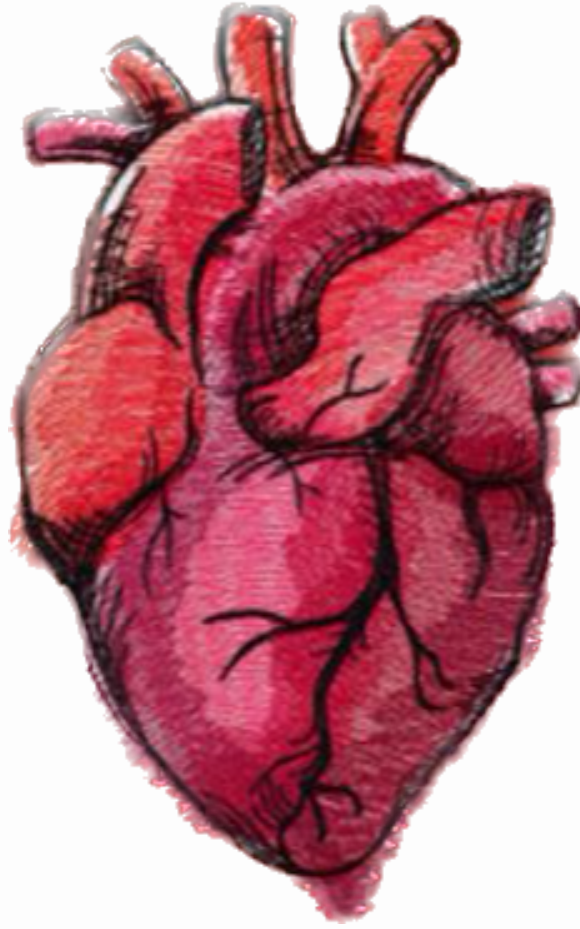




CARDIOVASCULAR SYSTEM



SUBJECT : _____

LEC NO. : 2

DONE BY : Tabark Aldaboubi, Raneem Azzam



اعترفوا يا سادة
لا تستطيعون العيش من دوني

ما تنسوننا من دعواتكم حياة
وَقُلْ رَبِّ زِدْنِي عِلْمًا

Arrhythmias

- The normal heart beats is regular.
- Arrhythmias and conduction disorders are caused by **abnormalities in the generation or conduction of the heart electrical impulses or both.**

• Causes:

1. Heart disorders:

- Including **congenital abnormalities of structure** e.g., **accessory atrioventricular connection**
- **Function** (e.g., hereditary ion channelopathies)

ارتبط (Ventricle و atrium) لربهم وارتبطوا متصلين ابدا
بها في الحالة يكونوا متصلين

مشكلة باد Channel ↓

يكون في مشكلة بالقلب عند الاطفال
abnormal (Na و K و Ca) → Action Potential
بالتالي يغير الـ Potential

2. Systemic factors:

- **Electrolyte abnormalities** (particularly low potassium or magnesium), hypoxia
- **Hormonal imbalances** (e.g., hypothyroidism, hyperthyroidism)
- **Drugs and toxins** (e.g., alcohol, caffeine).

Common in thale →

Tachycardia يعمل ↓

Very important to correct (Potassium)
لأنه مهم للقلب



Arrhythmias

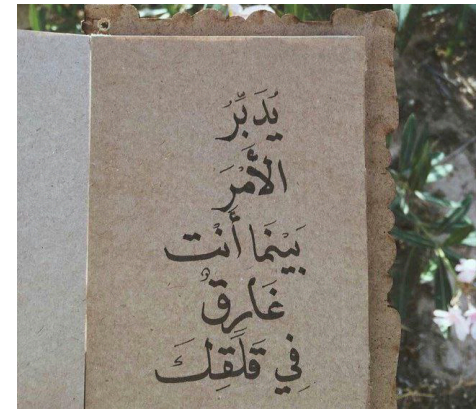


NB: This video is for you to understand arrhythmias and it is not required for the exam.

- <https://youtu.be/6LrptveKYus>

* موجود الفيديو على التيمز
شرح لارتواع الـ Arrhythmia

Arrhythmias



Symptoms of Cardiac Arrhythmia

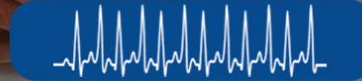
* Slow Heartbeat



Symptoms include:

Fatigue, palpitations, wooziness, dizziness, fainting, unconsciousness or even sudden death

* Fast Heartbeat



Symptoms include:

Palpitations, heart, chest pain, dizziness, risk of heart failure and sudden death

Antiarrhythmic drugs

Three Primary Indications for Treatment of Cardiac Arrhythmias

1. Arrhythmias that **decrease cardiac output** (e.g., severe bradycardia, ventricular tachycardia or fibrillation)
Oxygenation to the Tissue نقل ال
stroke volume ← *cardiac output* تنقل ال
Cardiac output هو عبارة عن ال *heart rate* مع ال *stroke volume* اذا قل *HR* ينقل ال *Cardiac output* حركتنا بالمسيو انه ال
2. Arrhythmias that are likely to **precipitate more serious arrhythmias** (e.g., atrial flutter may lead to sustained ventricular tachycardia)
Cardiac output بالدياب ما يتكون بجاش على ال
Cardiac output وما يتكون خطيرة ف لازم اعالجها قبل ما تفسد خطير
3. Arrhythmias that are likely to **precipitate an embolism** due to creation of vascular stasis (e.g., chronic atrial fibrillation)
embolism بعضيهم *anticoagulating* كما ان عشان. يضر ال

Antiarrhythmic drugs

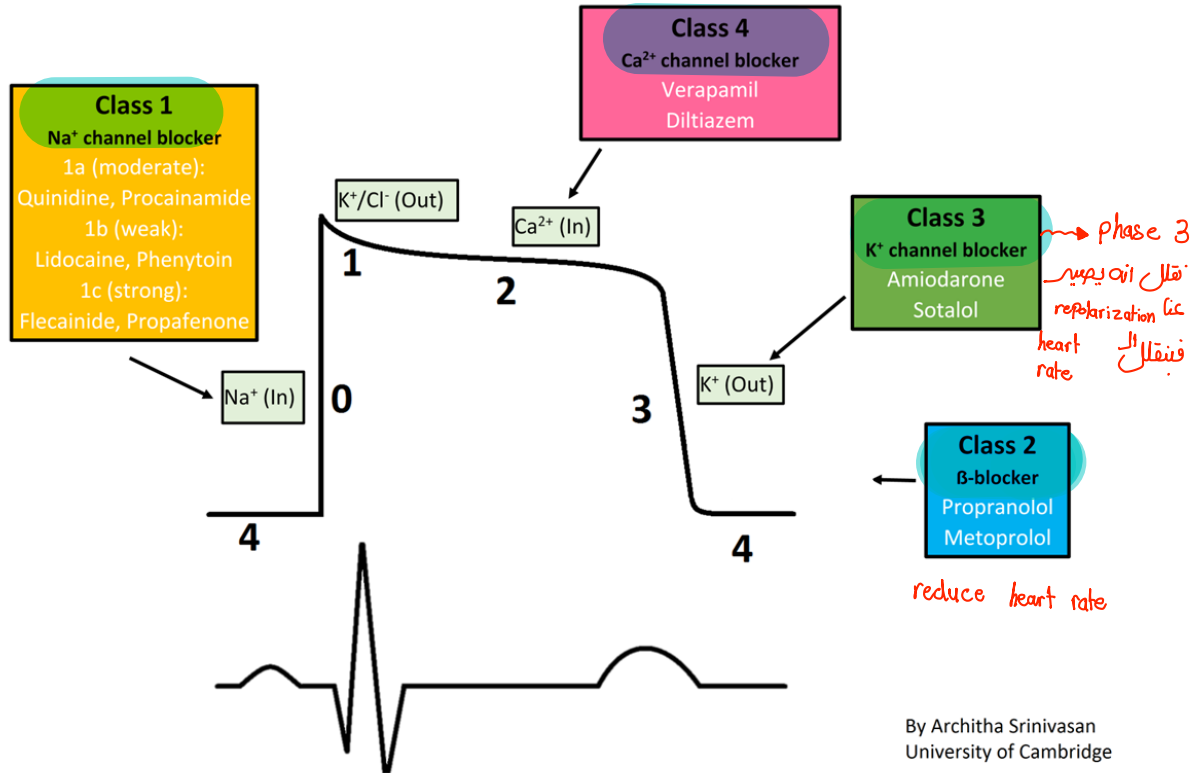
While drug therapy is still the most common method for treating arrhythmias, other non-pharmacological therapies are also in current use. Which includes:

في بعض الحالات ما يعطيها دواء مثل ↴

1. DC cardioversion, implanting of a pacemaker, or defibrillator device (ICD)
إذا عنده فستمن يركبوا Pacemaker وقتها يبطل ال AS node اللي يهمل ال AP ونا ال Pacemaker اللي ركبتوا
2. Carotid sinus massage (↑ vagal tone) ↴
بالتالي ال heart rate ينزل
3. Surgical or catheter-mediated ablation of an ectopic focus, coronary bypass surgery →
إذا كان عند المريض Atherosclerosis يكون جزء من القلب ما فيه ال electrical signal يبطل فيه refractory period بتعتبر تروح على Tissue مش لازم تروح عليها بتدخل ب cycle وبصير فيه extra beat
4. Lifestyle modification (avoiding events that aggravate an arrhythmia - e.g., exertion, emotional stress, non-ideal diet)

Antiarrhythmic drugs

التقسيمات على حسب وظيفتها

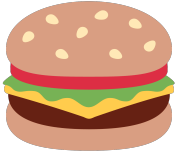




Antiarrhythmic drugs



Antiarrhythmic Drugs



هسا مع
اختصارات الاكل
كلنا بنصير
شاطرين بالحفظ
👉🤔🤔

Class Ia

1 Double Quarter Pounder

Disopyramide

Quinidine

Procainamide

Class Ib

with Lettuce, Mayo

Lidocaine

Mexiletine

Class Ic

برغر بدون بطاطا ما بنفع 🤔

Fries Please!



Flecainide

Propafenone

Class II

Beta blockers? Lol

Propranolol

Atenolol

Metoprolol

Class III

This is SAD

Sotalol

Amlodarone

Dofetilide



ما تتحمسوا وتزعلوا خليككم ميسوتين 🤔

Class IV

I and V in Class IV?

Diltiazem

Verapamil

رج نوحدهم بأكثر
عن مرهني

المشغلين
اللي موتا بجات
ولا لواحد من
بقية ال Class

Class I Antiarrhythmic Drugs

- Act by blocking **voltage-sensitive Na⁺ channels**.
- They **bind more rapidly to open or inactivated Na⁺ channels** than to channels that are fully repolarized.
- These drugs have **use dependence (or state dependence)** property as show a greater degree of blockade in tissues that are frequently depolarizing. Which enables these drugs **to block cells that are discharging at an abnormally high frequency, without interfering with the normal beating of the heart.**

لما يصير ال AP ال Na Channel تبكون مسخرة ولما يصير depolarization
بتفتح بعد ما تفتح بفترة بصير لها inactivation بعد Phase 0 بتغير شكل ال Channel تبطل تدخل ال Na بس بنفس الوقت ما يتسع انما تفتح صحت لو اجا AP حيلو .

بالناس الي عندهم
Arrhythmia
هاي ال channel
بتفتح اكثر
لهيك بجلي
use dependance

↓
بشتغل الدوا على ال channel الي موجودة بلا cell الي فيها Abnormality

Class I Antiarrhythmic Drugs

- They have proarrhythmic effects, particularly in patients with reduced ^② left ventricular function and atherosclerotic heart disease.
اذا افطوا لبعض المرضى ممكن تعمل عندهم Arrhythmia ثانية اخطر (مين هذوا المرضى!) الى عندهم ↑
- Class I drugs are further subdivided into three groups according to their effect on the duration of the cardiac action potential.

CLASSIFICATION OF DRUG	MECHANISM OF ACTION	COMMENT → <i>MOA ما الهم دمل بال Side effect ممكن الهم عارضة بال</i>
IA	Na ⁺ channel blocker	Slows Phase 0 depolarization in ventricular muscle fibers
IB	Na ⁺ channel blocker	Shortens Phase 3 repolarization in ventricular muscle fibers
IC	Na ⁺ channel blocker	Markedly slows Phase 0 depolarization in ventricular muscle fibers

ما تخافو من معلومات الفسيو الاسئلة بالامتحان إن شاء الله رح تكون واضحة مافيها شي مش واضح

Class I Antiarrhythmic Drugs

+ حبه وين بتشغل؟

Bind to and block the **fast Na channels** in **non-nodal tissue** (e.g., myocytes of the atria and ventricles, His-Purkinje system):

SA node
AV node

شوبير؟

• Blocking fast Na channels ↑:

- ↓ Slope of phase 0 → ↓ in the amplitude of action potential = ↓ velocity
- ↓ Velocity of action potential: **Transmission within the heart (↓**

conduction velocity)

A.P. ينتقل من myocytes from one cell to another يعني رح يقل ،،،، مثلا، اذا كان 1 cell فيها 50 A.P رح ينتقل
اللي بعدها ف يصير عن 10 cell رح يوصلها 20 A.P من ال 50 لانو صارلها slow down ف في cell مارح يصيرلها conduction

- Important mechanism for suppressing tachycardias caused by abnormal conduction (e.g., reentry mechanisms)
- Reentry mechanisms can be interrupted by ↓ abnormal conduction

ارجع شوف
الفديو

بعض ال tissue فيها accessory node بطلع منها فايرنغ، او ممكن تمشي في دائرة مختلفة يصيرلها reentry mechanism تدخل
في continuous activation تزيد النبضات بدون ما يكون عنا initiation من (SA, AV node)، ف انا هون لما نقلل الكونداكشن من
myocyte ل myocyte منقلل ال HR الي مش لازم يصير (يقال ال abnormal conduction)

Class IA Antiarrhythmic Drugs

1. Quinidine is the prototype class IA drug.
2. Procainamide
3. Disopyramide

* يعني يشتغل على هيريتة I و III

- ❖ They have concomitant class III activity (K channel blockers).
- ❖ They can cause **arrhythmias that can progress to ventricular fibrillation.**

← شأن هيك ونستفهم في بعض الحالات مشه كلل امر خبي

Class IA Antiarrhythmic Drugs

Mechanism of action:

I • Na channel effects:

- Intermediate speed of binding and dissociation from voltage-gated Na channels

III • Slows the upstroke of action potential and conduction

• K channel effects:

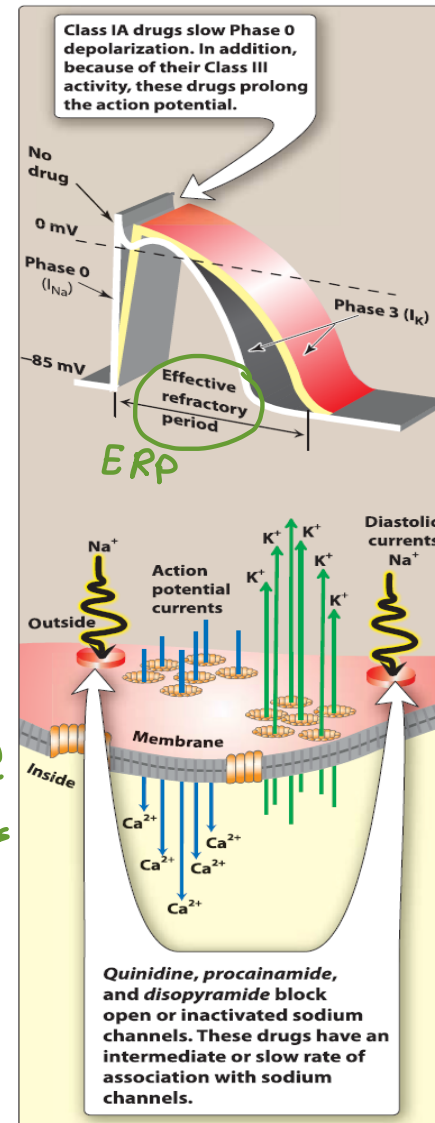
- Blocks K channels \rightarrow \downarrow K efflux \rightarrow slows repolarization
- Leads to \uparrow ERP and action potential duration \rightarrow QT prolongation

• Other effects:

- Anticholinergic activity \rightarrow can \uparrow sinoatrial rate and atrioventricular conduction (\downarrow Myocardial contractility)

بتحفز ال SA node بتالي بتزود ال HR

سبب \uparrow HR ال يقل



الخروج من الغلابة

يعني

بتطول effective refractory period

هسا بدنا نحكي عن ال ERP منطقيا in general لما تطول بصير عنا slowing of A.P بالتالي يقل HR وهاد مش دايمًا، ممكن ERP ممكن تطول شوي او ما تتغير بس يقل ال HR اليه؟؟ لانو حكينا قبل شوي عنا ميكانيزم ثانية وهي ال conduction، ركزوا الله يرضا عليكم، لانو الدكتوروة بدناش نربط انها الايشي الوحيد الي بتقلل النبض

يؤدي

بدون ال HR

conduction، ركزوا الله يرضا عليكم، لانو الدكتورَة بدناش نربط انها الايشي
الوحيد الي بتقلل النبض

• Other effects:

- Anticholinergic activity → can ↑ sinoatrial rate and atrioventricular conduction (↓ Myocardial contractility)
- بدون Anti ↓ HR
بتحفز ال SA node بتالي بتزود ال HR
سبب ↑ HR في نظام

*هسا هون ركزت الدكتورَة نعرف انو ال anticholinergic هو سايد ايفكت لادوية ال class 1 مشان هيك ممكن تعمل عنا proarrhythmia وممكن تعمل عنا مشاكل لانو عندها anticholinergic

إذا ... (class I A drugs) → has anticholinergic activity that might ↑ SA Rate

تذكر ك' صاير ايفكيت



Class IA Antiarrhythmic Drugs

الوحيد الذي يستعمل على α

- **Quinidine** has mild α -adrenergic blocking and anticholinergic actions.
- **Procainamide** has no α -adrenergic blocking and less anticholinergic activity than quinidine
- **Disopyramide** has no α -adrenergic blocking and more anticholinergic activity than quinidine. It produces a greater negative inotropic effect, and unlike the other drugs, it causes peripheral vasoconstriction.

ليه بعملها؟ لانو ما عندو α -Adrenergic blocking

**أكدت الدكتورة مارح تصعب الاسئلة
احفظو الشغلات الmajor واتظمنو



بدنا نركز على الي بالمحاضرة الجاي ونهتم بالي رح
نناقشهم هسا واحفظوهم مزبوط في جدول المحاضرة

Class Ia

1 Double Quarter Pounder

Disopyramide

Quinidine

Procainamide

Class IA Antiarrhythmic Drugs

• Therapeutic uses:

1. **Quinidine**: atrial, AV junctional, and ventricular tachyarrhythmias.
2. **Procainamide**: to treat acute atrial and ventricular arrhythmias but it is not often use as it is replaced by electrical cardioversion or defibrillation and amiodarone
3. **Disopyramide** : Ventricular tachyarrhythmias and atrial fibrillation and flutter (not first choice)

• **Contraindications**: patients with atherosclerotic heart disease or systolic heart failure. **

• **Sides effects**: Large doses of **quinidine** may induce the symptoms of cinchonism (blurred vision, tinnitus , headache , disorientation, and psychosis).

Commonly use

Class IB Antiarrhythmic Drugs

- Lidocaine and Mexiletine
- Mechanism of action :

1. Na⁺ channel blockade: bind primarily to channels in the inactivated state.

Very useful for arrhythmias in **ischemic myocardium**: because ischemia leads to slow cellular depolarization that inactivates sodium channels, and therefore enhanced binding of IB drugs.

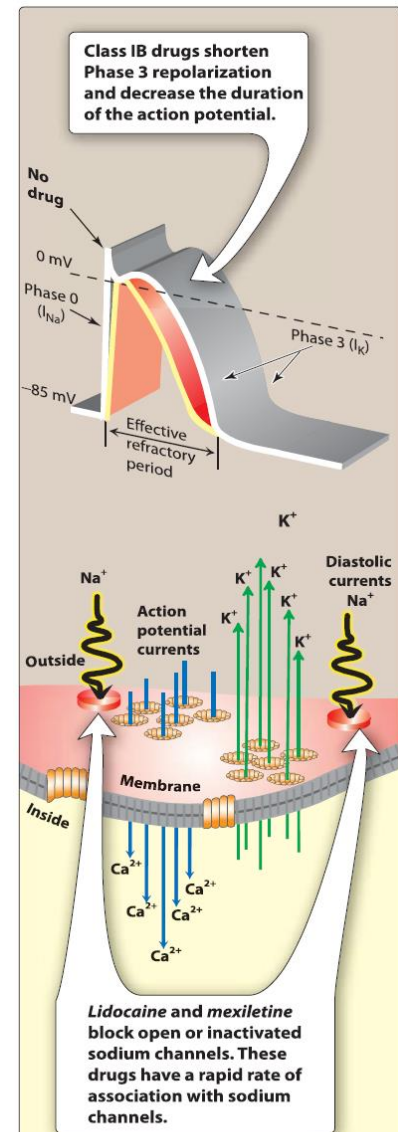
أعطى

→ Mechanism → داليل الو

2. ↓ Velocity of action potential: **Transmission within the heart (↓ conduction velocity)**

Shorten phase 3 repolarization and decrease the duration of the action potential → بصير فيه ب وعا الو علاته. Mechanism

• Neither drug contributes to negative inotropy. → بس بتقلو على ال tissue ال فيه ischemia



Class IB Antiarrhythmic Drugs

- Therapeutic uses:

- **Lidocaine:** *→ in acute*

1. As an alternative to amiodarone ventricular fibrillation or ventricular tachycardia (VT).
2. May also be used in combination with amiodarone for VT storm.

- **Mexiletine:** *↪ in*

Is used for chronic treatment of ventricular arrhythmias, often in combination with amiodarone.



Class IB Antiarrhythmic Drugs

- Adverse effects:
- **Lidocaine:** Central nervous system (CNS) effects include nystagmus (early indicator of toxicity), drowsiness, slurred speech, paresthesia, agitation, confusion, and convulsions.
- **Mexiletine:** Nausea, vomiting, and dyspepsia.

Class Ic

Fries Please!

Flecainide

Propafenone

Class IC Antiarrhythmic Drugs

- Flecainide and propafenone
- Mechanism of action:
- **Suppresses phase 0 upstroke in Purkinje and myocardial fibers** (slowing of conduction in all cardiac tissue) → Like groups A+B
- Automaticity is reduced by an increase in the threshold potential

on Na⁺ channel

less firing ف بصير

↓
set
عنا

هاي المرة المية لدو علي برجع و بقول ركزو على الجدول للمحاضرة الجاي
ومرو على هالسلايدات قراءة بسسسس والدكتورة مارح تجيب شغلات ديبيب

Class Ic

Fries Please!

Flecainide

Propafenone

لا قوا نهم كلام

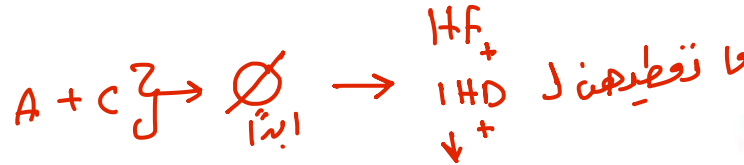
Class IC Antiarrhythmic Drugs

- Therapeutic uses:
- **Flecainide**
 1. Maintenance of sinus rhythm in atrial flutter or fibrillation
 2. Treating refractory ventricular arrhythmias.
- **Propafenone**: Restricted mostly to **atrial arrhythmias**
 1. Rhythm control of atrial fibrillation or flutter and paroxysmal supraventricular tachycardia
 2. Prophylaxis in patients with AV reentrant tachycardias.

Fries Please!

Class IC Antiarrhythmic Drugs

Flecainide and propafenone



Flecainide
Propafenone

- Due to their negative inotropic and proarrhythmic effects, use of these agents **is avoided in patients with structural heart disease (left ventricular hypertrophy, heart failure, atherosclerotic heart disease).**

- Automaticity is reduced by an increase in the threshold potential

less firing ف بصير less firing
 ↓
 بصير
 عينا

Fries Please!

Flecainide

Propafenone

Class IC Antiarrhythmic Drugs

Adverse effects:

- **Flecainide** :blurred vision, dizziness, and nausea ✓
- **Propafenone**: blurred vision, dizziness, nausea and may cause bronchospasm and should be avoided in **patients with asthma**.

* **Fries please ,don't give propafenon to asthma patients!!**

حفظوا لهم امين