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 %✂ > Antiarrhythmic Agents

BACKGROUND† Inside cell: K+ ; Outside cell: Na +, Ca + +, Cl - Action potential (AP) and
 Phase 0-4 phase0---reactivity---conduction--- Na + phase1----- K +
 phase2, 3---early afterdepolarization--- Ca + +; K + phase4---automaticity--- Na + ---delayed
 afterdepolarization-- Ca + +---Na + Effective Refractory Period: membrane potential~ -60 mV Y|Z-

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 B_7L b.^L_r^L -^L_r^L BACKGROUND† Classification of ARR

according to The mechanism of ARR (disturbances of Impulse formation or conduction) The Site of the
 origin (SupraVentricular, V.) HR increased or not (Tachycardia, Brady.) Etiology Carditis, AMI, CHD,
 coffee, tea, alcohol, drug& # 0_r_♫_♫_♫_♫_ f f f 3 f
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Mechanisms of arrhythmia "←_r→_r

2Electrophysiologic effects of Antiarrhythmic drugs \$↑_r^L ✂_r^L + 1.To
 decrease Automaticity Increase MDP, Slow down slope of phrase 4 , increase threshold potential (TP)
 2.To reduce post depolar. and triggered activity accelerate repolar. current to decrease EAD inhibit
 inward ion to decrease EAD increase TP to decrease EAD increase outward repolar. current to increase
 MDP antagonize cellular Ca to impaired LAD Inhibit Na influx to impaired LAD l_7_r_r_ZU+Z1_r_r_Z Z_7_r_r
 // 返_ :_r // X_7_r^L f \$ j c 2Electrophysiologic

effects of Antiarrhythmic drugs \$↑_r^L ✂_r^L + 0_r3.To change membrane responsiveness, so to
 change the conduction, hence to terminate reentry To increase MR so suppress unidirectional block To
 decrease MR so change unidirectional block to bidirectional block 4.To change ERP and APD so
 terminate reentry Absolutely prolong ERP and APD Relatively prolong ERP(shorten APD) Symmetric
 ERP r]_r_r_Zu+Z-_r_r_ZQ_r *Z]_r // -_r^L // "7

Antiarrhythmic agents Classification `!→sodium channel
 antagonists `!A →block Na+ C + +, quinidine `!B →+ lidocaine `!C →+++ flecanide a!→
 -R blockers: propranolol b!→selective prolong repolar.: amiodarone c!→CCBs:
 verapamil Z// i_r v^L[✂_7_↑_€ € H€ € € #_€ €

similar to quinidine Pharmacologic effects automaticity (pukinj fiber) conduction ERP,APD Adverse reaction: cardiotoxic effects; Extracardiotoxicity: syndrome resembling lupus erythematosus--- increased antinuclear Ab titer

Antiarrhythmic agents-Ib IB-lidocaine Mechanism: Na, + K Pharmacologic effects automaticity (slow velocity of P-4 in PF) APD/ERP terminate reentry + K hyperpolarization

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

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

Antiarrhythmic agents-Ic IC-Propafenone Mechanism Na, (0, 4), -R Pharmacologic effects automaticity conduction Clinic use: supraventricular arrhythmias ADR: QT ;metallic taste, constipation

Antiarrhythmic agents-II -R antagonists Mechanism -R, Na, ERP + K Clinic use SVT, Af, hyperthyrosis ADR SB, AVB, HF, Hypotension, Asthma.

Antiarrhythmic agents-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 : " Supraventricular (atrial fibrillation), ventricular (tachycardia/fibrillation) tachyarrhythmias AR: photosensitive skin, thyroid abnormalities (hypo- and hyper-), pulmonary fibrosis, corneal deposits, neurological and gastrointestinal disturbances

Antiarrhythmic agents-IV CCB: verapamil Pharmacological actions " reduce automaticity in sinoatrial node and atrioventricular 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

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 treat Bradycardia Atropine, Iso. 8 Common ADR of Antiarrhythmic agents is proarrhythmia.

The classification of arrhythmia The classification of Antiarrhythmic agents . The mechanisms of them Common ADR of them

Review & questions The classification of arrhythmia The classification of Antiarrhythmic agents . The mechanisms of them Common ADR of them

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