○ CLASS I — Sodium Channel Blockers

These drugs block fast Na⁺ channels in phase 0. — Chronot ropic + 1 Dromotropic Effects
Subgroups differ by binding kinetics, effect on AP duration, and clinical use.

Class IA — Procainamide

Mechanism rollier 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.
- <u>Drug-induced lupus-like syndrome</u> (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 roupid responses · Rapid block of Na+ channels during phase 0 and phase 2. / No K+ Blokege > No QT probable

- Use-dependent block \rightarrow 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 (2)7. • WPW syndrome → increases conduction through accessory pathway → VF. AV Node Conduction

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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.

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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

Although No QT Polongation

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

Uses

with normal hearts

• Supraventricular arrhythmias in patients with normal hearts.

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 Flecaride

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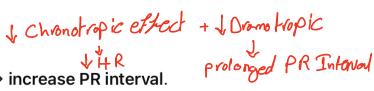
KEY TAKEAWAY

Class IC with β-blocking properties, unlike flecainide.

Primarily affect atria → SA node → AV node.

Reduce HR, prolong AV nodal refractory period → increase 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.

>Ex: Smallers

· Reduce catecholamine-induced ectopy. (Tacchy cardia)

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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 <u>acute perioperative arrhythmias</u>.

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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 Slable Policules 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)
- 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.