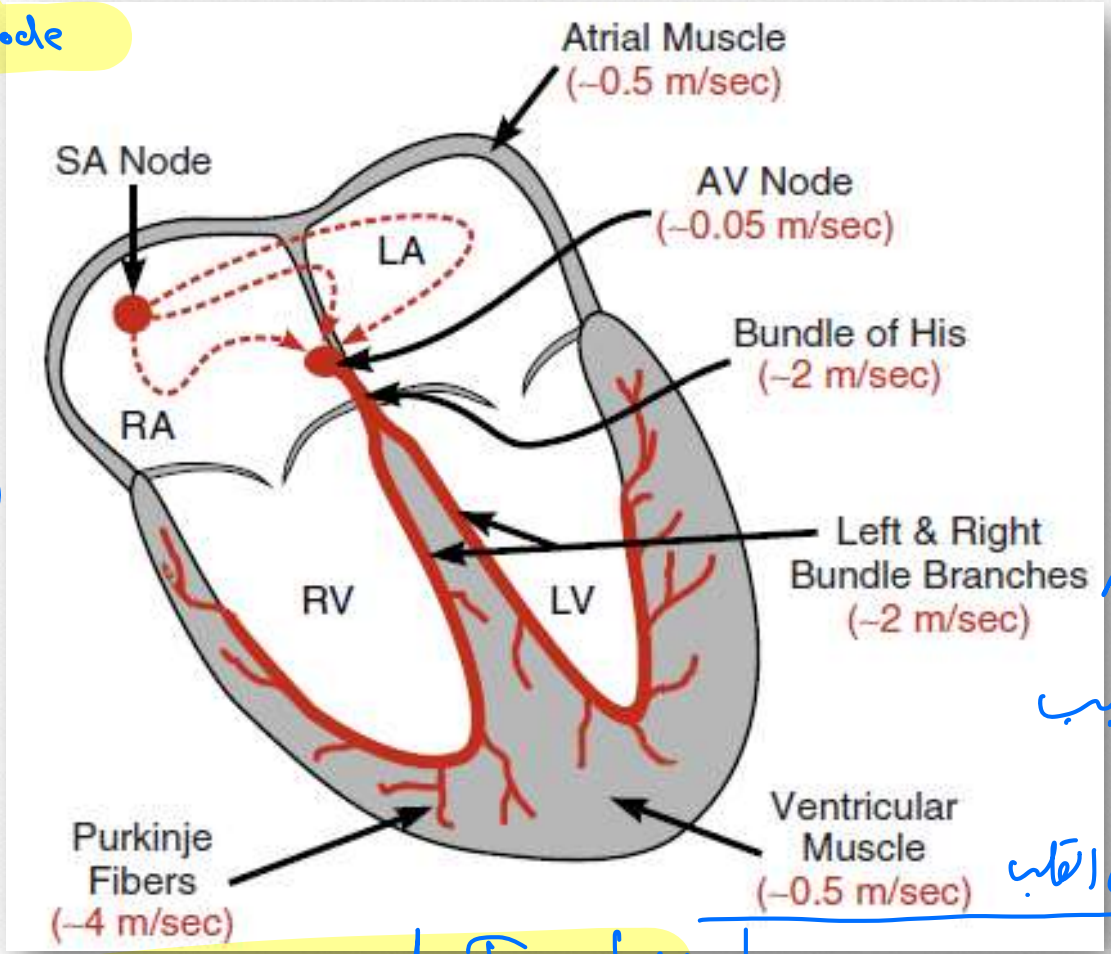
Atrial cell = (55-60)

AV node = (45-50)

Bundle His = (45-50)

Purkinje = (35-40)

Ventricl myocardium = (30-35)



< 60 → bradycardia

> 100 → Tachycardia

* وقد يكونا انخفاض وارتفاع بنبضات القلب Normal مثل اثناء النوم ↓ او اثناء ممارسة الرياضة ↑

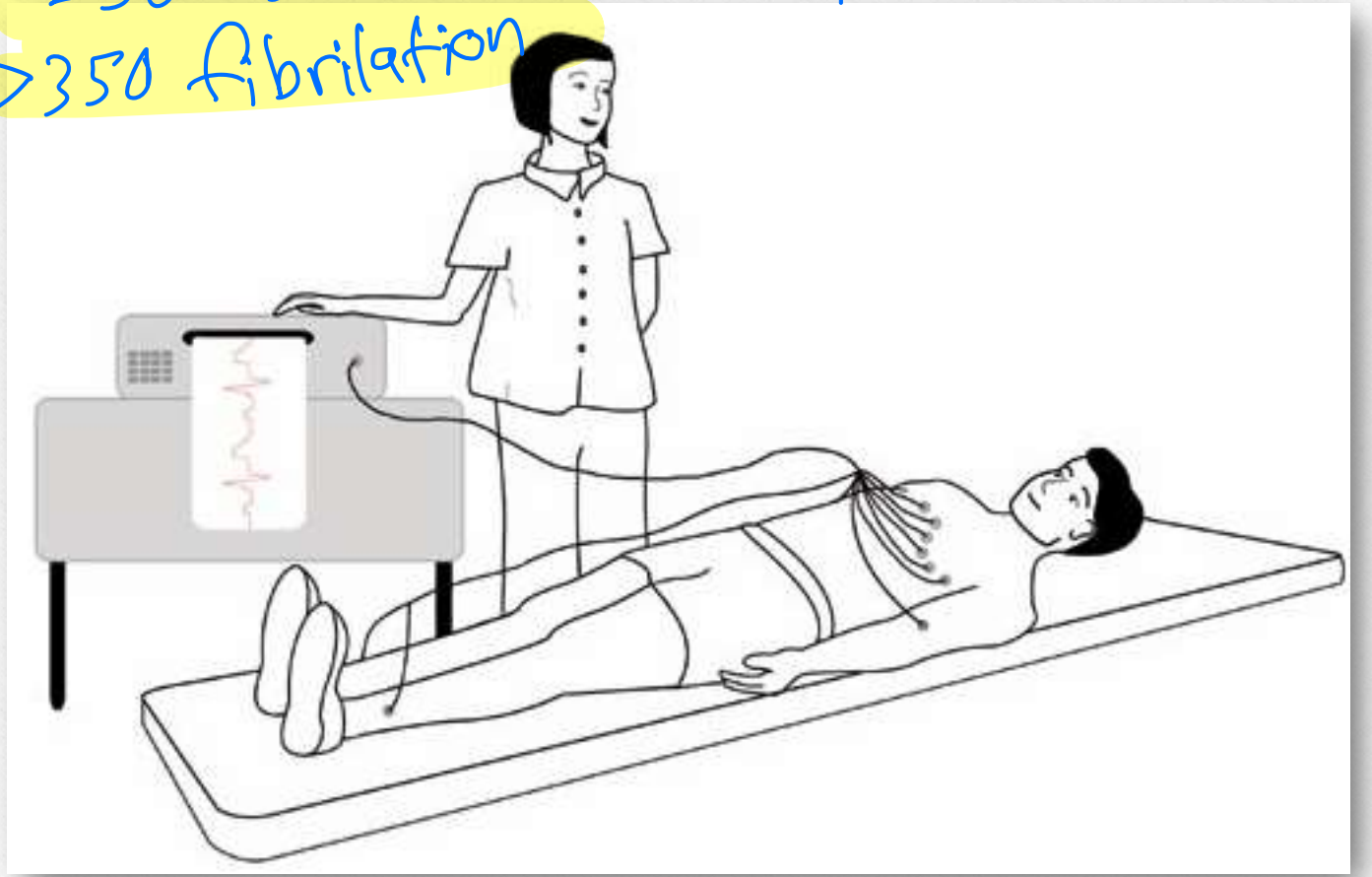
* وقد يكون Abnormal بسبب مرض مثل Hypothyroidism ويسبب ↓ ضربات القلب

100-150 simple Tachy

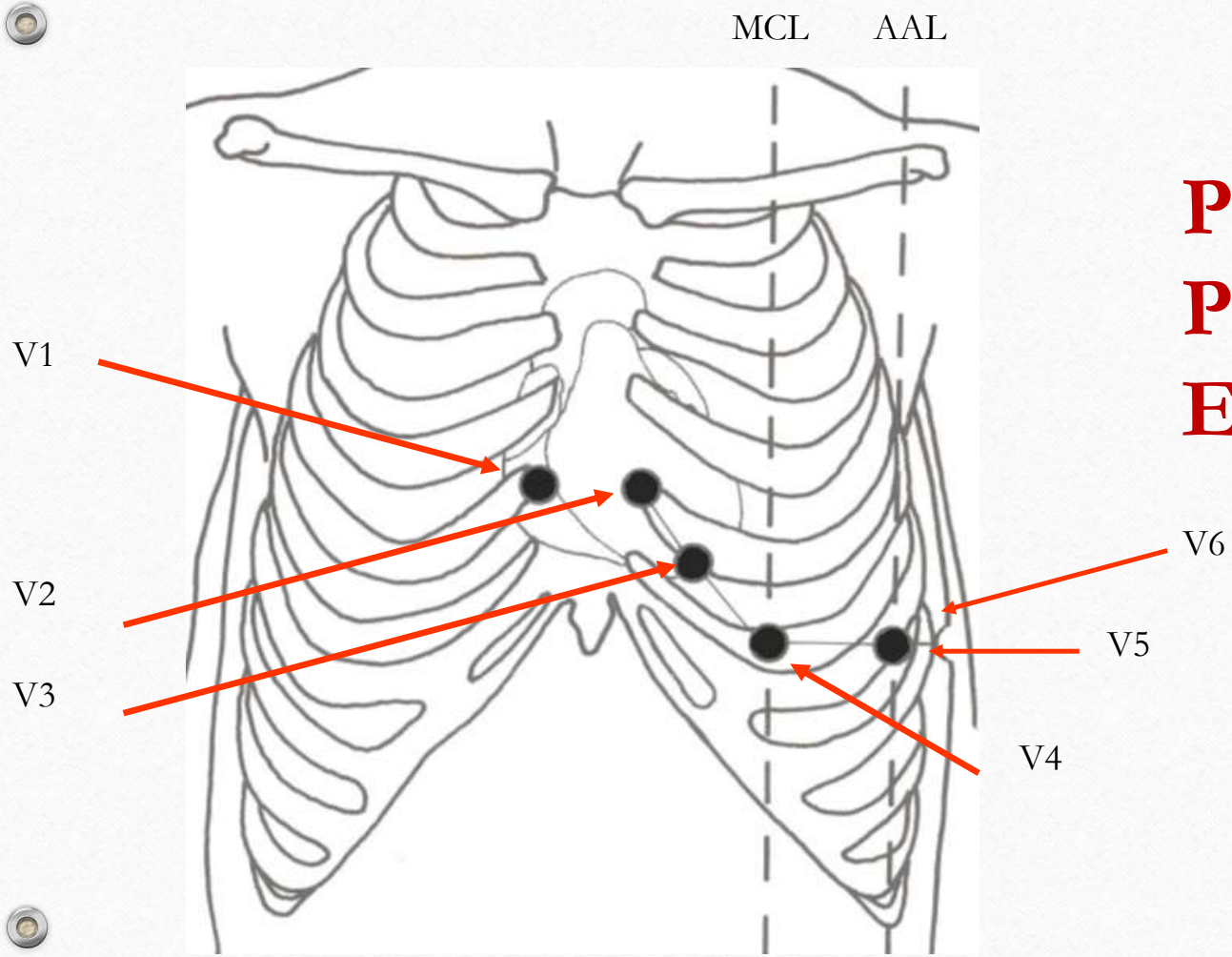
40-60 simple brady

150-250 Proximal Tachy
250-350 Atrial flutter
>350 fibrilation

20-40 moderate brady
0-20 severe brady



Placement of Precordial Electrodes



Interval	Average time (sec)	Range time (sec)	Events in the heart during interval
		* الارقام مطلوبة	
P wave			Atrial depolarization
PR interval	0.18	0.12 to 0.2 إذا زادت المدة عن 0.2 هناك delay إذن brady أما إذا قلت المدة عن 0.12 tachy	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

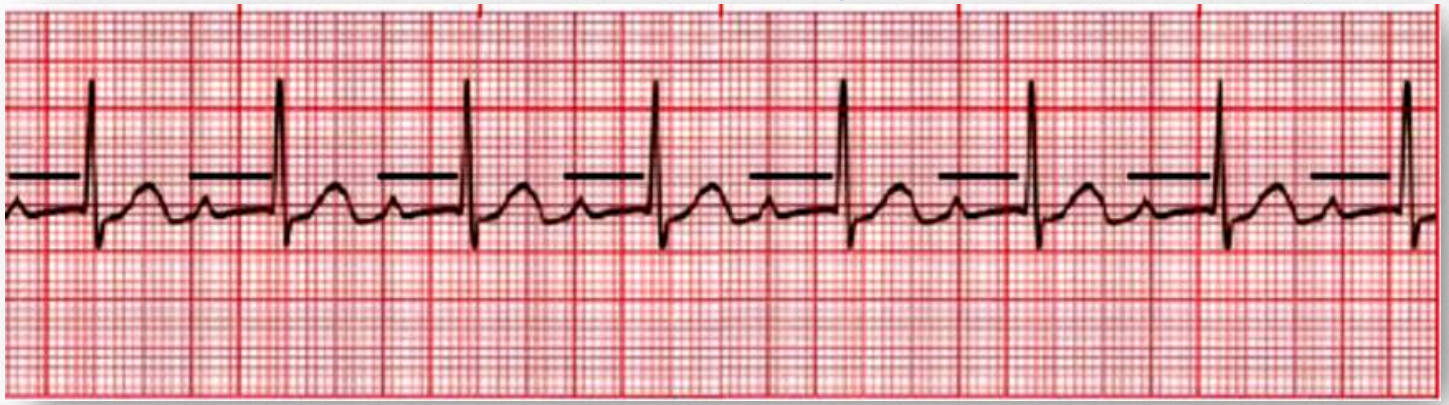
* سؤال الامتحان
التعريف وانت تختار نوع ال Arrythmia

Heart blocks:

I. Block at the level of AV node:

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.

* السبب الرئيسي استخدام دواء digoxin



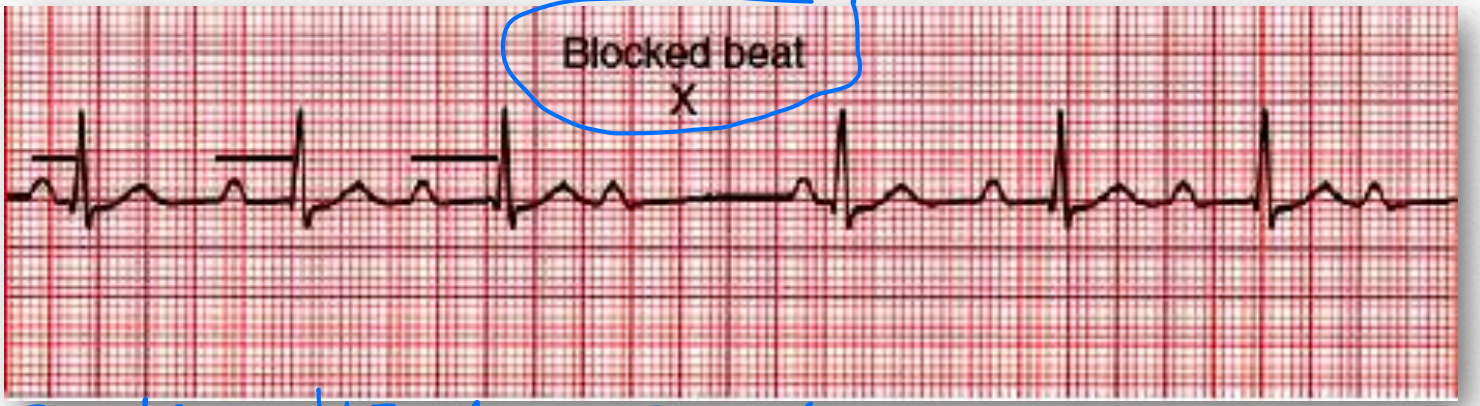
* digoxin يحفز ال Parasympathetic ويثبط ال sympathetic عن طريق إنشاء الـ Action Potential من ال vagus nerve

* ويستخدم هذا الدواء في حالات ال Tachy

* جرعة كالية من ال B blocker توقف ال sympathitic

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

* ٢ يوجب QRS ويوجب Ventricle contraction



* وبما ان QRS لم تظهر فلن تظهر T wave

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

* Atrium ال
و ال Ventricle
كل واحد يعمل
على حدى

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.

* اذا اعطيناه
Ca blocker

تكلس او تضيق

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

*اذا كان جرعة عالية

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

غير مهم

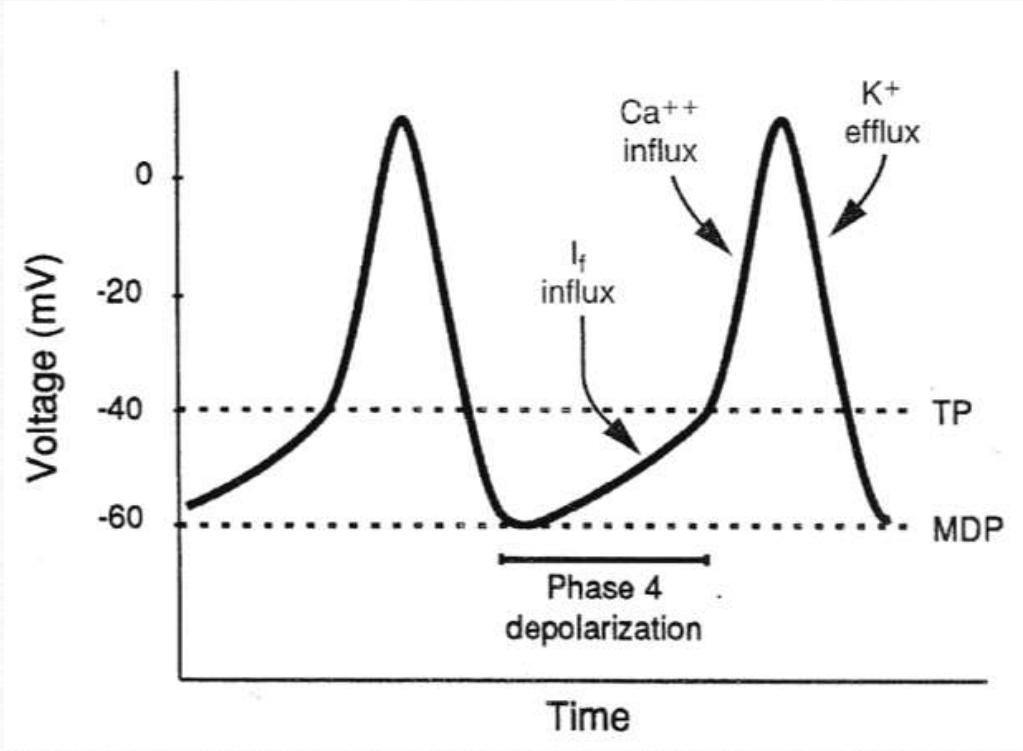
❑ Sinus rhythm:

صادرة

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:

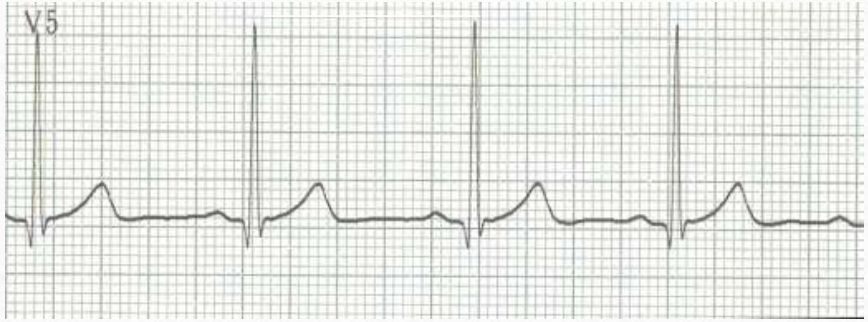
- Sinus node disorders.

- 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.

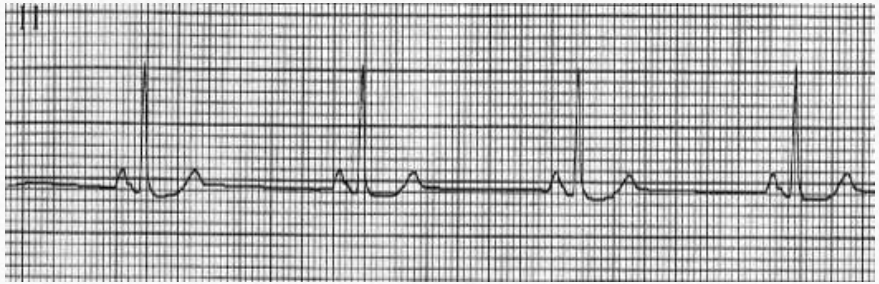
Normal Sinus Rhythm: 60 – 100 beats per minute (bpm)



Sinus cycle length=920 ms
Heart rate = 65 bpm

1

Sinus Bradycardia: < 60 bpm



Sinus cycle length=1400 ms
Heart rate = 43 bpm

2

* لم تتكلم الدكتورة عن الأرقام

bradycardia إذن في تأخير

إذن سيزداد الطول لـ ECG

1. Sinus bradycardia:

Causes: خارج القلب قلة درجة الحرارة

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

ارتفاع ضغط العين digoxin = digitalis

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

داخل القلب

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.**

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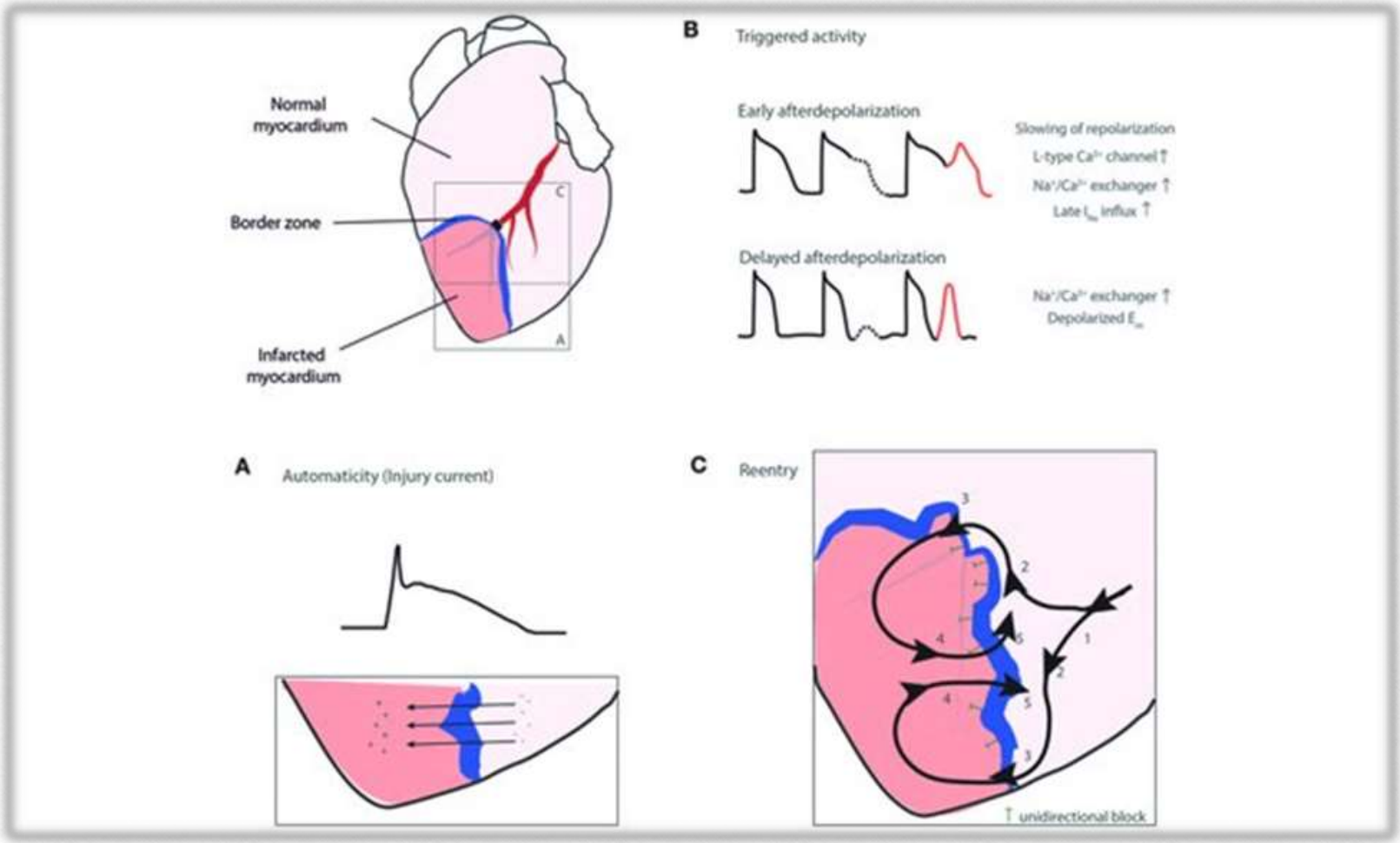
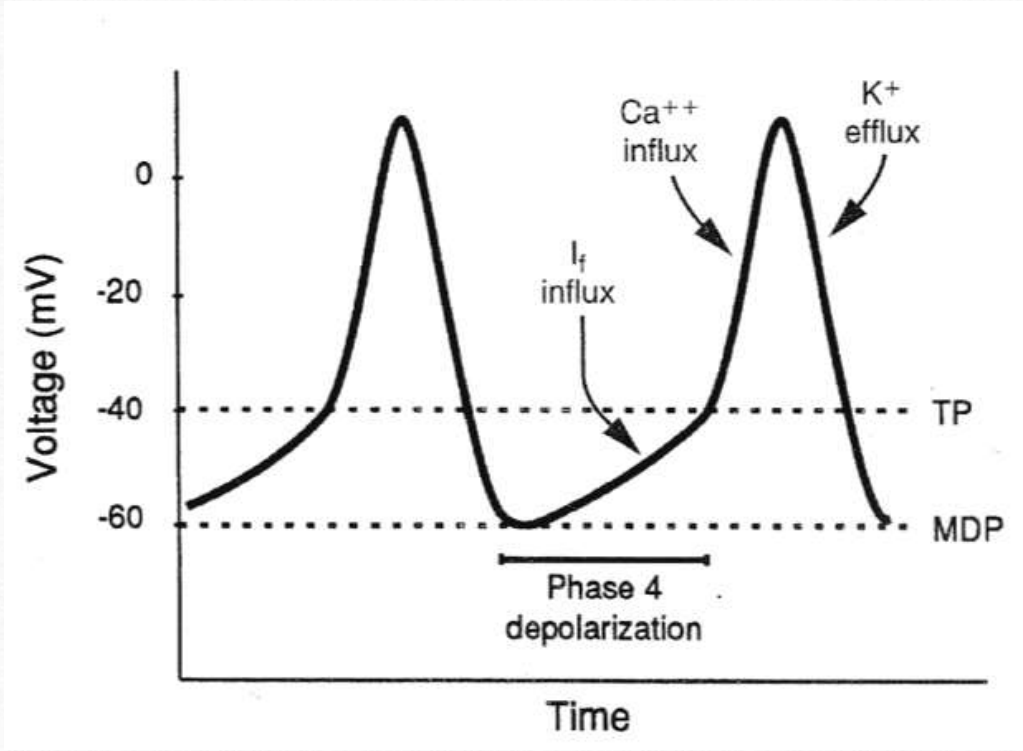


https://www.researchgate.net/publication/337023955_Ventricular_Arrhythmias_in_First_Acute_Myocardial_Infarction_Epidemiology_Mechanisms_and_Interventions_in_Large_Animal_Models/figures?lo=1

Normal Pacemaker Activity in the SA Node



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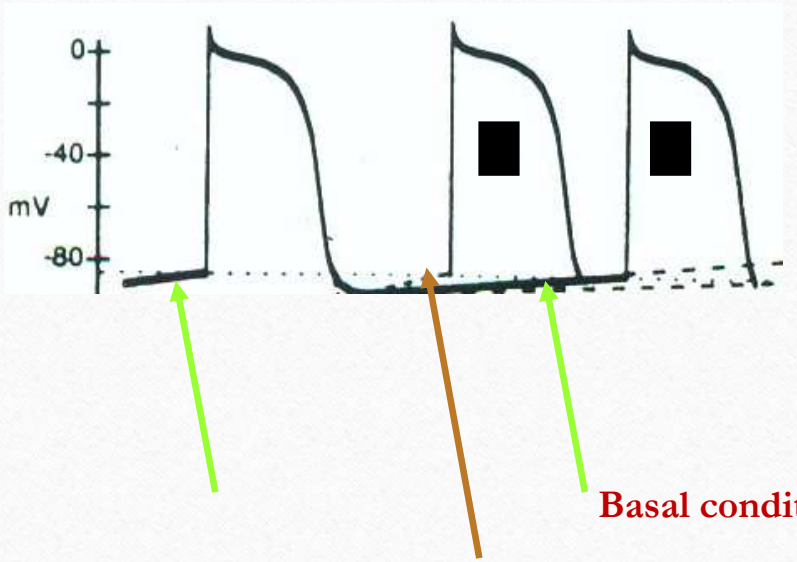
2. Sinus tachycardia

* تظهر كل ال waves
ولكن بشكل متتابع

Enhanced Normal Automaticity



Increased slope of phase 4 depolarization

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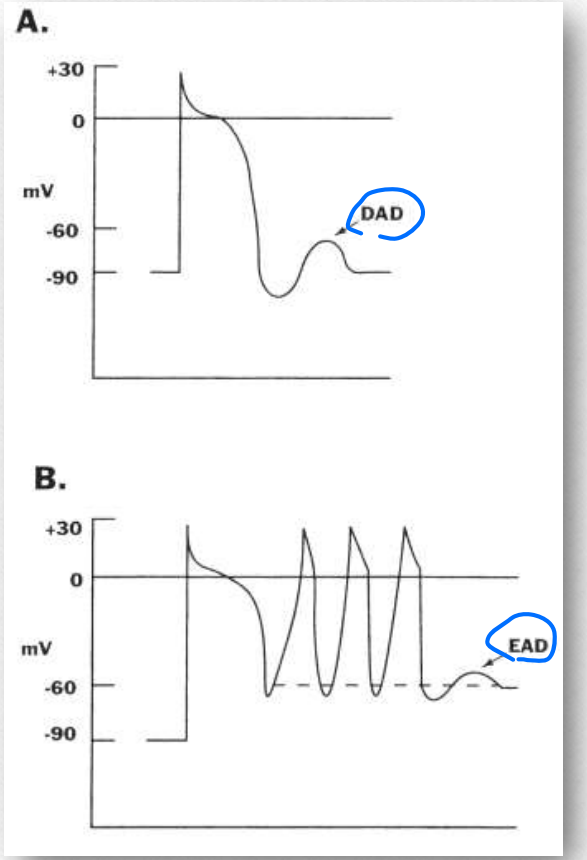
2. Sinus tachycardia

Triggered activity



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

تظهر بعد انتهاء الrepolarization

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

تظهر قبل انتهاء الrepolarization

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2. Sinus tachycardia

Reentry

- **Requisites:**

لیحصل عندنا الـ reentry یجب توفر مسارین او اکثر

≥ 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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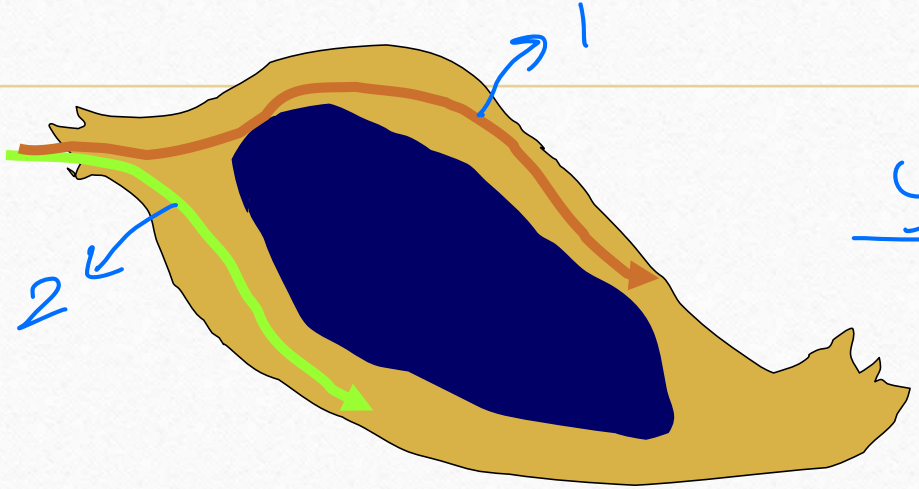
Requirements for Reentry



السلوك الطبيعي

Basal state – conduction over both pathways – wavefronts collide

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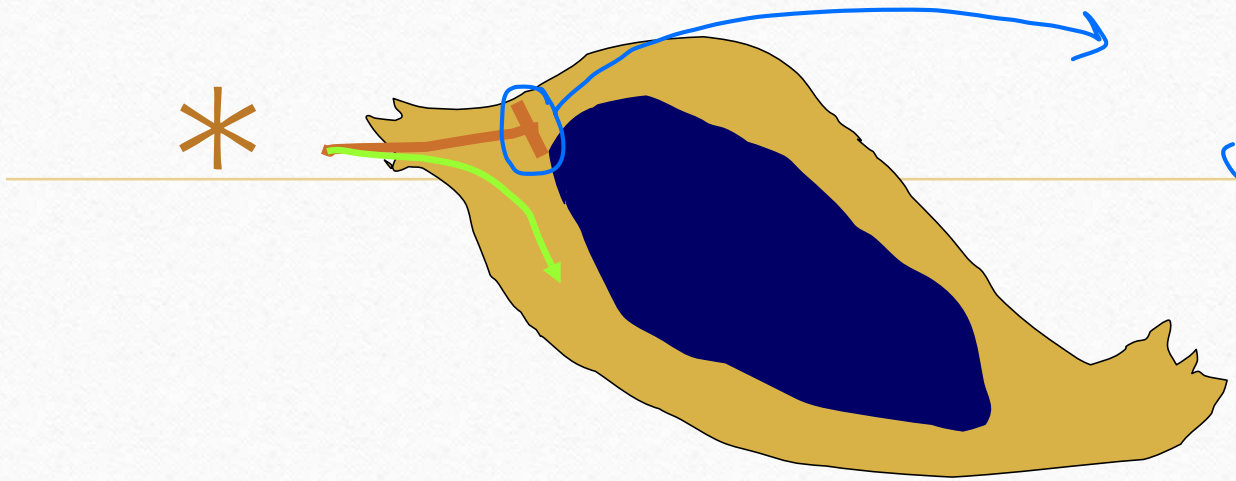
Initiation of Reentry



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

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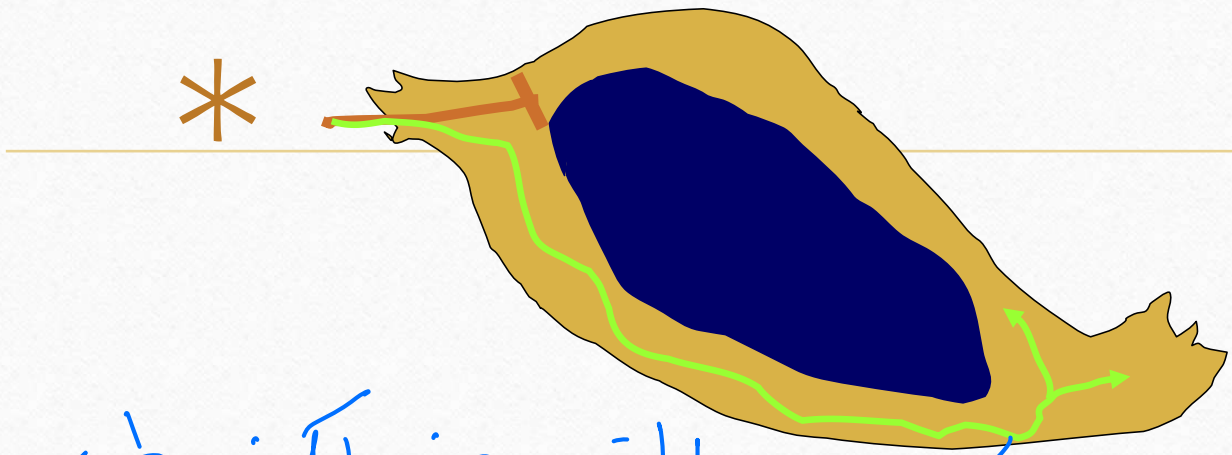
Initiation of Reentry



يقوم بالتعويض لكن ببطء

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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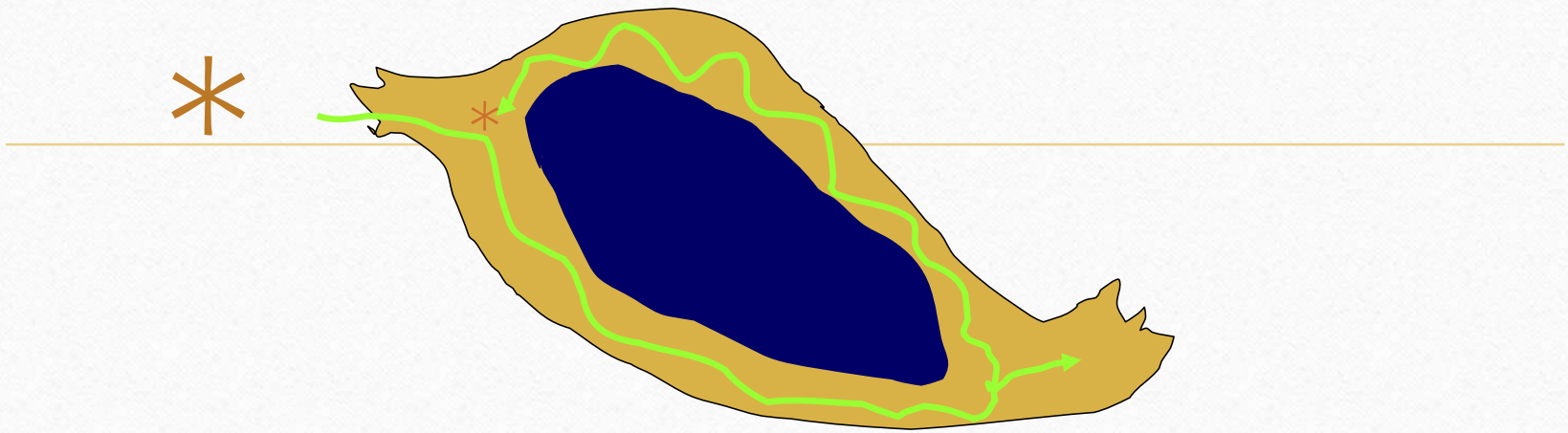
Initiation of Reentry



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 (حرارة), acute heart failure, B. chronic causes: pregnancy, anemia, hyperthyroidism, excess catecholamine. ECG: short R-R interval.

ضعف الدم سيحتاج رفع ضربات القلب للتعويض

epinephron
norepinephron

R-R = P-R

نبضة مفاجئة

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

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.

* الأسباب المحتملة
- جلطات
- تكلس
- التهاب
- دواء
- دخان، نيكوتين
- كافيين، قهوة

Possible causes of ectopic foci are:

AV node أو Purkinje

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.

* إذا حدثت الحالة لمرة واحدة مش مشكلة
أما إذا تكررت تصبح خطيرة وتحدث

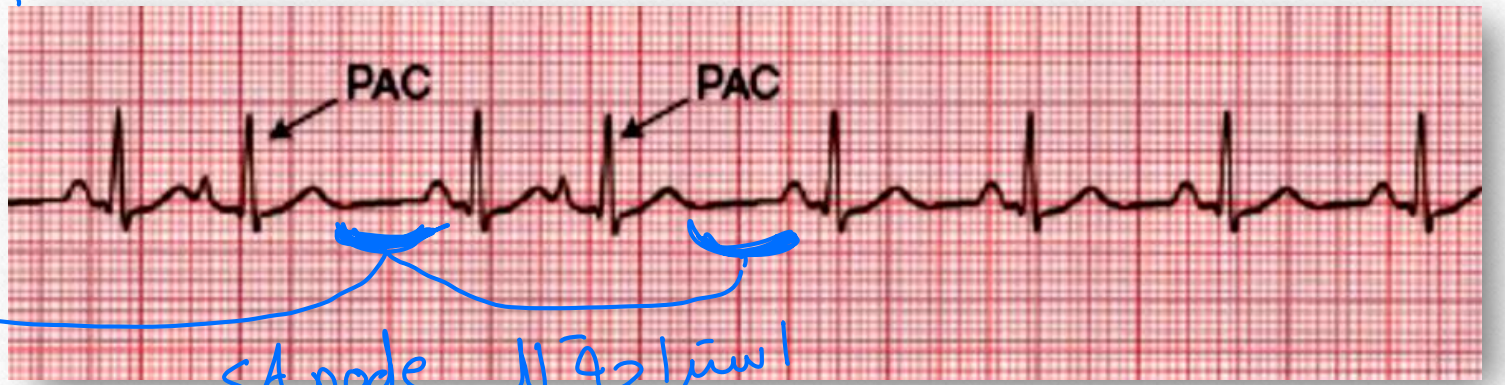
It could be:

1. **Atrial ectopic:** The ECG changes are:

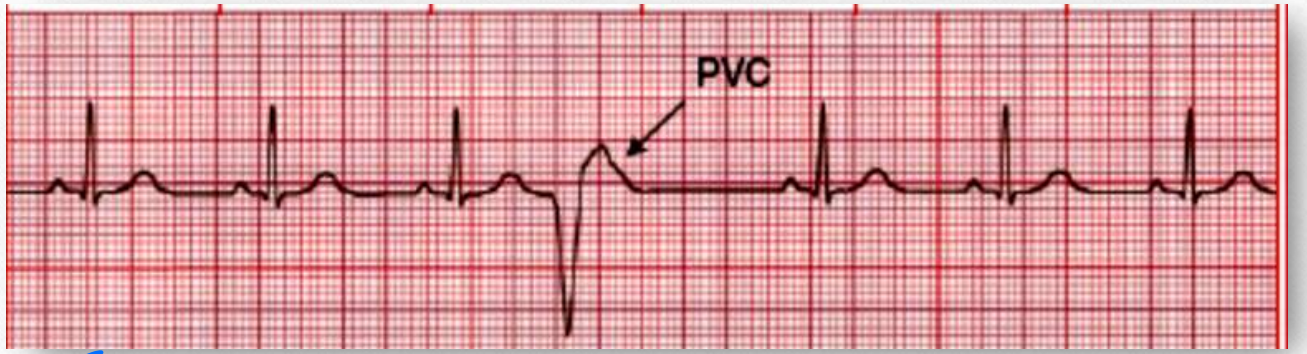
❶ The P wave of this beat occurs too soon in the heart cycle,

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

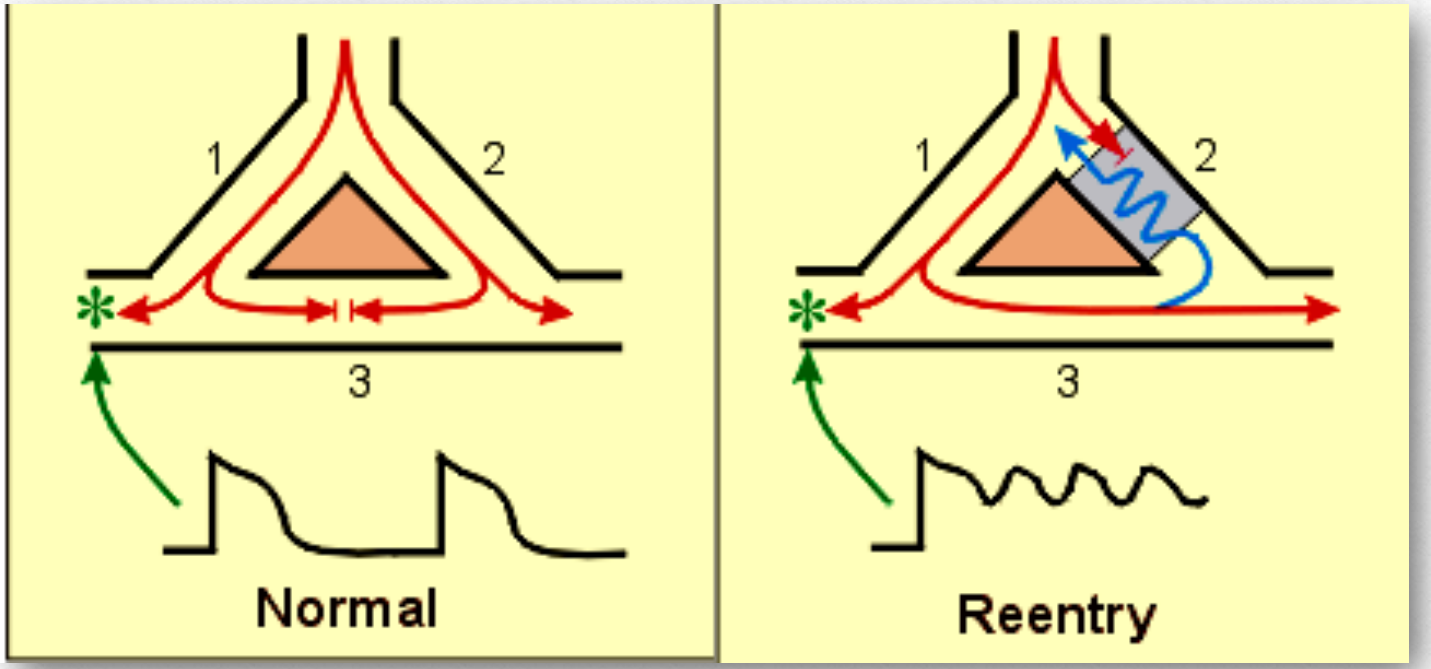
❸ The interval between the premature contraction and next succeeding contraction is slightly prolonged, which is called (compensatory pause).



2. ventricular ectopic:



بسبب قوتها تنعكس

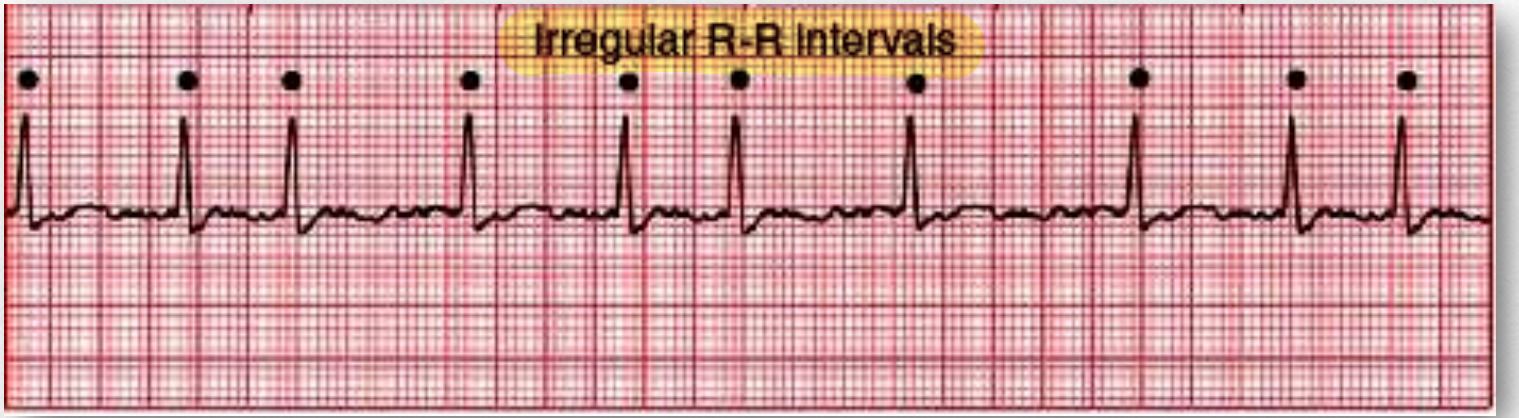


يسبب شيئين
-Atrial fibrillation
-ventricl fibrl
أخطر

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.

* الـ Atrium كتير قوي في الـ contraction
و الـ P-wave تختفي من الـ ECG

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



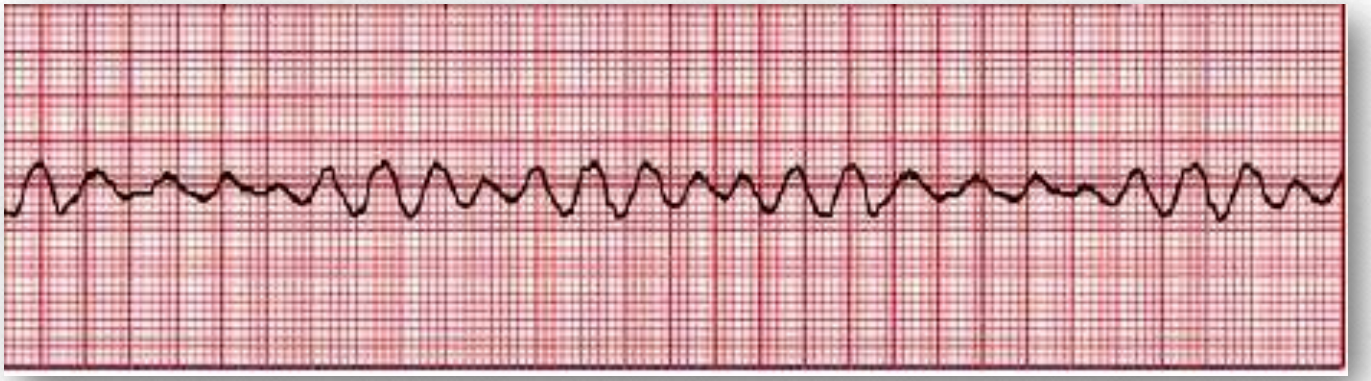
B. Ventricular fibrillation:

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.

* لا يوجد QRS وبالتالي لا يوجد T-wave

B. Ventricular fibrillation:

* فقط يظهر P-wave



* جداً خطير ويعتبر حالة طارئة وقد يؤدي للوفاة

❑ Anti-arrhythmic drugs:

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

*اصناف العلاج

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:

* علاجات ارتفاع نبضات القلب

Class I:

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

* كلهم ادوية بتقلل ال Heartrate عن طريق تحفيز ال Para وتثبيط ال symp

* الدكتورة ما بتركز كتير لانواع ندرسها بالتفصيل

* قالت الدكتورة بتركز على ال ClassII

Class II:

عكس ال symp

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:

β blocker

لتنتهي بـ lol

a. Propranolol [Inderal],
b. Atenolol,
c. Metoprolol
d. Bisoprolol
e. Sotalol

Class III:

* زيادة فاعلية فترة الـ refractory Period مما يؤدي لـ تنظام نبضات القلب ونقصانها

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.

* تقليل الـ deporalization من طريق الـ Na و K

- 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:

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)

low theraptic index

the drug must be dosed carefully and the person receiving the drug should be monitored closely for any signs of drug toxicity.

* كلاجات انخفاض ضربات القلب

❑ Treatment of Brady-arrhythmia:

1. Atropine

* تحفيز الـ Para وتثبيط الـ Symp

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

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

2. Isoproterenol [Isuprel]

تحفيز الـ Symp

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