Antiarrhythmic drugs

Antiarrhythmics (also antidysrhythmics) are drugs used to treat heart rhythm disorders, and in some cases preventively. They affect cardiac contractility and hemodynamics. [1]

Arrhythmia pharmacotherapy depends on the type of arrhythmia, its duration, severity, and the condition of the heart muscle. Arrhythmias are divided into tachyarrhythmias and bradyarrhythmias.

The mechanisms of tachyarrhythmias may be increased irritability, increased automaticity, or reentry. Treatment - reduction of excitability and automaticity, treatment of ischemia is derived from the diagnosis of the mechanism of arrhythmia. [2]

See the Arrhythmia page for more information.

Therapeutic usage

The main goal of antiarrhythmic treatment is **to restore normal heart rhythm and transmission**; possibly at least to prevent more severe to fatal arrhythmias. They reduce or increase the speed of conduction, change the excitability of the heart cells and suppress abnormal automation.

All antiarrhythmics alter membrane conductivity by the following mechanisms:

- **By blocking fast sodium channels**. These channels determine the rate of membrane depolarization during the action potential, which can help eliminate tachyarrhythmias caused by the reentry mechanism.
- **By influencing the course of action potentials** and especially the relative refractory period. Prolonging the relative refractory period can often eliminate tachycardias. These drugs affect potassium channels and delay the repolarization phase.
- **By blocking slow calcium channels**. These drugs reduce the sinus frequency by slowing the depolarization of pacemaker cells. They also reduce the excitation speed of the AV node.
- **By blocking sympathetic activity**, which can also cause arrhythmias, β1-adrenergic receptor blocking drugs are used to suppress this sympathetic effect on the heart. Because β-adrenoceptors are coupled to ion channels, β-blockers also indirectly alter ion flux across the membrane, especially calcium and potassium.
- In the case of AV block, **vagal inhibitors** (eg atropine, a muscarinic receptor antagonist) are sometimes used. AV block may occur during treatment with β-blockers.
- In some cases, the ventricular rate is inadequate because it is initiated by atrial flutter or atrial fibrillation. Because it is very important to prevent ventricular tachycardia, drugs are often used **to slow the conduction of excitation through the AV node**. Calcium channel blockers and β-blockers are often used for this purpose. For the same reason, the parasympathomimetic effect of digitalis can be used.[3][4]

Antiarrhythmics often have a **proarrhythmic effect**, so they should only be used for symptomatic arrhythmias that worsen the patient's quality of life or prognosis.[2]

Classes of drugs used to treat arrhythmias[4]

1. **Class I - Fast sodium channel blockers** - atrial fibrillation cardioversion, etc.
    1. Ia - blockade of Na + channels - quinidine,
    2. Ib - blockade of Na + channels - lidocaine, trimecaine, phenytoin
    3. Ic - blockade of Na + channels - propafenone, flecainide
2. **Class II - β-blockers** (see above) - control of ventricular response in supraventricular tachycardia,
3. **Class III - Potassium channel blockers** (eg amiodarone) - supraventricular and ventricular tachycardia,
4. **Class IV - Calcium channel blockers** (verapamil, diltiazem) - supraventricular tachyarrhythmias only.
5. **Next:**
    1. **adenosine**,
    2. **electrolyte supplementation** (magnesium and potassium salts),
    3. **cardiac glycosides** (digitalis),
    4. **atropine** (muscarinic receptor antagonist),
    5. **bradins** (SA node blockers).[2]

Class Ia of antiarrhythmics

Class Ia antiarrhythmic sodium channel blockade prolongs action potential duration and slightly prolongs repolarization.[1]

Quinidine

For pharmacological cardioversion of atrial fibrillation and flutter. It has many side effects.

Procainamide

Used to treat ventricular and supraventricular arrhythmias.

Disopyramide

For the treatment of tachyarrhythmias, especially after a heart attack.

Class Ib of antiarrhythmics

They block the sodium channel, but have little effect on the rate of increase in action potential. They shorten the repolarization time.[1]

Lidocaine, trimecaine

Used mainly in the treatment of ventricular tachycardia.

Class Ic of antiarrhythmics

They block the sodium channel, significantly slowing down the rate of action potential onset and conduction. The time of repolarization is little affected by them.[1]

Propafenone

Used to treat atrial fibrillation and ventricular tachycardia.

Class I antiarrhythmics are not commonly used today, except for propafenone and phlecainide (both of Class Ic).[2]

Class II of antiarrhythmics

These are β-adrenergic blockers. They reduce calcium channel phosphorylation. They negatively affect the frequency of spontaneous depolarization in the SA and AV nodes. They do not affect the repolarization time

Class III of antiarrhythmics

They block potassium channels, prolong the action potential and dampen the effect of sympathetic nervous system. They prolong the refractoriness of the atria, transmission system and ventricles.[2] They are used in atrial fibrillation and ventricular tachycardia.

Amiodarone

It has a slow onset of action and an extremely long elimination half-life (up to 100 days), so we must monitor its plasma level. It is the most effective antiarrhythmic in suppressing ventricular and supraventricular tachycardias. It is indicated after an acute myocardial infarctionTemplate: Replenish the source, at a high risk of sudden cardiac death and impaired left ventricular systolic function. Amiodarone has an undesirable negative inotropic effect, which requires careful use in heart failure. At the same time, however, it is the only antiarrhythmic that reduces the risk of atrial fibrillation (eg again in heart failure)[2]. It has numerous side effects, especially **thyroid disorders** (hypothyroidism, rarely hyperthyroidism), bradycardia, pulmonary fibrosis, hepatotoxicity and corneal deposits.

Sotalol

It prolongs the duration of the action potential and slows down the repolarization phase. Use is limited due to its lower antiarrhythmic effect.

Class IV of antiarrhythmics

Calcium channel blockers **verapamil** and **diltiazem** inhibit conduction in the AV node. They do not affect the repolarization time. They are mainly used for supraventricular tachycardias.[1]

Adenosine

Adenosine acts by stimulating potassium channels. It is given intravenously for its short duration of action. It reduces the automation of the sinus node and slows down the conduction of excitation in the atrial node. It slows the response of the ventricles in supraventricular arrhythmias (it is the medicine of first choice). It can also be given during pregnancy.

Bradins[2]

Bradins act selectively in the sinus node, where they slow down spontaneous diastolic depolarization. Their effect is only to slow down the heart rate.

The main indication for bradin is angina pectoris.

Summary of antiarrhythmic classes

Class IA: atrial fibrillation, flutter; supraventricular and ventricular tachyarrhythmias

chinidin	anticholinergic (medium)	cinchonismus (vision disorders, tinnitus, headache); nausea; potentiates digitalis toxicity
procainamide	anticholinergic (weak); relatively short half-life	lupus-like syndrome in 25-30% of patients
disopryamide	anticholinergic (strong)	negative inotropic effect

Class IB: ventricular tachyarrhythmias (VT)

lidocaine	IV only; VT and PVC	high efficacy in ischemic myocardium
mexiletine	an orally active lidocaine analog	high efficacy in ischemic myocardium
fenytoine	digitalis-induced arrhythmias	

Class IC: life-threatening supraventricular tachyarrhythmias (SVT) + ventricular tachyarrhythmias (VT)

flecainide	SVT	may induce life-threatening VT
propafenone	SVT and VT	β-blocking and Ca^{2+} channel blocking activity may exacerbate heart failure
moricizine	VT	activity of IB

Arrhythmia treatment - summary[4]

Condition	Drug	Comment
Sinus tachycardia	Class II, IV	Other related conditions may also require treatment.
Atrial fibrillation / flutter	Class IA, IC, II, III, IV, digitalis, adenosine	The goal is to control the ventricular rate; necessary anticoagulant treatment.
Paroxysmal supraventricular tachycardia	Class IA, IC, II, III, IV, adenosine	
AV reentry tachycardia	atropine	Acute reversal.
Ventricular tachycardia	Class I, II, III	
Premature ventricular contractions (PVCs)	Class II, IV; Salts of Mg^{2+}	Often benign and do not require treatment.
Digitalis toxicity	Class IB, Salts of Mg^{2+}; KCl	

Links

Related articles

- Electrical conduction system of the heart
- Heart rhythm disorder
- Hearing examinations and disorders
- Heart Attack
- Heart Development

External links

- https://www.wikiskripta.eu/w/Antiarytmika

Source

- https://www.wikiskripta.eu/w/Antiarytmika
- HYNIE, Sixtus. *Pharmacology in a nutshell.* - edition. Triton, 2001. 520 pp. ISBN 9788072541812 .
- LINCOVA, Dagmar. *Basic and applied pharmacology.* - edition. Galén, 2007. 672 pp. ISBN 9788072623730 .
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