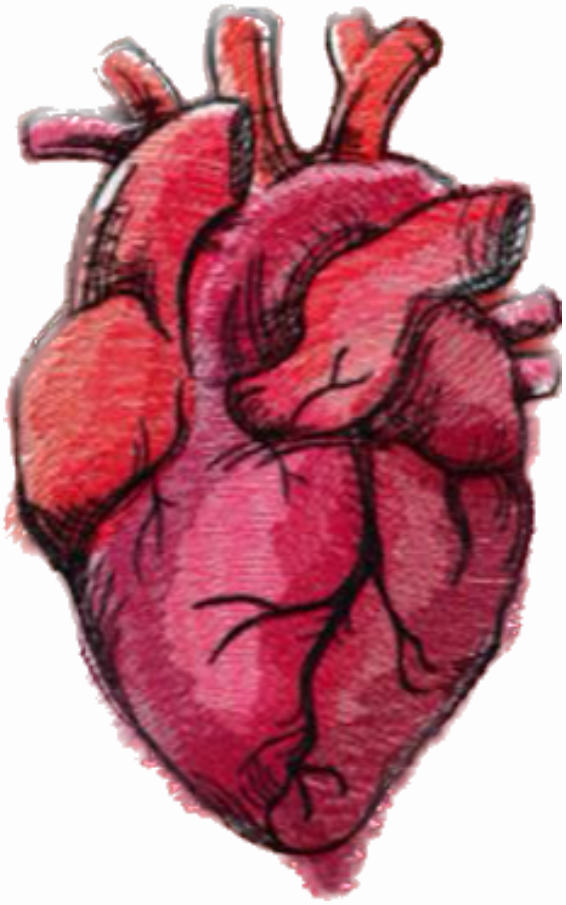
النادي الطبي

CARDIOVASCULAR SYSTEM



SUBJECT : Antiarrhythmics 2

LEC NO. : Summary L3

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وقل رب زدني علماً

Class II Antiarrhythmic Drugs (β-blockers)

When were used as antiarrhythmic they are cardio selective

Mechanism of Action

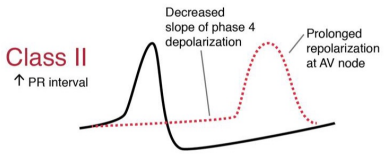
Decrease slope of phase 4 depolarization — Reaching the threshold, harder

Diminish phase 4 depolarization, depress automaticity, prolong AV conduction, decrease heart rate and contractility.

Therapeutic Uses

1. Treating tachyarrhythmias caused by increased sympathetic activity.
2. Atrial flutter/fibrillation, AV nodal reentrant tachycardia.
3. Prevent life-threatening ventricular arrhythmias post-MI.

One of the complications of myocardial infarction is ventricular arrhythmias



Drugs

β-receptors → heart → ↑HR; bronchi → bronchodilation

selective

- Metoprolol (most widely used) — most widely used β -blocker for the treatment of cardiac arrhythmias. Compared to nonselective β -blockers, such as propranolol it reduces the risk of bronchospasm.
- Esmolol (fast-acting for IV administration) — very short and fast-acting β -blocker>>> used for intravenous administration in acute arrhythmias that occur during surgery or emergency situations.

Adverse Effects

Bradycardia, hypotension, fatigue.

Class III Antiarrhythmic Drugs

All class III drugs have the potential to induce arrhythmias(proarrhythmic)

Mechanism of Action

Prolonged Repolarization

Block K+ channels, prolonging action potential duration and refractory period. increasing refractoriness

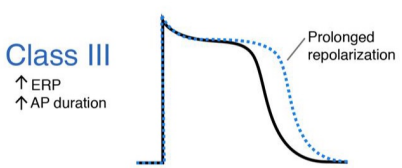
without altering phase 0 of depolarization or the resting membrane potential.

Subtypes

A) Amiodarone

B) Sotalol

C) Dofetilide



Amiodarone

Mechanism of Action

Complex effects: Class I, II, III, and IV actions, α-blocking activity. Prolongs action potential duration and refractory period by blocking K+ channels.

Therapeutic Uses

Effective in severe refractory supraventricular and ventricular tachyarrhythmias. Mainstay for rhythm management of atrial fibrillation/flutter.

ease.

Adverse Effects

Pulmonary fibrosis, neuropathy, hepatotoxicity, corneal deposits, optic neuritis, blue-gray skin discoloration, hypo- or hyperthyroidism. Least proarrhythmic among class I and III antiarrhythmics.

Sotalol

Description

Class III antiarrhythmic agent with nonselective β-blocker activity.

Mechanism of Action

Blocks rapid outward K+ current (delayed rectifier current).

type of potassium ion current involved in the repolarization phase (phase 3) of the cardiac action potential. helps to restore the cell's membrane potential to its resting state following depolarization.

Therapeutic Uses

لحتى dsotalol بنعطي
remodelling نقلل ال

patients with left ventricular hypertrophy or atherosclerotic heart disease.
ممكن يصير عندهم remodelling (يتحول myocytes الى fibrous tissue غير فعالة مثا رح يعمل pumping للدم)

Used for maintaining sinus rhythm in atrial fibrillation/flutter, refractory paroxysmal supraventricular tachycardia, and treating ventricular arrhythmias. Commonly used in patients with left ventricular hypertrophy or atherosclerotic heart disease.

For his β -blocking properties

Precautions

- To reduce the risk of proarrhythmic effects, sotalol should be initiated in the hospital to monitor QT interval.

Dofetilide

Description

Pure K+ channel blocker.

Therapeutic Uses

Can be used as a first-line antiarrhythmic agent in patients with persistent atrial fibrillation and heart failure, or those with coronary artery disease.

Precautions

in hospital

Initiation limited to the inpatient setting due to the risk of proarrhythmia.

Class IV Antiarrhythmic Drugs (Ca2+ channel blockers)

- Although voltage sensitive Ca2+ channels occur in many different tissues, the major effect of Ca2+ channel blockers is on vascular smooth muscle and the heart.

Mechanism of Action

Low effective on normal cells — Highly effective on more exited tissue(use dependent)

Bind to open depolarized voltage-sensitive Ca2+ channels, decreasing inward current carried by Ca2+.

Therapeutic Uses

Don't give Ca2+ channel blockers to patients with heart failure because it reduces heart contractility

Treating reentrant supraventricular tachycardia, reducing ventricular rate in atrial flutter/fibrillation.

Drugs

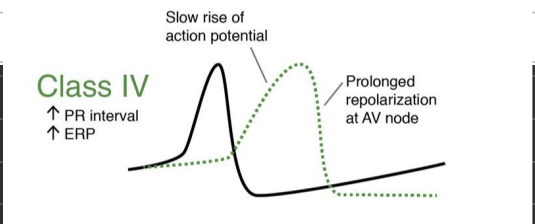
- Non-dihydropyridine Ca2+ channel blockers
  - Verapamil
  - Diltiazem

- These drugs are use dependent as they prevent repolarization until the drug dissociates from the channel, resulting in a decreased rate of phase 4 spontaneous depolarization.
- They also slow conduction in tissues that are dependent on Ca2 currents, such as the AV and SA node.

Adverse Effects

Bradycardia, hypotension, peripheral edema.

Calcium channel blockers can have negative inotropic effects, meaning they reduce the strength of the heart's contractions. In systolic heart failure, where the heart's pumping function is already impaired, further weakening of cardiac contractility can exacerbate symptoms and worsen heart failure.



Other Antiarrhythmic Drugs

Digoxin

Mainly used in heart failure

- Inhibits Na+/K+-ATPase pump, shortening refractory period in atrial/ventricular Myocardial cells while prolonging the effective refractory period and diminishing conduction velocity in the AV node.
- Controls ventricular response rate in atrial fibrillation/flutter. however, sympathetic stimulation easily overcomes the inhibitory effects of digoxin.
- Causes ectopic ventricular beats at toxic concentrations that may result in VT and fibrillation.

Adenosine

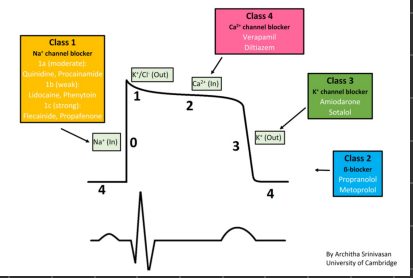
- Decreases conduction velocity, prolongs refractory period, decreases automaticity in AV node. At high doses
- Drug of choice for converting acute supraventricular tachycardias. Intravenous adenosine
- Causes flushing, chest pain, hypotension. short duration of action and fast onset بسبب

Magnesium Sulfate

- Slows SA node impulse formation, prolongs conduction time.
- Used to treat torsades de pointes, digoxin-induced arrhythmias. Used in patients with long QT interval

CLASSIFICATION OF DRUG	MECHANISM OF ACTION	COMMENT
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
II	β-Adrenoreceptor blocker	Inhibits Phase 4 depolarization in SA and AV nodes
III	K^+ channel blocker	Prolongs Phase 3 repolarization in ventricular muscle fibers
IV	Ca^{2+} channel blocker	Inhibits action potential in SA and AV nodes

Antiarrhythmic drugs



This common arrhythmia involves multiple ectopic foci of atrial cells, creating a chaotic movement of impulses through the atria. The ventricular response may be rapid (100–150 beats per minute) and irregular. Cardiac output is decreased and exercise intolerance is common.

β-Blockers are used in atrial fibrillation or flutter, because they decrease heart rate and promote conversion to sinus rhythm. Long-term, oral anticoagulant therapy reduces the risk of stroke that is associated with atrial fibrillation or flutter.

TYPE OF ARRHYTHMIA	ANTIARRHYTHMIC DRUGS				
	Class I	Class II	Class III	Class IV	Other
ATRIAL ARRHYTHMIAS					
ATRIAL FLUTTER		Metoprolol		Verapamil	Digoxin
ATRIAL FIBRILLATION	Propafenone	Metoprolol	Amiodarone, Dofetilide	Diltiazem	Anticoagulant therapy, Digoxin
SUPRAVENTRICULAR TACHYCARDIAS					
AV NODAL REENTRY		Metoprolol		Verapamil	Digoxin
ACUTE SUPRA-VENTRICULAR TACHYCARDIA				Diltiazem	Adenosine
VENTRICULAR TACHYCARDIAS					
ACUTE VENTRICULAR TACHYCARDIA	Lidocaine		Amiodarone		
VENTRICULAR FIBRILLATION (not responding to electrical defibrillation)	Lidocaine		Amiodarone		Epinephrine

دائمًا بنعطي anticoagulant مع atrial في علاج anti arrhythmic fibrillation

Conduction is slowed through the AV node with *metoprolol, verapamil, or digoxin.*

Implantable cardioverter defibrillators are commonly used to terminate ventricular arrhythmias.

بنعطيه مع anti arrhythmic حتى نمنع cardiac arrest

beginning with

This arrhythmia is a common cause of death in patients who have had a myocardial infarction. Cardiac output is impaired, and tachycardia may deteriorate into ventricular fibrillation. Therefore, ventricular tachycardia requires prompt management.

Key: Drug name — Commonly used drugs; Drug name — Alternative drugs