**Agents used in HT, CHF, Arrhythmia and Angina**

<table>
<thead>
<tr>
<th>Drug Class</th>
<th>Hypertension</th>
<th>HF</th>
<th>Arrhythmia</th>
<th>Angina</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beta-Blockers</td>
<td></td>
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<td></td>
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<tr>
<td>Ca++-Channel blockers</td>
<td></td>
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<tr>
<td>ACEI / ARBs</td>
<td></td>
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<tr>
<td>Diuretics (Thiazides)</td>
<td></td>
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<tr>
<td>Cardiac glycosides</td>
<td></td>
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<tr>
<td>Vasodilators</td>
<td></td>
<td></td>
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<tr>
<td>N+, Channel blockers</td>
<td></td>
<td></td>
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<tr>
<td>Nitrates</td>
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</tr>
</tbody>
</table>

**Contraindications/Cautions/Notes**
- HF (C), unstable HF, bronchoconstriction, significant bradycardia, depression: Raynaud D. Caution in diabetes, asthma (see D-)
- Angioedema, hypokalemia, cough (ace), tetrazepam, glos-sitis, taste
- Effects enhanced in depolarized, damaged tissue, Phase I, II CV
- Many Rx interactions, [K+], Lox NF important, low K+ toxicity
- Fluiding, dizziness, headache, nausea, reflex tachycardia
- Effects enhanced in depolarized, damaged tissue, Phase I, II CV
- Non-GMP, tolerance (off periods), flushing, dizziness, headache, reflex tachycardia, many forms

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**Heart Physiology**

- **Closed system**
  - Pressure driven
  - Supply nutrients/O₂
  - Remove metabolites

- **Electrocardiogram (ECG)**

- **P** - atria depolarization
- **QRS** - ventricle depolarization
- **PR** - conduction A-V
- **T** - ventricle repolarization
- **QT** - duration ventricle of repolarization

---

**Can’t see the forest for the trees**

*Hung up on the detail, you can not see the big picture*
The response of excitable cells to electrical stimulation is a function of the number of available Na channels (an index is dV/dt or Phase 0).

- Steady membrane depolarization decreases Na current, dV/dt and conduction velocity.
- Abnormal heart tissue is usually depolarized.
- Na channel availability results from Na channels being in different states.

Effect of drugs on Na channels available and recovery time constant...depends on the resting membrane potential.

What this means:
1. Conduction in damaged/abnormal heart tissue is decreased.
2. Antidysrhythmics will work better on Na channels in depolarized cells, and slow their recovery from excitation.

Characteristics of Arrhythmias

Definitions:
- Normal sinus rhythm (60-90 bpm), SA node pacemaker.
- Arrhythmia; any abnormality of firing rate, regularity or site of origin of cardiac impulse or disturbance of conduction that alters the normal sequence of activity of atria and ventricles.

Occurrence:
- 80% of patients with acute myocardial infarctions.
- 50% of anaesthetized patients.
- About 25% of patients on digitalis.

Classification of arrhythmia

1. Characteristics:
   a. Flutter – very rapid but regular contractions
   b. Tachycardia – increased rate
   c. Bradycardia – decreased rate
   d. Fibrillation – disorganized contractile activity

2. Sites involved:
   a. Ventricular
   b. Atrial
   c. Sinus
   d. AV node
   e. Supraventricular (atrial myocardium or AV node)

Examples of Arrhythmias

The ECG traces show each heartbeat as a series of electrical waves. Three of these waves, the P-wave, the QRS complex and the T-wave, are associated with the heart’s contraction. The P-wave reflects atrial depolarization, the QRS complex and T-wave reflect activity in the lower chambers.
Premature ventricular contraction (PVC) [Ventricular premature beat (VPB)]

Cardiac Action Potentials

Ion Permeability

Mechanisms of arrhythmias

Abnormal impulse generation (abnormal automaticity)
1. automaticity of normally automatic cells (SA, AV, His)
2. generation of impulses in normally non-automatic cells
   - development of phase 4 depolarization in normally non-automatic cells
   - 'triggered activity' due to afterdepolarizations
   - early afterdepolarization
   - delayed afterdepolarization

Abnormal impulse conduction (more common mechanism)
1. AV block – ventricle free to start own pacemaker rhythm
2. Re-entry: re-excitation around a conducting loop, which produces tachycardia
   - unidirectional conduction block
   - establishment of new loop of excitation
   - conduction time that outlasts refractory period

Abnormal impulse generation: 'triggered activity': EADs and DADs

Abnormal impulse conduction: Reentry
Unidirectional Block

Damaged tissue is usually depolarized → ↓ conduction velocity

Strategies of Antidysrhythmic Agents

Suppression of dysrhythmias

A. Alter automaticity
i. decrease slope of Phase 4 depolarization
ii. increase the threshold potential
iii. decrease resting (maximum diastolic) potential

B. Alter conduction velocity
i. mainly via decrease rate of rise of Phase 0 upstroke
ii. decrease Phase 4 slope
iii. decrease membrane resting potential and responsiveness

C. Alter the refractory period
i. increase Phase 2 plateau
ii. increase Phase 3 repolarization
iii. increase action potential duration

Classification of Antidysrhythmic Drugs

Vaughan-Williams classification (1970), subsequently modified by Harrison.

Helpful, But?

1. based on electrophysiological actions in normal tissue
2. presumes a mechanism of action of antidysrhythmic drugs
3. consists of four main classes and three subclasses
4. does not include actions of other agents (ie. adenosine)

Vaughan-Williams Classification

<table>
<thead>
<tr>
<th>Subclass</th>
<th>Mechanism</th>
<th>Prototype</th>
</tr>
</thead>
<tbody>
<tr>
<td>IA</td>
<td>Moderate Na block Ph.0; slow conduction; ↑ APD</td>
<td>Quinidine, Procanide</td>
</tr>
<tr>
<td>IB</td>
<td>Minimal Na block Ph.0; slow conduction (less); shorten Ph.3 repolarization</td>
<td>Lidocaine, Phenytoin</td>
</tr>
</tbody>
</table>
| IC       | Marked Na block Ph.0; slow conduction; no change APD or repolarization. Increased suppression of Na channels | Class III
| Class III | Beta blockers; decrease adrenergic input. No major effect on APD, suppress Phase 4 depolarization | Propranolol, others |
| Class IV  | Prolong repolarization/refractory period other means than exclusively Na block (mainly K+ channel blockade) | Amiodarone, Bretylum |
| Class IV  | Ca channel blockers. Slow conduction and ↑ effective refractory period in normal tissue (A-V node) and Ca-dependent slow responses of depolarized tissue (atria, ventricle, Purkinje) | Verapamil, Diltiazem |
| Others   | Adenosine, Digoxin, Anticoagulants, ANS agents | |

Electrophysiological Properties Of Specialized Cardiac Fibers

<table>
<thead>
<tr>
<th>CLASS OF ANTARRHYTHMIC DRUG</th>
<th>IA</th>
<th>IB</th>
<th>IC</th>
<th>II</th>
<th>III</th>
<th>IV</th>
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</thead>
<tbody>
<tr>
<td>Sinus node Automaticity</td>
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<td>0↓</td>
<td>0↓</td>
<td>0↓</td>
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<td>AV node</td>
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<td>0↓</td>
</tr>
<tr>
<td>Effective refractory period (ERP)</td>
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<td>0↓</td>
<td>0↓</td>
<td>0↓</td>
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<tr>
<td>Purkinje fibers</td>
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<td>0↓</td>
<td>0↓</td>
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<tr>
<td>Action potential amplitude</td>
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<td>0↓</td>
<td>0↑</td>
<td>0↑</td>
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<tr>
<td>Phase-0 Vmax</td>
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<td>0↓</td>
<td>0↓</td>
<td>0↓</td>
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<tr>
<td>Action potential duration (APD)</td>
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<tr>
<td>Membrane responsiveness</td>
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</tr>
<tr>
<td>Automaticity</td>
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<td>0↓</td>
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<td>0↑</td>
<td>0↑</td>
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</table>

Action Potential – Ion Flow

<table>
<thead>
<tr>
<th>mM</th>
<th>Na⁺</th>
<th>K⁺</th>
<th>Ca²⁺</th>
</tr>
</thead>
<tbody>
<tr>
<td>Out</td>
<td>140</td>
<td>4</td>
<td>2.5</td>
</tr>
<tr>
<td>In</td>
<td>10</td>
<td>150</td>
<td>0.1</td>
</tr>
</tbody>
</table>

Na⁺/Ca²⁺ exchange (3:1) 
Na⁺/K⁺ ATPase (3:2)
Shortcomings: Vaughan-Williams system (V-W)

1. Hybrid: Class I, III & IV agents block ion channels, Class II block receptors. Single class can effect multiple mechanisms. Amiodarone: properties of classes (I-IV).
2. Describes drugs that block ion channels/receptors and does not consider drugs that activate channels or receptors.
3. Incomplete: eg. adenosine, digitalis, cholinergic agonists, alpha adrenergic blockers or agents that modulate gap junctions, ion pumps or exchangers. Also ignores drug metabolites.
4. Based on response in normal tissue, not damaged
5. Does not incorporate variable mode of action: slowing tachycardias, terminating dysrhythmias, or preventing them.

Amiodarone: Only Antiarrhythmic with All Four Vaughan-Williams’ Class Effects

Drugs work better on depolarized tissue....

Key Aspects of Drug Action and Therapy...

A. Drug action is state-dependent: channel open, closed, or inactivated. Two current models:
   1. modulated receptor hypothesis: different states have different affinities
   2. guarded receptor hypothesis: channel gate limits drug access to site
B. Drugs selectively affect firing and conduction in abnormal/depolarized cells.
C. Transitions between states dependent on membrane voltage and cell firing frequency. Membrane voltage and spike frequency. Binding to inactivated states and slowing recovery from inactivation, some drugs can increase the time needed for recovery from inactivation.
Key Aspects of Drug Action and Therapy...
D. Drugs affect different parts of the heart: Beta-blockers and CCBs used for SVT
1. Ca channel blockers (Class IV) are selective for A-V and S-A nodes, where Ca action potentials predominate.
2. Lidocaine (Class IB) has been useful for treating PVCs in Purkinje fibers, since longer APDs in Purkinje yield more inactivated Na channels. Lidocaine selectively blocks inactivated state and some open Na channels. Lidocaine has little effect, in contrast, on atrial tissue.
3. Quinidine affects both atrial and ventricular dysrhythmias (but has been mostly used to treat atrial fibrillation).

Other Aspects of Drug Action and Therapy...
A. Multiple actions hinder understanding MOA
B. Can be prodrhythmic under certain circumstances
C. Often affect ANS and cardiovascular system
D. May have significant cardiac and extracardiac toxicity; eg. Class 1A drugs can depress cardiac contractility
E. Can interact with other drugs. Useful interactions as well as deleterious interactions occur, with the former exploited in combination therapy (e.g. quinidine and digoxin to treat supraventricular tachycardias). Combinations of antidysrhythmics are used to increase the efficacy of therapy and to avoid the necessity of using higher doses of any particular drug alone.

Quinidine (Class IA prototype)
Other examples: Procainamide, Disopyramide
1. General properties:
   a. D-isomer of quinine
   b. As with most of the Class I agents
      - moderate block of sodium channels
      - decreases automaticity of pacemaker cells
      - increases effective refractory period/AP duration

Actions of Quinidine
Cardiac effects
   a. ↓ automaticity, conduction velocity and excitability of cardiac cells.
   b. Preferentially blocks open Na channels
   c. Recovery from block slow in depolarized tissue; lengthens refractory period (RP)
   d. All effects are potentiated in depolarized tissues
   e. Increases action potential duration (APD) and prolongs AP repolarization via block of K channels; decreases reentry
   f. Indirect action: anticholinergic effect (accelerates heart), which can speed A-V conduction.

Actions & Toxicity of Quinidine
Extracardiac
   a. Blocks alpha-adrenoreceptors to yield vasodilatation.
   b. Other strong antimuscarnic actions

Toxicity
   - "Quinidine syncope"(fainting)- due to disorganized ventricular tachycardia
   - associated with greatly lengthened Q-T interval; can lead to Torsades de Pointes (VT, precursor to ventricular fibrillation)
   - negative inotropic action (decreases contractility)
   - GI - diarrhea, nausea, vomiting
   - CNS effects - headaches, dizziness, tinnitus (quinidine "Cinchonism")

Quinidine: Pharmacokinetics/therapeutics
   a. Oral, rapidly absorbed, 80% bound to membrane proteins
   b. Hydroxylated in liver; T 1/2 = 6-8 h
   c. Drug interaction: displaces digoxin from binding sites; so avoid giving drugs together or reduce dose
   d. Probably are active metabolites of quinidine
   e. Effective in treatment of nearly all dysrhythmias, including:
      1) Premature atrial contractions
      2) Paroxysmal atrial fibrillation and flutter
      3) Intra-atrial and A-V nodal reentrant dysrhythmias
      4) Wolff-Parkinson-White tachycardias (SVT, A-V bypass)
      5) Premature ventricular contractions (PVCs)
   f. Useful in treating chronic dysrhythmias requiring outpatient treatment
Procainamide (Class 1A) also Disopyrimide

Cardiac effects
a. Similar to quinidine, less muscarinic & alpha-adrenergic blockade
b. Has negative inotropic action also

Extracardiac effects
a. Ganglionic blocking reduces peripheral vascular resistance

Toxicity
a. Cardiac: Similar to quinidine; cardiac depression
b. Noncardiac: Syndrome resembling lupus erythematosus

Pharmacokinetics/therapeutics
a. Administered orally, i-v and intramuscularly
b. Major metabolite in liver is N-acetylprocainamide (NAPA), a weak Na channel blocker with class III activity. Bimodal distribution in population of rapid acetylators, who can accumulate high levels of NAPA.
c. T₁/₂ = 3-4 hours; necessitates frequent dosing; kidney chief elimination path. NAPA has longer T₁/₂ and can accumulate
d. Usually used short-term. Commonly used in ICUs for ventricular dysrhythmias associated with acute myocardial infarctions (MI)

Lidocaine (Class IB prototype)

Other examples: Mexiletine, Phenytoin, Tocainide

General
a. Commonly used antidyssrhythmic agent in emergency care (decreasing use)
b. Given i-v and i-m; widely used in ICU-critical care units (old DOC, prior 2001)
c. Low toxicity
d. A local anesthetic, works on nerve at higher doses

Lidocaine Actions

Cardiac effects
a. Generally decreases APD, hastens AP repolarization, decreases automaticity and increases refractory period in depolarized cells.
b. Exclusively acts on Na channels in depolarized tissue by blocking open and inactivated (mainly) Na channels
c. Potent suppresser of abnormal activity
d. Most Na channels of normal cells rapidly unblock from lidocaine during diastole; few electrophysiological effects in normal tissue

Toxicity:
- least cardiotoxic; high dose can lead to hypotension
- tremors, nausea, slurred speech, convulsions

Pharmacokinetics/therapy
a. i-v, i-m since extensive first pass hepatic metabolism
b. T₁/₂ = 0.5-4 hours
c. Effective in suppressing dysrhythmia associated with depol. tissue (ischemia; digitalis toxicity); ineffective against dysrhythmias in normal tissue (atrial flutter).
d. Suppresses ventricular tachycardia; prevents fibrillation after acute MI; rarely used in supraventricular arrhythmias
e. Drug interaction: Propranolol, cimetidine, ↓ plasma clearance

Phenytoin (Class IB)

1. Non-sedative anticonvulsant used in treating epilepsy (‘Dilantin’)
2. Limited efficacy as antidyssrhythmic (second line antiarrythmic)
3. Suppresses ectopic activation by blocking Na and Ca channels
4. Especially effective against digitalis-induced dysrhythmias
5. T₁/₂ = 24 hr - metabolized in liver
6. Gingival hyperplasia (40%)
Propranolol (Class II, beta adrenoreceptor blockers)

Other agents: Metoprolol, Esmolol (short acting), Sotalol (also Class III), Acesubutolol

a. Slow A-V conduction
b. Prolong A-V refractory period
c. Suppress automaticity

cardiac effects (of propranolol), a non-selective beta blocker

a. Main mechanism of action is block of beta receptors; ↓ Ph 4 slope, which decreases automaticity under certain conditions
b. Some direct local anesthetic effect by block of Na channels (membrane stabilization) at higher doses
c. Increases refractory period in depolarized tissues
d. Increases A-V nodal refractory period

Non-cardiac: Hypotension

Therapeutics

a. Blocks abnormal pacemakers in cells receiving excess catecholamines (e.g. pheochromocytoma) or up-regulated beta-receptors (e.g. hyperthyroidism)
b. Blocks A-V nodal reentrant tachycardias; inhibits ectopic foci
c. Propranolol used to treat supraventricular tachydysrhythmias
d. Contraindicated in ventricular failure; also can lead to A-V block.

Oral (propranolol) or IV. Extensive metabolism in liver.

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Oral (propranolol) or IV. Extensive metabolism in liver.
AHA Guidelines for VT/VF 2000
1. Amiodarone
2. Procainamide, [Bretylium]
3. Sotalol
4. Lidocaine

AHA Guideline Prior to 2000
1. Lidocaine
2. Procainamide
3. Bretylium
4. Electrical cardioversion

Cardiac effects of Amiodarone
a. Block Na channels (1A), but low affinity for open channels; mainly blocks inactivated Na channels
b. Block is most pronounced in tissues with long action potentials, frequent spiking or more depolarized diastolic potential
c. Weak Ca channel blocker also (Class IV activity)
d. A powerful inhibitor of abnormal automaticity, decreases conduction, increases refractory period and APD.
e. Has antianginal effects (blocks alpha/beta receptors and Ca channels)
Extracardiac effects: Vasodilation via block of Ca channels and alpha receptors

Adverse effects: Amiodarone
A. Cardiac
i. Sinus bradycardia, increase QT interval
ii. Negative inotropic action due to block of Ca channels and beta receptors; but can improve heart failure via vasodilation.
iii. A-V block, paradoxical VTs.
B. Non-cardiac:
   i. Deposits into almost every organ
   ii. Reduces clearance of drugs like procainamide, flecainide, digitalis, quinidine and diltiazem.
   iii. Thyroid dysfunction (hypo or hyperthyroidism)
   iv. Pulmonary fibrosis is most serious adverse effect
   v. Paresthesias (tingling, pricking, or numbness)
   vi. Photosensitivity
   vii. Corneal microdeposits and blurred vision
   viii. Ataxia, dizziness, tremor
   ix. Anorexia, nausea, vomiting

Amiodarone: Pharmacokinetics and therapeutics
• $T_{1/2} = 13-103$ days (weeks) very long for one dose; very lipidsoluble; metabolized in liver
  • Effective against many arrhythmias: atrial, A-V and ventricular dysrhythmias; prevention of atrial fibrillation/flutter; PVCs, nonsustained & sustained VTs.
  • Multiple interactions with other drugs such as:
    i. Amiodarone is a CYP3A4 substrate and inhibitor and thus may enhance the effect of other CYP3A4 substrates eg. Warfarin, Simvastatin, Verapamil
    ii. Amiodarone may increase the serum concentration of Cardiac Glycosides

Pulmonary fibrosis

Bretylium (Class III, $K^+$ channel blockers)
Others Amiodarone, Ibutilide, (Sotalol, also beta-blocker)
General: originally used as an antihypertensive agent
Cardiac effects
a. Direct antidysrhythmic action
b. Increases ventricular APD and increases refractory period; decreases automatically
c. Most pronounced action in ischemic cells having short APD
d. Initially stimulates and then blocks neuronal catecholamine release from adrenergic nerve terminals
e. Blocks cardiac K channels to increase APD
Extracardiac effects: Hypotension (from block of NE release)
Pharmacokinetics/therapeutics
a. iv or intramuscular
b. Excreted mainly by the kidney
c. Usually for emergency use only: ventricular fibrillation when lidocaine and cardioversion therapy fail. Increases threshold for fibrillation.
d. Decreases tachycardias and early extrasystoles by increasing effective refractory period
Ibutilide (Class III).
1. Prolongs cardiac action potential without additional effects.
2. Mechanism incompletely understood but known to increase inward Na current and decrease repolarizing K current.
3. Can be administered I-V or orally. Most effective current agent to convert atrial fibrillation and flutter of recent onset to normal rhythm. Low incidence of Torsades (about 2%), compared to other drugs.
4. More effective for flutter than fibrillation.
5. Generally well tolerated.

Sotalol (Class III and Class II)
1. Non-selective beta blocker, increases AP duration.
2. Dysrhythmias of supraventricular and ventricular origin.

Ca++ Channel Blockers - Actions
Extracardiac:
- Peripheral vasodilatation via effect on smooth muscle
- Used as antianginal / antihypertensive
- Hypotension may increase HR reflexively

Toxicity:
- Cardiac: Too negative inotropic for damaged heart, depresses contractility; Can produce full A-V block
- Extracardiac: Constipation, nervousness; Gingival hyperplasia

Pharmacokinetics/Therapeutics:
- T$_1$/2 = 7h, metabolized by liver
- Oral administration; also available parenterally
- Great caution for patients with liver disease

Dysrhythmics - Others
1. Adenosine: i.v. (15 secs), activates P1 purinergic receptors (A1) coupled to K channels, ↓CV, ↑refractory period. SVT. Flushing, hypotension, burning sensation
2. Potassium ions (K+): Depress ectopic pacemakers
3. Digoxin: used to treat atrial flutter and fibrillation
4. Autonomic agents: used to treat A-V block - β-agonists (ie. EPI), anticholinergics (ie. atropine)
5. Anticoagulant therapy: - prevent formation of systemic emboli & stroke

Drug interactions involving antidysrhythmics
A. These drugs must be used very carefully
B. Sometimes interactions can be counter-intuitive
Problems with selecting drugs:
A. Do not always know the cause of the dysrhythmia, thus what to treat?
B. Multiple mechanisms of dysrhythmogenesis
C. Drugs are both anti- and pro-dysrhythmias
D. Drugs do not really fix the damage; usually they restore function by breaking something else

Verapamil (Class IV, Ca++ channel blockers)
Other example: Diltiazem - CCBs increasing use and importance
a. Blocks active and inactivated Ca channels, prevents Ca entry
b. More effective on depolarized tissue,issue firing frequently or areas where activity dependent on Ca channels (SA node, A-V node)
c. Increases A-V conduction time and refractory period; directly slows SA and A-V node automaticity
d. Suppresses oscillatory depolarizing after depolarizations due to digitalis
### Pharmacokinetic Properties of Antiarrhythmic Drugs

<table>
<thead>
<tr>
<th>Drug</th>
<th>Class</th>
<th>Plasma Binding %</th>
<th>$T_{1/2}$ (hrs)</th>
<th>Drug Excretion</th>
<th>Unchanged</th>
</tr>
</thead>
<tbody>
<tr>
<td>Quinidine</td>
<td>IA</td>
<td>90</td>
<td>6</td>
<td>20-40%</td>
<td></td>
</tr>
<tr>
<td>Procainamide</td>
<td>IA</td>
<td>15</td>
<td>4</td>
<td>60%</td>
<td></td>
</tr>
<tr>
<td>Disopyramide</td>
<td>IA</td>
<td>30-95</td>
<td>5</td>
<td>50-70%</td>
<td></td>
</tr>
<tr>
<td>Lidocaine</td>
<td>II</td>
<td>40</td>
<td>2</td>
<td>&lt;10%</td>
<td></td>
</tr>
<tr>
<td>Tocainide</td>
<td>IB</td>
<td>10</td>
<td>14</td>
<td>40%</td>
<td></td>
</tr>
<tr>
<td>Mexiletine</td>
<td>IB</td>
<td>65</td>
<td>12</td>
<td>10%</td>
<td></td>
</tr>
<tr>
<td>Flecainide</td>
<td>IC</td>
<td>45</td>
<td>15</td>
<td>40%</td>
<td></td>
</tr>
<tr>
<td>Propranolol</td>
<td>II</td>
<td>90</td>
<td>4</td>
<td>&lt;1%</td>
<td></td>
</tr>
<tr>
<td>Metoprolol</td>
<td>II</td>
<td>15</td>
<td>4</td>
<td>&lt;3%</td>
<td></td>
</tr>
<tr>
<td>Esmolol</td>
<td>III</td>
<td>(hydro. esterase)</td>
<td>9 min</td>
<td>&lt;1%</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>III</td>
<td>9</td>
<td>90%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amiodarone</td>
<td>III</td>
<td>95</td>
<td>&gt; 25 days</td>
<td>&lt;1%</td>
<td></td>
</tr>
<tr>
<td>Sotalol</td>
<td>III</td>
<td>5</td>
<td>9</td>
<td>80%</td>
<td></td>
</tr>
<tr>
<td>Verapamil</td>
<td>IV</td>
<td>50</td>
<td>3</td>
<td>2%</td>
<td></td>
</tr>
<tr>
<td>Adenosine</td>
<td>Misc (other)</td>
<td>15 sec</td>
<td>90%</td>
<td>2%</td>
<td></td>
</tr>
</tbody>
</table>

### Dysrhythmia Treatment

**Treatment**
- Acute vs Chronic

**Site**
- Ventricular vs Supraventricular