Antiarrhythmic Medications

Class I (sodium channel blocking) antiarrhythmics			
	Specific agents	**Inhibition of phase 0 depolarization**	**Effect on length of action potential**
Class IA	Quinidine, procainamide, disopyramide	Intermediate	Prolonged
Class IB	Lidocaine, mexiletine	Weak	Shortened
Class IC	Flecainide, propafenone	Strong	No change

Class	**Drug Examples**	**Mechanism of Action**	**Effect on Action Potential (AP) & ECG**	**Clinical Use**	**Notable Risks**
Class I: Na+ Channel Blockers					
IA	Quinidine, Procainamide, Disopyramide	Intermediate Na+ blockade, Moderate K+ blockade	*Prolongs* AP duration, Prolongs QT interval	Atrial & ventricular arrhythmias	Risk of **QT prolongation**, **torsades de pointes**
IB	Lidocaine, Mexiletine	Weak Na+ blockade, Rapid dissociation	*Shortens* AP duration, Minimal QT effect	Ischemia-induced ventricular arrhythmias	Minimal risk of torsades
IC	Flecainide, Propafenone	Strong Na+ blockade, Slow dissociation	*No effect* on AP duration, **Prolongs QRS**	Supraventricular arrhythmias	High proarrhythmic risk, **Use dependence** (increased effect at high HR)
Class II: Beta Blockers	Metoprolol, Atenolol, Esmolol	Block β-adrenergic receptors	↓ HR & contractility, No QT effect	Rate control in atrial fibrillation	Bradycardia, Hypotension
Class III: K+ Channel Blockers	Amiodarone, Sotalol, Dofetilide	Block K+ channels, Prolongs repolarization (Phase 3)	**Prolongs QT interval, AP duration**	Atrial & ventricular arrhythmias	**Torsades risk** (highest with Sotalol, lower with Amiodarone)
Class IV: Ca2+ Channel Blockers	Verapamil, Diltiazem	Block L-type Ca2+ channels	**Slows AV conduction, ↓ HR**, No QT effect	Rate control in atrial fibrillation	Hypotension, Bradycardia
Other Agents					

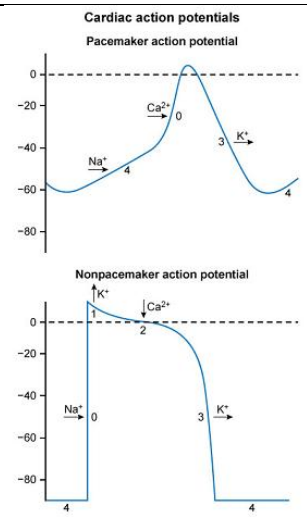
Class	Drug Examples	Mechanism of Action	Effect on Action Potential (AP) & ECG	Clinical Use	Notable Risks
Adenosine	-	Activates K+ channels via A1 receptors	**Slows AV conduction**, transient asystole	**Acute termination of PSVT**	Flushing, Hypotension
Digoxin	-	Inhibits Na+/K+ ATPase, Enhances vagal tone	**Slows AV conduction**, Can shorten QT	**Atrial fibrillation with heart failure**	**Toxicity: Arrhythmias, GI distress**

Key ECG Effects

- **QRS Prolongation:** Class IA, IC
- **QT Prolongation:** Class III, IA
- **No QT Effect:** Class IB, II, IV
- **Torsades Risk:** High (Sotalol), Moderate (IA), Low (Amiodarone)
- **Use Dependence:** Class IC (effects increase with faster HR)

Cardiac Action Potential Phases & Drug Effects

Phase	Activity	Affected by Drug Class
0 (Rapid Depolarization)	Na+ influx	Blocked by **Class I**
1 (Early Repolarization)	K+ efflux	Minimal drug effects
2 (Plateau Phase)	Ca2+ influx & K+ efflux balance	Blocked by **Class IV**
3 (Late Repolarization)	K+ efflux	Blocked by **Class III**
4 (Resting Potential)	Na+/K+ ATPase pump	Affected by **Beta Blockers & Class IV**



Clinical Applications

- **Atrial Fibrillation:**
    - **Rate control:** Beta Blockers, Calcium Channel Blockers
    - **Rhythm control:** Class III (Amiodarone, Sotalol, Dofetilide)
- **Ventricular Arrhythmias:**
    - **Ischemic-related:** Class IB (Lidocaine, Mexiletine)
    - **Structural heart disease:** Amiodarone preferred
- **PSVT Termination: Adenosine**
- **Heart Failure + Atrial Fibrillation: Digoxin**

Conclusion

Antiarrhythmic drugs function by modifying ion channel activity during cardiac action potentials. Their clinical use depends on arrhythmia type, patient factors, and risk of proarrhythmia. **Close ECG monitoring is essential**, especially for drugs that prolong the **QT interval**.