Class I Antiarrhythmics (Na+ Channel Blockers) Overview

Class I Antiarrhythmics treat tachyarrhythmias by blocking fast Na⁺ channels and there are several subcategories of drug based on their specific effect on cardiac action potentials. These drugs are used to restore normal pacemaker and conduction activity, and are selective for depolarized tissue, which is known as state dependant. Class I antiarrhythmic drug's blockade of Na⁺ channels reduces rate of phase 0 depolarization and prolongs effective refractory period. This Na⁺ blockade also increases the threshold for firing within abnormal pacemaker cells. These drugs are contraindicated in hyperkalemic states, as excess potassium increases resting membrane potential and can produce a sodium-channel blockade so pronounced that asystole may result in patients taking Class I antiarrhythmics.

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Indications

Arrhythmias
Broken Arrhythmia-drum
These drugs are indicated for arrhythmias, which generally refer to abnormal heart rate or regularity. They can be caused by either irregular impulse conduction or impulse generation.

Local Anesthesia
A-nest of Local Anethesia
Class I antiarrhythmics also have local anesthetic properties.

Mechanism of Action

Block or Slow Conduction
Slowed Snail-conductor
Class I drugs act by blocking fast sodium channels. This slows or halts conduction, especially within depolarized cells.

Block Na+ Channels
Block-guy Blocking Salt-shaker Channel
Blockage of Na+ channels reduces rate of phase 0 depolarization and prolongs effective refractory period.

Raise Threshold
Raised Blocks
Because of the Na+ channel blockage, these drugs increase the threshold for firing within abnormal pacemaker cells.

State Dependent
State Dependent Mine
The drugs in this class are state dependant, meaning they selectively depress tissue that is frequently depolarized, versus normally polarized tissue. This can include ischemic tissue.

Contraindication

Hyperkalemia
Caution-tape Hiker-banana
These drugs are contraindicated in hyperkalemic states as they cause increased toxicity for all class I agents. This is because excess potassium increases resting membrane potential and can produce a sodium-channel blockade so pronounced that asystole may result.