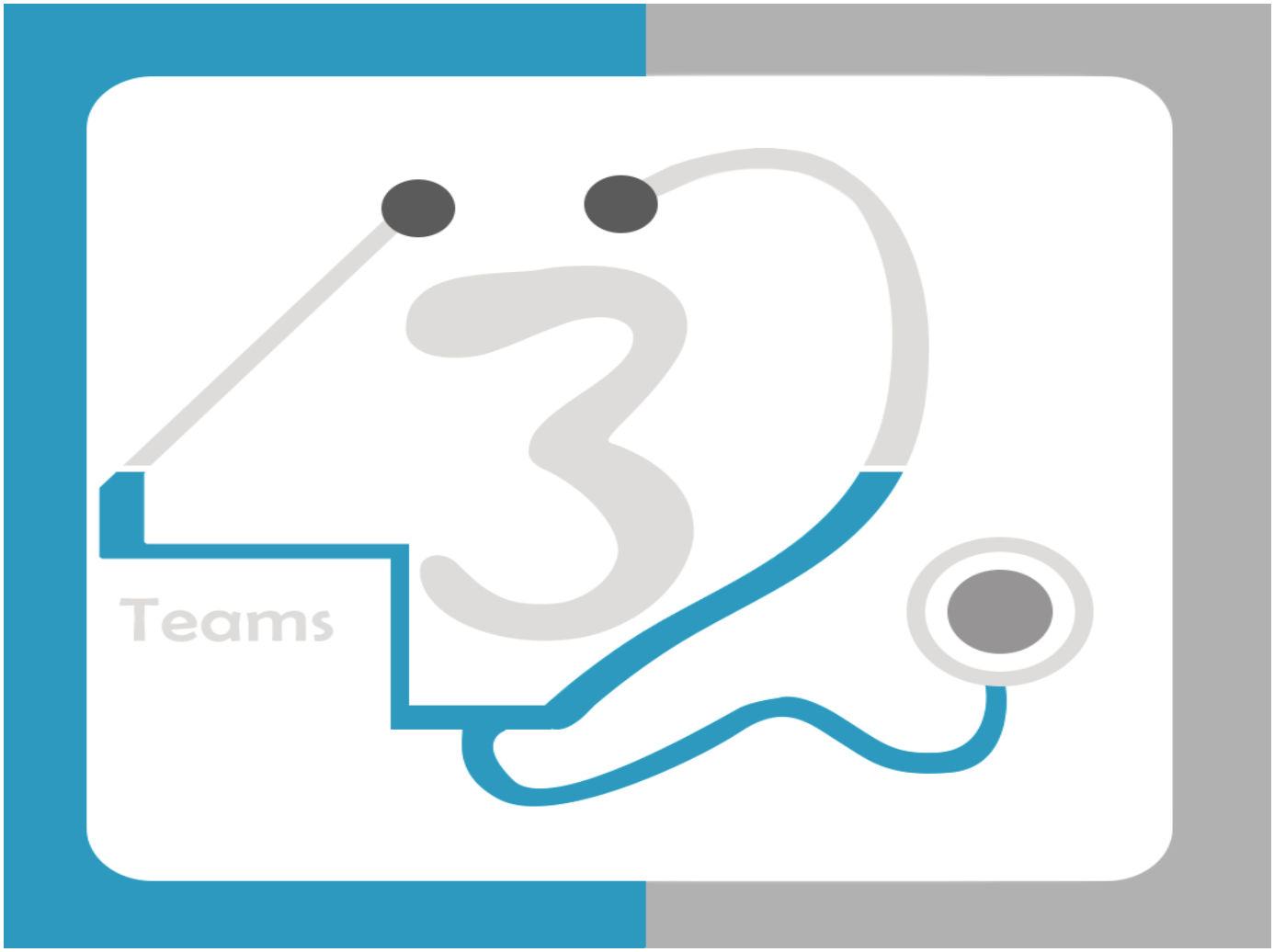

Pharmacology Team

Done by : Sara Alabdualqader

Notes From : Ibrahim AL-Salim

Red	**Important**
Purple	Extra Notes
blue	Males' notes
Black	From the slides

<table>
<tr><th colspan="3">Antiarrhythmic Drugs
Vaughan-Williams classification</th></tr>
<tr><th>Class I Na channel blocker</th><th colspan="2">Discussed in lecture 3</th></tr>
<tr><td rowspan="2">Class II β- ADRENOCEPTOR blockers

Propranolol: non selective Bete Blocker and it is used as local anesthetic (Just like class 1 antiarryhmitic drug).</td><td colspan="2">Pharmacological action

blocking β_1- receptors in the heart → reduce the sympathetic effect on the heart causing :

• decrease automaticity of S.A. node and ectopic pacemakers.
• slow conduction of the A.V node.</td></tr>
<tr><td colspan="2">Clinical uses:

1- atrial arrhythmias associated with emotion, exercise and thyrotoxicosis (arrhythmias caused by sympathetic-over activity)

2- digitalis-induced arrhythmias

3- Prophylactic or treatment of choice for post myocardial infarction arrhythmias

(Decrease the incidence of sudden death)

e.g. Propranolol (-lol family)

Digitalis is toxic flower that cause bradycardia and arrhythmia</td></tr>
<tr><td rowspan="2">Class III potasium channel blocker</td><td rowspan="2">• Prolong the action potential duration & effective refractory period . (Prolong phase 3)

Common side effect: torsades de pointes (ventricular arrhythmias)</td><td>amiodarone</td></tr>
<tr><td>Ibutilide</td></tr>
</table>

<table>
<tr><td rowspan="2">Class IV calcium channel blockers

Affect mainly the myocardium to Depress the myocardium porlong (delay) conduction of AV node.

Act on L-type Ca channel.</td><td>• Site of action is A.V.N & S.A.N .
• Inhibits calcium entry through L-type calcium in the myocardium and depress AV nodal transmission.

• Phase 0 & phase 2 (palteu)</td></tr>
<tr><td>Clinical uses:
• Atrial flutter - fibrillation
• Av nodal reentry

NOT effective in ventricular arrhythmias</td></tr>
</table>

Drugs	
Amiodarone; Class III **Amiodarone doesn't cause torsades de pointes.. Due to it has wide range of activity, it has got class I antiarrhythmic Class II, Class IV as well as calss III. And can block α- and β-adrenoceptor . (it is non selective)**	• **vasodilating effects** **(due to α- and β-adrenoceptor blocking effects and calcium channel blocking effects)** • **It has the action of class I, II & IV**
	Clinical uses: **Treatment of recurrent ventricular tachycardia – Fibrillation** **It is restricted for life-threatening arrhythmias.**
	Adverse effects: • **Bradycardia & heart block, heart failure, pulmonary fibrosis ,interstitial pneumonitis** • **hyper- or hypothyroidism** • **Skin deposits causes photodermatitis , gray-blue skin rash.** • **Peripheral neuropathy** • **constipation** • **corneal opacities** • **Hypotension**
	Drug Interactions: **Reduce renal clearance of several drugs e.g.** **quinidine, warfarin, procaiamide, flecainide**
Ibutilide ; Class III	• **rapid (short acting)** • **I.V. infusion. (in emergency cases)**
	Clinical uses: acute conversion of atrial flutter or atrial fibrillation to normal sinus rhythm.
	Side effect: torsades de pointes. **ECG (prolonged QT interval)**

<table>
<tr><td rowspan="3">Adenosine; miscellaneous group</td><td>naturally occurring nucleoside

Mechanism: In cardiac tissues

Binds to type 1 (A1) receptors which are coupled to Gi-proteins , activation of this pathway causing :

1- Opening of potassium channels (hyperpolarization)
2- Decrease calcium influx
3- increases the vagal tone on the AV node (slow conduction)</td></tr>
<tr><td>Clinical uses:

drug of choice for acute management of: paroxysmal supraventricular tachycardia

given 6 mg I.V. bolus followed by 12 mg if necessary</td></tr>
<tr><td>Adverse effect:

• Flushing & headache vascular
• Hypotension
• Dyspnea and chest pain (non-vascular)</td></tr>
<tr><td>Atropine; for Brady arrhythmias

Used as Temporary treatment of bradycardia.</td><td>• can be used in sinus bradycardia after myocardial infarction and in heart block
• in emergency heart block isoprenaline may be combined with atropine</td></tr>
</table>

Non-pharmacologic Therapy Of Arrhythmias

- Implantable Cardiac Defibrillator (ICD) can automatically detect and treat fatal arrhythmias such as ventricular fibrillation

Cases

A 62-year-old man is being managed in the intensive care unit following a large anterior wall MI. He has been appropriately managed with oxygen, aspirin, nitrates, and β-adrenergic receptor blockers but has developed recurrent episodes of ventricular tachycardia. During these episodes he remains conscious but feels dizzy, and he becomes diaphoretic and hypotensive. He is given an IV bolus of lidocaine and started on an IV lidocaine infusion.

Q1: what class of anti-arrythmitic drugs does Lidocaine belong?

Q2: what is Lidocaine mechanism of action ?

A 69-year-old retired teacher presents with a 1-month history of palpitations, intermittent shortness of breath, and fatigue. She has a history of hypertension. An ECG shows atrial fibrillation with a ventricular response of 122 bpm and signs of left ventricular hypertrophy. She is anti-coagulated with warfarin and started on sustained-release propranolol l60 mg/d. After 7 days, her rhythm reverts to normal sinus spontaneously. However, over the ensuing month, she continues to have intermittent palpitations and fatigue. Continuous ECG recording over a 48-hour period documents paroxysms of atrial fibrillation with heart rates of 88–114 bpm. An echocardiogram shows a left ventricular ejection fraction of 38% with no localized wall motion abnormality.

Q1: At this stage, would you initiate treatment with an antiarrhythmic drug to maintain normal sinus rhythm, and if so, what drug would you choose?