

● CLASS I — Sodium Channel Blockers

These drugs block fast Na^+ channels in phase 0. → *↓ Chronotropic + ↓ Dromotropic Effects*
Subgroups differ by **binding kinetics**, **effect on AP duration**, and **clinical use**.

Class IA — Procainamide

Mechanism → *other than the main one? (3)*

- Blocks fast Na^+ channels → slows phase 0 upstroke → slows conduction, prolongs QRS.
- Also blocks K^+ channels → prolongs action potential, prolongs QT.
- Direct depressant effect on SA + AV nodes (↓ HR, ↓ AV conduction).
- Anticholinergic / antivagal activity partially counters bradycardia.
- Ganglion-blocking effect → ↓ peripheral vascular resistance → postural hypotension.

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Therapeutic Uses

- Not first-line for any arrhythmia.
- Effective for atrial and ventricular arrhythmias.
- Second/third choice (after lidocaine or amiodarone) for sustained ventricular arrhythmias post-MI.

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Adverse Effects

- QT prolongation → torsades de pointes (very important).
- Excessive conduction slowing → new arrhythmias.
- Drug-induced lupus-like syndrome (arthralgia, arthritis, pleuritis, pericarditis, rarely renal).

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KEY TAKEAWAY vs Other Class I drugs

- Only Class IA drug mentioned.
- Only drug associated with lupus-like syndrome.
- Prolongs AP + QT, unlike Class IB (shorten/neutral) and Class IC (neutral AP).

Class IB — Lidocaine

Mechanism

→ Used in rapid responses

- Rapid block of Na^+ channels during **phase 0** and **phase 2**. / No K^+ Blockage → No QT prolongation → No TdP
- Use-dependent block → more effective at **higher HR** or active ischemic tissue.
- ↓ **Ventricular excitability**.

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Uses

- IV only (destroyed in GI tract).
- **Ventricular tachycardia** and **ventricular fibrillation** especially:
 - Post-MI
 - Post-cardiac surgery
 - Post-catheterization

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Contraindications

- **WPW syndrome** → increases conduction through accessory pathway → VF. (2)? as it inhibits the AV Node Conduction
- **Severe heart block** → may → complete block/asystole.

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Adverse Effects

- **Cardiac**: hypotension, HF, bradycardia, cardiac arrest.
- **CNS**: light-headedness, seizures, unconsciousness, tinnitus, visual disturbances.

- **Respiratory depression.** طنين الأذن

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Related Drug

- **Mexiletine** = oral analogue of lidocaine.

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KEY TAKEAWAY vs Other Class I

- Best for **acute ischemic ventricular arrhythmias**.
- **Fastest kinetics**, least proarrhythmic.
- Does **NOT** prolong QT.
- Opposite of procainamide: shortens conduction without QT prolongation.

Class IC — Flecainide

Mechanism

- Potent blockade of Na⁺ and K⁺ channels with slow unbinding kinetics ("slow on-off").
→ Although No QT Prolongation
- Despite K⁺ block → **NO QT or AP prolongation.** *→ No TdP*
- Suppresses PVCs. *(Premature Ventricular Contractions)*

Uses

- Supraventricular arrhythmias in patients with **normal hearts**.
→ Slow ON/OFF → can't be given to patients with sudden transient changes
- Not first-line.

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Adverse Effects

- Major **proarrhythmic risk** in:
 - patients with prior MI
 - structural heart disease
 - ventricular tachyarrhythmias
- **Increases mortality** in these patients.

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KEY TAKEAWAY

- Strongest Na⁺ blocker but **dangerous in structural heart disease**.
- Does **not affect AP duration** (unlike IA or III drugs).

Class IC — Propafenone

Mechanism

- Same Na⁺ channel-blocking kinetics as flecainide (slow unbinding).
- Weak β -blocking activity (unique).
- Does **not** prolong AP.

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Uses

- Primarily for supraventricular arrhythmias.

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Adverse Effects

- **Metallic taste** (like metronidazole).
- **Constipation.**
- Can worsen arrhythmias. *just like Flecainide*

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KEY TAKEAWAY

- Class IC with **β -blocking properties**, unlike flecainide.

● CLASS II — Beta Blockers

Primarily affect atria → SA node → AV node.

Reduce HR, prolong AV nodal refractory period → increase PR interval.

↓ Chronotropic effect + ↓ Dromotropic
↓ HR
↓ prolonged PR Interval

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Mechanism of Action

- Negative chronotropy (↓ HR).
- Slow AV nodal conduction → useful in atrial flutter/fibrillation to slow ventricular rate.
- Reduce catecholamine-induced ectopy. (Tachycardia)

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↳ Ex: Smolters

Therapeutic Uses

- Supraventricular + ventricular arrhythmias.
- Post-MI survival improvement → ↓ O₂ demand, ↓ ischemia, ↓ risk of ventricular arrhythmias.
- Atrial flutter/fibrillation rate control.
- Smokers with catecholamine-triggered extrasystoles.

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Special Drugs

Esmolol

- Very short-acting IV β-blocker.
- Used for acute perioperative arrhythmias.

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Propranolol / Nadolol

- Nonselective β-blockers.
- More effective subgroup for arrhythmia suppression.

Adverse Effects

- Bradycardia.
 - ↓ contractility → **HF worsening** if started rapidly in unstable patients. *(Given for stable patients only)*
 - Mask hypoglycemia symptoms in diabetics (esp. non-selective β -blockers).
 - Fatigue, depression, sexual dysfunction.
 - Dyslipidemia (\uparrow TG, \downarrow HDL) — **not seen with vasodilating β -blockers** (carvedilol, labetalol). *(slight weight gain)*
 - **Withdrawal syndrome** → tachycardia + ischemia.
(Rebound Syndrome)
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- A potential adverse effect of β -blockers is the **worsening of heart failure** if they are started during acute decompensation or if the dose is increased too rapidly. They have **cardioprotective effects when initiated at low doses**. Therefore, β -blocker therapy should be started **only in clinically stable patients**, with slow and careful dose titration.