ANTIARRHYTHMIC DRUGS

Class 1: Sodium Channel-Blocking Drugs

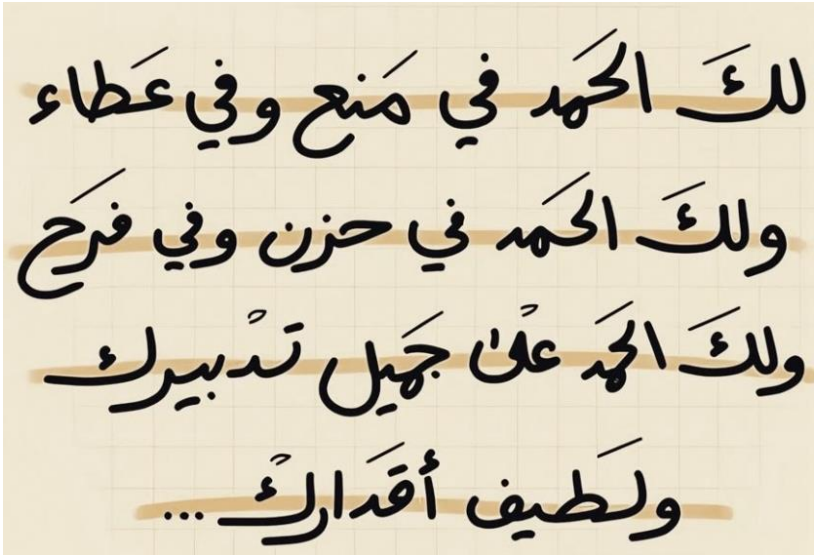
These drugs primarily slow the upstroke (Phase 0) of the action potential by blocking sodium channels, thereby slowing conduction.

Drugs	Mechanism of Action	Therapeutic Uses	Adverse Reactions
Procainamide	-Slows action potential upstroke (slowing conduction) and prolongs QRS duration. -Direct depressant actions on SA and AV nodes. -Ganglion-blocking properties, causing hypotension.	-Effective against most atrial and ventricular arrhythmias. -Second/third choice for sustained ventricular arrhythmias associated with acute myocardial infarction.	-Lethal arrhythmias (torsades de pointes, syncope) and QT-interval prolongation. -Excessive slowing of conduction . -Lupus erythematosus-like syndrome (arthralgia, arthritis) in one-third of patients.
Lidocaine, Mexiletine	-Rapid sodium channel blocker during Phase 0 and Phase 2, decreasing ventricular excitability. -Use-dependent binding (more blockade at higher heart rates).	**-Lidocaine** (IV infusion): Ventricular tachycardia and fibrillation following acute myocardial infarction, cardiac surgery, and catheterization. -**Mexiletine** is an orally active analogue.	-Hypotension, cardiovascular collapse,cardiac arrest, bradycardia, convulsions, light-headedness, unconsciousness, visual disturbances, respiratory depression, tinnitus ,vomiting. -**Contraindicated in** : -Wolff-Parkinson-White syndrome (can increase conduction through accessory pathway) and severe heart block.
Flecainide, Propafenone	Potent blocker of sodium (and some potassium) channels with slow unblocking kinetics. **Flecainide** does not prolong the action potential or QT interval. **Propafenone** has weak beta-blocking activity.	**Flecainide**: Supraventricular arrhythmias in patients with normal hearts (not first-line); suppresses premature ventricular contractions. **Propafenone**: Primarily for supraventricular arrhythmias.	Severe exacerbation of arrhythmia and increased mortality rate, especially in patients with pre-existing ventricular tachyarrhythmias or prior myocardial infarction. **Propafenone**: metallic taste and constipation.

Class 2: Beta-Adrenergic Blockers

These drugs reduce heart rate and slow AV nodal conduction by blocking beta-adrenergic receptors.

Drugs	Mechanism of Action	Therapeutic Uses	Adverse Reactions
Propranolol, Esmolol, Sotalol (also Class 3), Nadolol...	-Negative chronotropic effect (bradycardia). -Slows AV nodal conduction and prolongs its refractory period (prolonging PR interval). -Suppresses catecholamine-precipitated ectopic beats.	Effective in both supraventricular and ventricular arrhythmias. Slows ventricular response rate in atrial flutter/fibrillation by increasing AV nodal refractory period. Improves survival following myocardial infarction. **Esmolol** is useful for acute perioperative arrhythmias (short duration of action). The non selective beta blockers **Propranolol and Nadolol** are more effective subgroup of beta blockers for prevention and treatment of cardiac arrhythmias	Sinus bradycardia, depression of myocardial contractility (leading to heart failure). May mask symptoms of hypoglycemia in diabetics. It's not advisable to use the non selective agents in insulin dependent diabetics . Fatigue, depression, sexual dysfunction. Acute withdrawal can cause sudden tachycardia and exacerbation of ischemic symptoms.



Class 3: Drugs that Prolong the Effective Refractory Period by Prolonging the Action Potential

These agents prolong the action potential duration by blocking potassium channels in Phase 3.

Drugs	Mechanism of Action	Therapeutic Uses	Adverse Reactions
Amiodarone Dronedarone	-Markedly prolongs action potential (and QT interval) by blocking multiple potassium channels (I_{kr}, K_{ATP}, I_{k1}, etc.). -Also blocks inactivated sodium channels, calcium channels, and alpha,beta-adrenergic receptors.	-Amiodarone: ventricular and supraventricular arrhythmias (including atrial fibrillation). -Dronedarone for atrial flutter and fibrillation.	1. Bradycardia and heart block 2. Massive tissue accumulation 3. Pulmonary toxicity (fatal pulmonary fibrosis (~1%)). 4. Hepatotoxicity 5. Skin changes photosensitivity 6. Corneal deposits asymptomatic micro-deposits - Blocks CYP enzymes 7. Visual halos 8. Optic neuritis(rare). 9. Inhibits T4 → T3 conversion 10. Large iodine load - Inhibits several cytochrome P450 enzymes
Sotalol	-Both,beta-adrenergic-blocking activity (L-isomer) and action potential-prolonging effects (D- and L-isomers). -Potent inhibitor of I_{kr} (rapid delayed rectifier potassium current). -Not selective.	-Life-threatening ventricular arrhythmias. -Maintenance of sinus rhythm in atrial fibrillation. - Treatment of supraventricular and ventricular arrhythmias in the pediatric	-Dose-related torsades de pointes. -May depress left ventricular function in patients with heart failure.
Dofetilide Ibutilide	-Dose-dependent blockade of the rapid component of the delayed rectifier potassium current (I_{kr}). -Does not block other channels or adrenergic receptors. -100% Bioavailable. -eliminated by kidney	-Maintenance and restoration of normal sinus rhythm in patients with atrial fibrillation. -Treatment should be initiated in hospital after baseline measurement of the rate-corrected QT interval (QTc) and serum K + , and Mg2+ . -During loading, the QTc is measured before the second and subsequent doses; an increase in the QTc to ≥500 ms is an indication to reduce the dose or discontinue the drug.	**Contraindicated in :** 1. A baseline QTc of greater than 450 ms (500 ms in the presence of an intraventricular conduction delay) 2. Bradycardia of less than 50 bpm 3. Hypokalemia

Class 4: Calcium Channel-Blocking Drugs

These drugs block L-type calcium channels, primarily affecting tissues where activation depends on the calcium current, such as the SA and AV nodes.

Drugs	Mechanism of Action	Therapeutic Uses	Adverse Reactions
Verapamil, Diltiazem	-Blocks activated and inactivated L-type calcium channels. -Prolongs AV nodal conduction time and refractory period. - Slows SA node rate. -Suppresses both early and delayed after depolarizations.	Supraventricular tachycardia. To reduce ventricular rate (rate control) in atrial fibrillation or flutter.	Hypotension and ventricular fibrillation if misdiagnosed and given for ventricular tachycardia. May precipitate heart failure. Can induce AV block in large doses or in patients with AV nodal disease(This block can be treated with atropine and β -receptor stimulants) Constipation, peripheral edema. Avoid in digoxin-induced arrhythmias (increases digoxin concentration).

5. Unclassified Antiarrhythmic Agents

These agents have various mechanisms that do not fit into the primary Vaughan Williams classification scheme.

Drugs	Mechanism of Action	Therapeutic Uses	Key Information/Adverse Effects
Adenosine	Activates K^{+} efflux and reduces Ca^{++} influx, causing marked hyperpolarization and suppression of calcium-dependent action potentials. Directly inhibits AV nodal conduction.	Drug of choice for prompt conversion of paroxysmal supraventricular tachycardia to sinus rhythm. · It is less effective in the presence of adenosine receptor blockers (theophylline or caffeine), and its effects are potentiated by adenosine uptake inhibitors such as dipyridamole.	Very short half-life (<10 seconds). Adverse reactions include flushing, shortness of breath, chest burning, and short-lived high-grade AV block.

Drugs	Mechanism of Action	Therapeutic Uses	Key Information/Adverse Effects
Ivabradine	Selective blocker of the "funny" current (I_{f}) in the SA node. Slows pacemaker activity by decreasing diastolic depolarization.	Useful for heart rate control in patients with coronary artery disease and chronic stable angina (Has antianginal/anti-ischemic effects). In patients with left ventricular dysfunction and heart rates greater than 70 bpm, it reduces mean heart rate.	Reduces heart rate without affecting myocardial contractility or conduction. Block of I_{f} in the retina may cause visual disturbances.
Magnesium	-Influences Na^{+}/K^{+} ATPase, sodium, certain potassium, and calcium channels.	-Indicated in patients with torsades de pointes (even if serum magnesium is normal). -Originally used for digitalis-induced arrhythmias with hypomagnesemia.	Mg^{2+} levels are important to maintain.
Potassium	-Increases potassium permeability (membrane potential stabilizing action) and has a resting potential depolarizing action. -Hypokalemia increases risk of after-depolarizations and ectopic activity.	-Therapy is directed toward normalizing potassium gradients.	-Both insufficient (hypokalemia) and excess (hyperkalemia) potassium are potentially arrhythmogenic. - Hyperkalemia can depress ectopic pacemakers and slow conduction.

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