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┌↑┌└ Clinic use : first choice for ventricular tachycardia and fibrillation after myocardial infarction Adverse reaction : Least cardiotoxic effects Neurologic: paresthesias,tremor, convulsions, slurred speech, hearing disturbances, lightheadedness x
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┌└P┌ Antiarrhythmic agents-Ib $☼┌└ IB-phenytoin sodium
Mechanism similar to lidocaine Pharmacologic effects * decrease automaticity in PF * compete with cardiac glycoside for combination of Na-K-ATPase Clinic use: to treat digoxin-induced dysrhythmias
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agents-Ic $☼┌└ IC-Propafenone Mechanism Na, (0,4), -R Pharmacologic effects automaticity conduction Clinic use: supraventricular arrhythmias ADR: QT ;metallic taste,constipation 14┌♬¶┌┌♬P┌┌┌
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antagonists Mechanism -R, Na, ERP + K Clinic use SVT, Af,hyperthyrosis ADR SB, AVB, HF, Hypotension, Asthma. _//% // % ! ’ $
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III Selectively prolong repolarization ?└# $// $☼┌└!♬┌└┌┼ Mechanism (Amiodarone) : block K+ ,Na+, Ca++ Pharmacological actions ″ Reduce automaticity in sinoatrial node and PF ″ slow conduction in atrioventricular node and PF ″ prolong ERP in atria and PF Clinic uses : ″ Supraventicular(atrial fibrillation), ventricular (tachycardia/fibrillation) tachyarrhythmias AR:photosensitive skin,thyroid abnormalities(hypo- and hyper-),pulmonary fibrosis, corneal deposits,neurological and gastrointestinal disturbances F┼Z€ Z♬┼Z` ┌┼♬Z Z ┘
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Pharmacological actions ″ reduce automaticity in sinoatrial node and atriaoventricular node by slowing P-4 velocity ″ slow conduction in atrioventricular node ″ prolong ERP Clinic uses: ″ to prevent or terminate recurrence of paroxysmal SVT; ″ to reduce the ventricular rate in patients with atrial fibrillation &┼Z Z ┼Z Z└ └| ┐└↑┘ € € € € &€
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↕┌└1◀┌└ Others : adenosine┼ Mechanism: Inhibit
atrioventricular nodal conduction Increase atrioventricular nodal refractory period
Pharmacokinetics: t <10 s Clinic use: paroxysmal supraventricular tachycardia; WPW

Antiarrhythmic agents

*Drugs to treate Bradycardia Atropine, Iso.

Antiarrhythmic agents-ADR

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Review & questions

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Antiarrhythmic agents-Ib $♬┌└◘┐┌└┌┼ IB-lidocaine Mechanism: Na, + K Pharmacologic effects automaticity(slow velocity of P-4 in PF) APD/ERP ERP terminate reentry + K hyperpolarization :┼Z6┌┼♬Z?┌┌┼♬Z"┌┼Z┘┌┼♬Z┐ ↑ ↑┐ ┐↑┌ ↑ 3 ↑┐6┘┐€ *◘┐€ € € € 3 € € € >└ ┌└;◀┌└e◀┌└ Clinic use : first choice for ventricular tachycardia and fibrillation after myocardial infarction Adverse reaction : Least cardiotoxic effects Neurologic: paresthesias,tremor, convulsions, slurred speech, hearing disturbances, lightheadedness p ^┌┌♬‼~ v└| ┘// € // € F I┌ Antiarrhythmic agents-Ib $♬┌└◘┐┌└┌┼ IB-phenytoin sodium Mechanism similar to lidocaine Pharmacologic effects * decrease automaticity in PF * compete with cardiac glycoside for combination of Na-K-ATPase Clinic use: to treat digoxin-induced dysrhythmias I┼Zd┌┼♬Z ┌┼Z(┌┌┼♬Z┘┌┼♬Z┐ └ └┐┌┌└┬ └┐└┘┐€ ″€ € B┼"€ € € € (└€ € t└ ┌└↔ ┌└% ┌└C ┌└┼

Antiarrhythmic agents-Ic $♬┌└◘┐┌└┌┼ IC-Propafenone Mechanism Na, (0,4), -R Pharmacologic effects automaticity conduction Clinic use: supraventricular arrhythmias ADR: QT ;metallic taste,constipation 14┌♬¶┌┌♬P┌┌┌

#┌◘ >└ ┌└J ┌└!┼┌└4 Antiarrhythmic agents-II -R antagonists Mechanism -R, Na, ERP + K Clinic use SVT, Af,hyperthyrosis ADR SB, AVB, HF, Hypotension, Asthma. _//%

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