Anti-arrhythmic Drugs

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Introduction

Drugs used for preventing and treating irregular heart rate and heartbeat are called antiarrhythmic drugs. Arrhythmia results from disruptions in the formation of electric impulses and their conduction to the heart.

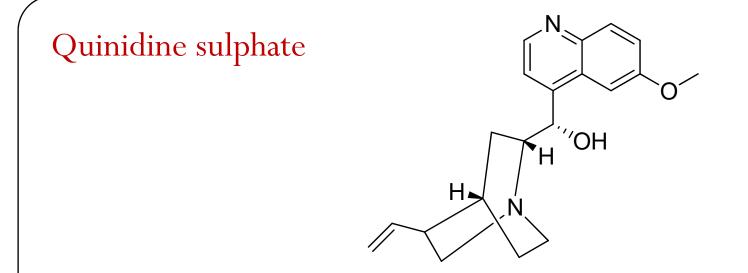
Drugs used for treating arrhythmia can have an effect on the electrical conduction system of the heart, its excitability, automatism, the size of the effective refractory period, and adrenergic and cholinergic heart innervation.

Group I Drugs

Drugs belonging to this group are, with a few rare exceptions, local anesthetics which form complexes with lipoproteins of cell membranes of the myocardia, thus blocking Na channel conductivity and the flow of Na into the cell, and facilitate the release of K from myocardial cells.

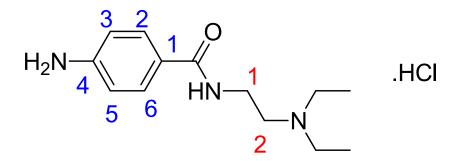
Subgroup IA

Drugs of this subgroup slow down the speed of transmitting excitation, reduce excitability of Purkinje fibers, suppress automatism of ectopic regions and increase the effective refractory period.

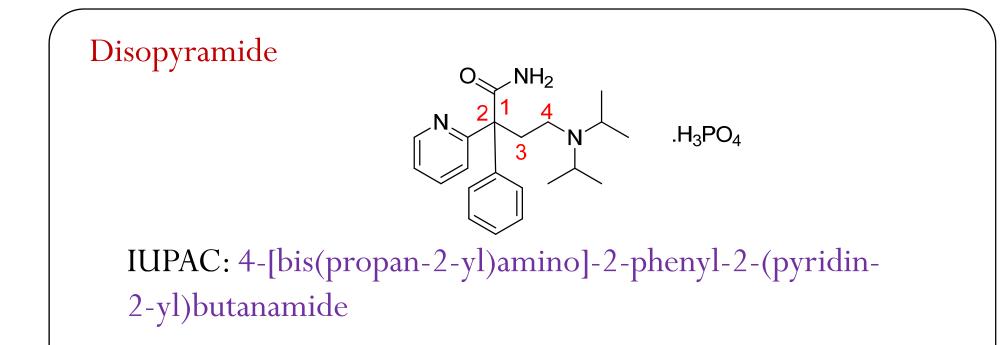


IUPAC: (R)-(6-Methoxyquinolin-4-yl)((3S,4R,7S)-3-vinylquinuclidin-7-yl) methanol

Procainamide hydrochloride



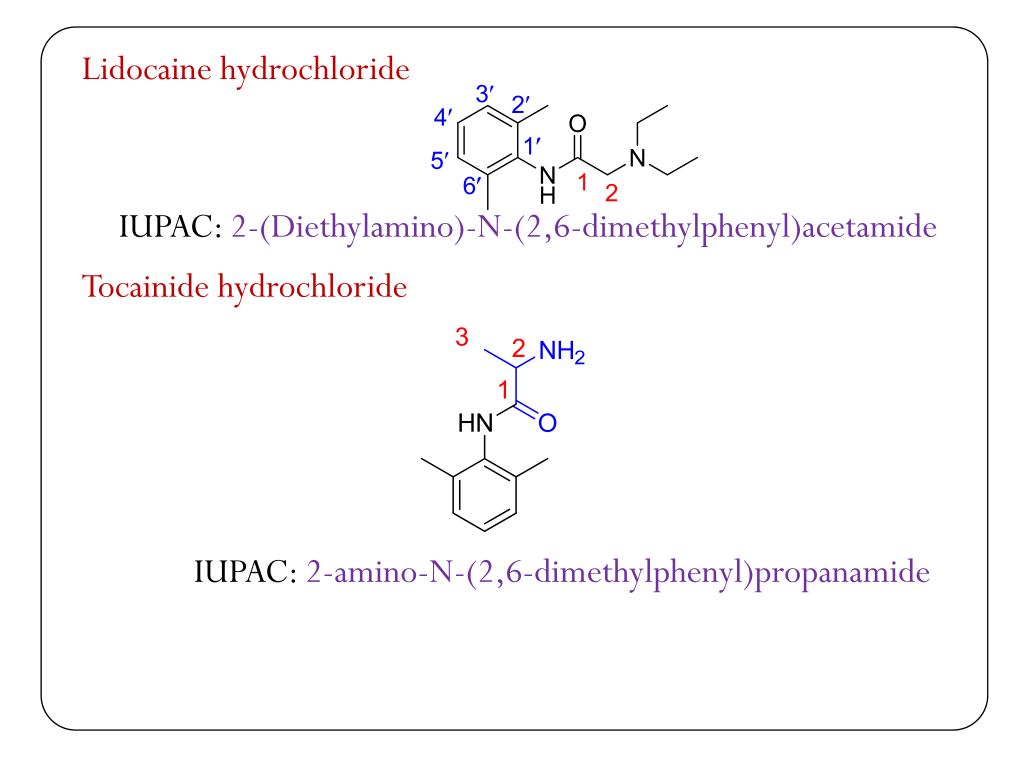
IUPAC: 4-Amino-N-(2-diethylaminoethyl)benzamide

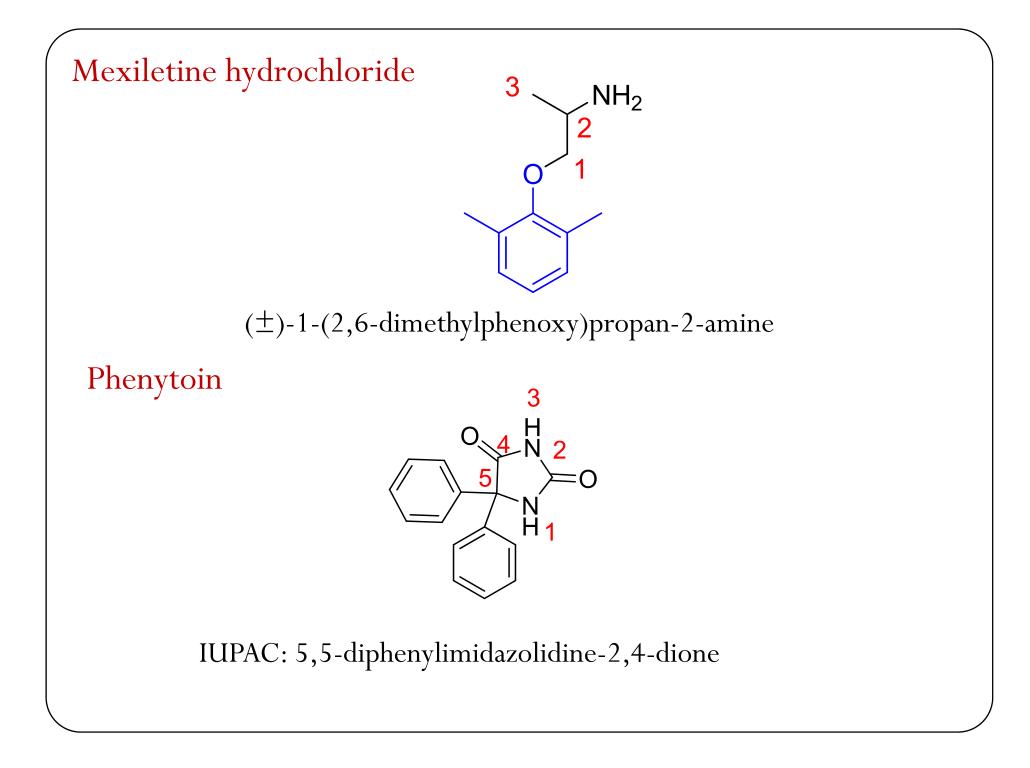


Subgroup IB

They differ from drugs of subgroup IA in that if they block open Na channels, then drugs of subgroup IB mainly block inactive Na channels. This means that they have little effect on healthy regions of the myocardium because they are quickly eliminated from normal, open Na channels.

Drugs of subgroup IB increase the electrical threshold of ventricular excitation during diastole, suppress automatism and diastolic depolarization, reduce the duration of the refractive Period.

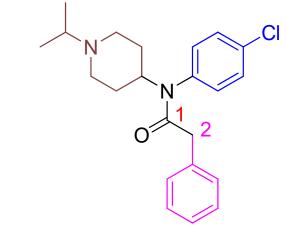




Subgroup IC

Drugs of this group are also referred to as sodium channel blockers. They substantially suppress depolarization of myocardial cells, while insignificantly reducing the time of their repolarization, and they also suppress automatism of sinoatrial nodes. These drugs differ from those examined above in that they reduce conductivity and increase the refractory period of the ventricles.

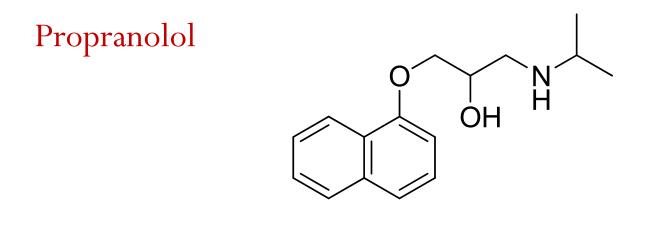
Lorcainide hydrochloride



N-(4-chlorophenyl)-2-phenyl-N-[1-(propan-2-yl)piperidin-4-yl]acetamide

Group II Drugs

This group consists of β -adrenergic receptor blockers, the antiarrhythmic activity of which is associated with inhibition of adrenergic innervation action of the circulatory adrenaline on the heart.

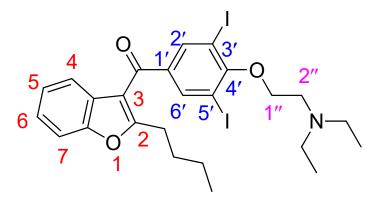


IUPAC: 1-(naphthalen-1-yloxy)-3-[(propan-2-yl)amino]propan-2-ol

Group III Drugs

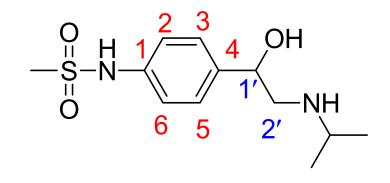
Drugs of this group exhibit antiarrhythmic action, slow repolarization, and increase conductivity of potential action and effective refractory period in all parts of the heart.

Amiodarone



2-Butyl-3-(3,5-diiodo-4-(2-diethylaminoethoxy) benzoyl)benzofuran

Sotalol

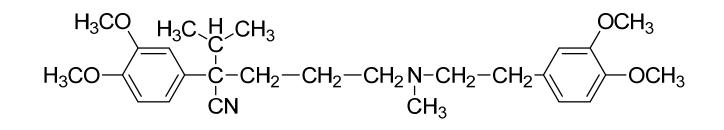


IUPAC: N-(4-{1'-hydroxy-2'-[(propan-2-yl)amino]ethyl}phenyl)methanesulfonamide

Group IV Drugs

Drugs of this group are calcium channel blockers that inhibit slow transmembrane calcium ion flow in the cell of the conductive system of the heart during depolarization, which causes a slowing of atrioventricular conductivity and increased effective refractive period of atrioventricular ganglia, which eventually leads to the relaxation of smooth muscle of heart musculature and restores normal sinus rhythm during supraventricular tachycardias.

Verapamil



IUPAC: 5-[(3,4-dimethoxyphenethyl)methylamino]-2-(3,4-dimethoxyphenyl) isopropylvaleronitrile